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NEW POPs: CRITERIA AND PROCESS

WHAT DOES THE STOCKHOLM CONVENTION SAY?

The Stockholm Convention on Persistent Organic Pollutants (POPs) is a living convention and does more than address the original 'dirty dozen' POPs chemicals. It recognizes the need to take global action on all chemicals with POP-like characteristics, such as:

- persistent in the environment;
- travel long distances via air and water;
- are toxic; and
- bioaccumulate in living things.

These chemicals pose an unacceptable threat to human health and the environment.

The Stockholm Convention established a 'science based' process for identifying candidate POPs. However, the process applies the precautionary principle by recognising that there does not have to be absolute proof that a chemical is doing harm before action on it is taken.

The Stockholm Convention including Article 8 and Annexes D, E, F establish the rules for identifying and listing additional POPs.

ANNEX D
TO THE STOCKHOLM CONVENTION
ON
INFORMATION REQUIREMENTS AND SCREENING CRITERIA

a) A Party submitting a proposal to list a chemical in Annexes A, B and/or C shall identify the chemical in the manner described in subparagraph (a) and provide the information on the chemical, and its transformation products where relevant, relating to the screening criteria set out in subparagraphs (b) to (e):

a) Chemical identity:
   i. Names, including trade name or names, commercial name or names and synonyms, Chemical Abstracts Service (CAS) Registry number, International Union of Pure and Applied Chemistry (IUPAC) name; and
ii. Structure, including specification of isomers, where applicable, and the structure of the chemical class;

b) Persistence:
  i. Evidence that the half-life of the chemical in water is greater than two months, or that its half-life in soil is greater than six months, or that its half-life in sediment is greater than six months; or
  ii. Evidence that the chemical is otherwise sufficiently persistent to justify its consideration within the scope of this Convention;

c) Bio-accumulation:
  i. Evidence that the bio-concentration factor or bio-accumulation factor in aquatic species for the chemical is greater than 5,000 or, in the absence of such data, that the log Kow is greater than 5;
  ii. Evidence that a chemical presents other reasons for concern, such as high bio-accumulation in other species, high toxicity or ecotoxicity; or
  iii. Monitoring data in biota indicating that the bio-accumulation potential of the chemical is sufficient to justify its consideration within the scope of this Convention;

d) Potential for long-range environmental transport:
  i. Measured levels of the chemical in locations distant from the sources of its release that are of potential concern;
  ii. Monitoring data showing that long-range environmental transport of the chemical, with the potential for transfer to a receiving environment, may have occurred via air, water or migratory species; or
  iii. Environmental fate properties and/or model results that demonstrate that the chemical has a potential for long-range environmental transport through air, water or migratory species, with the potential for transfer to a receiving environment in locations distant from the sources of its release. For a chemical that migrates significantly through the air, its half-life in air should be greater than two days; and

e) Adverse effects:
  i. Evidence of adverse effects to human health or to the environment that justifies consideration of the chemical within the scope of this Convention; or
  ii. Toxicity or ecotoxicity data that indicate the potential for damage to human health or to the environment.

2. The proposing Party shall provide a statement of the reasons for concern including, where possible, a comparison of toxicity or ecotoxicity data with detected or predicted levels of a chemical resulting or anticipated from its long-range environmental transport, and a short statement indicating the need for global control.

3. The proposing Party shall, to the extent possible and taking into account its capabilities, provide additional information to support the review of the proposal referred to in paragraph 6 of Article 8. In developing such a proposal, a Party may
ANNEX E
TO THE STOCKHOLM CONVENTION
ON
INFORMATION REQUIREMENTS FOR THE RISK PROFILE

The purpose of the review is to evaluate whether the chemical is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted. For this purpose, a risk profile shall be developed that further elaborates on, and evaluates, the information referred to in Annex D and includes, as far as possible, the following types of information:

a) Sources, including as appropriate:
   (i) Production data, including quantity and location;
   (ii) Uses; and
   (iii) Releases, such as discharges, losses and emissions;

b) Hazard assessment for the endpoint or endpoints of concern, including a consideration of toxicological interactions involving multiple chemicals;

c) Environmental fate, including data and information on the chemical and physical properties of a chemical as well as its persistence and how they are linked to its environmental transport, transfer within and between environmental compartments, degradation and transformation to other chemicals. A determination of the bio-concentration factor or bio-accumulation factor, based on measured values, shall be available, except when monitoring data are judged to meet this need;

d) Monitoring data;

e) Exposure in local areas and, in particular, as a result of long-range environmental transport, and including information regarding bio-availability;

f) National and international risk evaluations, assessments or profiles and labelling information and hazard classifications, as available; and

g) Status of the chemical under international conventions.

ANNEX F
TO THE STOCKHOLM CONVENTION
ON
INFORMATION ON SOCIO-ECONOMIC CONSIDERATIONS

An evaluation should be undertaken regarding possible control measures for chemicals under consideration for inclusion in this Convention, encompassing the full range of options, including management and elimination. For this purpose, relevant
information should be provided relating to socioeconomic considerations associated with possible control measures to enable a decision to be taken by the Conference of the Parties. Such information should reflect due regard for the differing capabilities and conditions among the Parties and should include consideration of the following indicative list of items:

a) Efficacy and efficiency of possible control measures in meeting risk reduction goals:
   (i) Technical feasibility; and
   (ii) Costs, including environmental and health costs;

b) Alternatives (products and processes):
   (i) Technical feasibility;
   (ii) Costs, including environmental and health costs;
   (iii) Efficacy;
   (iv) Risk;
   (v) Availability; and (vi) Accessibility;

c) Positive and/or negative impacts on society of implementing possible control measures:
   (i) Health, including public, environmental and occupational health;
   (ii) Agriculture, including aquaculture and forestry;
   (iii) Biota (biodiversity);
   (iv) Economic aspects;
   (v) Movement towards sustainable development; and
   (vi) Social costs;

d) Waste and disposal implications (in particular, obsolete stocks of pesticides and clean-up of contaminated sites):
   (i) Technical feasibility; and
   (ii) Cost;

e) Access to information and public education;

f) Status of control and monitoring capacity; and

g) Any national or regional control actions taken, including information on alternatives, and other relevant risk management information.

**PROCESS FOR NOMINATION OF NEW CHEMICALS**

Any country that has ratified the Convention can submit a proposal to the Secretariat for listing a chemical in Annexes A, B and/or C.

However, the proposal needs to contain the information specified in Annex D (Information Requirements And Screening Criteria).

This includes information on the:
• **Chemical identity and structure**, including trade name/names, synonyms, Chemical Abstracts Service (CAS) Registry number and International Union of Pure and Applied Chemistry (IUPAC) name;

• **Persistence** in the form of evidence of half-life in water greater than two months, or half-life in soil or sediment greater than six months, or other evidence that of persistence;

• **Bio-accumulation** in the form of evidence that the bio-concentration factor or bio-accumulation factor in aquatic species is greater than 5,000 or that the log Kow is greater than 5, or other evidence of high bio-accumulation in species, high toxicity or ecotoxicity, or monitoring data indicating bio-accumulation potential;

• **Potential for long-range environmental transport** in the form of measured levels of concern in locations distant from the source, or monitoring data showing long-range environmental transport, or environmental fate properties and/or model results that demonstrate the potential for long-range environmental transport through air (with a half-life in air should be greater than two days), water or migratory species; and

• **Evidence of adverse effects to human health or the environment** or toxicity or ecotoxicity data indicating potential to damage human health or the environment.

The nominating country also needs to state the reasons for concern and the need for global control. In preparing the nomination the country can use technical expertise from any source

The proposed new POPs and the supporting information is then reviewed by the Persistent Organic Pollutants Review Committee to see if the screening criteria have been fulfilled.

**POP REVIEW COMMITTEE (POPRC)**

**STAKEHOLDERS**

The members of the first the Persistent Organic Pollutants Review Committee (POPRC) were appointed by the First Conference of the Parties (COP1) of the Stockholm Convention (May 2005, Uruguay) on the basis of "equitable geographical distribution, taking into account gender and the need for a balance between different types of expertise."

The POPRC consist of 31 members:

- African States: 8
- Asian and Pacific States: 8
- Central and Eastern European States: 3
- Latin American and Caribbean States: 5
- Western European and other States: 7
Members of the POPRC are "government-designated experts in chemical assessment or management." To ensure effective rotation of the membership, one half of the members of each region were nominated for an initial term of two years, and the remaining members of each region were nominated for an initial term of four years.

**Africa:**
Chad, Côte d'Ivoire, Ethiopia, Mauritania (2 years);
Burkina Faso, Morocco, Sierra Leone, South Africa (4 years);

**Asia and the Pacific:**
Fiji, Philippines, Qatar, Yemen (2 years);
China, Japan, Jordan, Thailand (4 years);

**Central and Eastern Europe:**
Slovenia (two years);
Armenia, Czech Republic (four years);

**Latin America and the Caribbean:**
Trinidad and Tobago, Uruguay (two years);
Brazil, Ecuador, Mexico (four years);

**Western Europe and others:**
Norway, Spain, United Kingdom (two years);
Australia, Canada, Germany, Sweden (four years).

The COP1 chose Mr. Reiner Arndt (Germany) as Chair of the POPRC.

POPRC can also invite up to 30 experts (balancing developed and developing countries) to support its work. Countries can designate experts for inclusion in a roster of expert but if no expert on the roster has the specifically needed expertise on a certain issue, the POPRC can then invite other experts.

The POPRC meetings are open to observers, while countries submitting a proposal are invited to attend the relevant POPRC meetings.

The Persistent Organic Pollutants Review Committee (POPRC) reviews proposals for new POPs via three main stages.

1. If the proposal includes the required information, the POPRC consider whether the nominated chemical fulfills the criteria (POP-like characteristics) as outlined in Annex D.

2. If the POPRC find the chemicals fulfills the criteria, a risk profile based on the information in Annex E is then prepared.

3. If on the basis of the risk profile, the POPRC decides the chemical is likely as a result of its long range transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted then the
POPRC will prepare a risk management evaluation based on information outlined in Annex F.

The POPRC may then recommend to the COP that the chemical be added to the Convention.

The Conference of the Parties (all the countries that have ratified the Convention) make the final decision on whether to list a chemical as a POP.

There are many chemicals with POP-like characteristics which need priority consideration.

Some are already scheduled for elimination through countries' national action or regional treaties like the UNECE Convention on Long-Range Transboundary Air Pollution (LRTAP) on POPs and the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR). Listing on an international agreement will ensure that these chemicals are banned throughout the globe.

REVIEW PROCESS

Once the Secretariat verifies that a proposal for listing contains the required information, it is forwarded to the Persistent Organic Pollutants Review Committee (POPRC) for consideration.

If POPRC decides that screening criteria have been fulfilled, the proposal is made available to all Parties and observers and they are invited to submit the information specified in Annex E (Information Requirements for the Risk Profile).

This includes information on:
- Sources such as production quantity and location, uses and releases;
- Hazard assessments for the endpoints of concern, including consideration of interactions with other chemicals;
- Environmental fate such as environmental transport and transfer, degradation, measured bio-concentration or bio-accumulation factors;
- Monitoring data;
- Exposure and bio-availability;
- Other national and international risk evaluations, labelling information and hazard classifications; and
- Chemical status under international conventions.

The POPRC then further reviews the proposal, taking into account any relevant additional information received, and prepares a draft risk profile and makes that draft available to all Parties and observers for comment. Based on their technical comments, the POPRC completes the risk profile.

If, on the basis of that risk profile, the POPRC decides “the chemical is likely as a result of its long-range environmental transport to lead to significant adverse human health and/or environmental effects such that global action is warranted,” the proposal proceeds to the next stage.
Importantly, **lack of full scientific certainty can not prevent the proposal from proceeding.**

The Committee then invites all Parties and observers to provide any information on socio-economic considerations as described in Annex F (Information on Socio-Economic Considerations). An evaluation of possible control measures and options, including management and elimination is required. The evaluation should consider:

- Efficacy and efficiency of possible control measures including technical feasibility; and environmental and health costs; and
- Alternatives including their technical feasibility, environmental and health costs, efficacy, risks, availability and accessibility, societal impacts of implementing possible control measures, including impacts on public, environmental and occupational health; agriculture, biota (biodiversity), economic aspects; sustainable development, social costs, waste and disposal implications, technical feasibility, costs, information access and public education, control and monitoring capacity; and national or regional control actions.

Based on this information a risk management evaluation including analysis of possible control measures is prepared.

The POPRC then based on the risk profile and the risk management evaluation recommend whether the chemical should be considered by the Conference of the Parties for listing in Annexes A, B and/or C.

Then taking account of the recommendations of the POPRC, including *any scientific uncertainty, and in a precautionary manner*, the Conference of the Parties decide whether to list the chemical, and specify the control measures, in Annexes A, B and/or C.

If a Proposal is rejected, it can be resubmitted and a Party can challenge the decision of the POPRC.
B. NOMINATED CHEMICALS – AN OVERVIEW

The following chemicals have been officially nominated by Parties for consideration by the POPs Review Committee for inclusion in the POPs list.

1. Alpha-hexachlorocyclohexane
2. Beta-hexachlorocyclohexane
3. Chlordecone
4. Endosulfan
5. Hexabromobiphenyl
6. Lindane
7. Octabromodiphenyl ether
8. Pentachlorobenzene
9. Pentabromodiphenyl ether
10. Perfluorooctane sulfonate
11. Short-chained chlorinated paraffins (SCCPs)

Detailed Profile of each of these chemicals is available in the following documents. Brief summary is also available.
## NOMINEES

<table>
<thead>
<tr>
<th>Substance</th>
<th>Abbreviation</th>
<th>Proposing Party</th>
<th>Evaluation Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha hexachlorocyclohexane</td>
<td>Alpha HCH</td>
<td>Mexico</td>
<td>Annex E</td>
</tr>
<tr>
<td>Beta hexachlorocyclohexane</td>
<td>Beta HCH</td>
<td>Mexico</td>
<td>Annex E</td>
</tr>
<tr>
<td>Chlordane</td>
<td></td>
<td>European Union</td>
<td>Annex F</td>
</tr>
<tr>
<td>Endosulfan</td>
<td></td>
<td>European Union</td>
<td>Annex D</td>
</tr>
<tr>
<td>Hexabromobiphenyl</td>
<td>HBB</td>
<td>European Union</td>
<td>Annex F</td>
</tr>
<tr>
<td>Lindane</td>
<td></td>
<td>Mexico</td>
<td>Annex F</td>
</tr>
<tr>
<td>Octabromodiphenyl ether</td>
<td>OctaBDE</td>
<td>European Union</td>
<td>Annex E</td>
</tr>
<tr>
<td>Pentabromodiphenyl ether</td>
<td>PentaBDE</td>
<td>Norway</td>
<td>Annex F</td>
</tr>
<tr>
<td>Pentachlorobenzene</td>
<td>PeCB</td>
<td>European Union</td>
<td>Annex E</td>
</tr>
<tr>
<td>Perfluorooctane sulfonate</td>
<td>PFOS</td>
<td>Sweden</td>
<td>Annex F</td>
</tr>
<tr>
<td>Short-chained chlorinated paraffins</td>
<td>SCCPs</td>
<td>European Union</td>
<td>Annex E</td>
</tr>
</tbody>
</table>

## PRODUCERS

<table>
<thead>
<tr>
<th>Substance</th>
<th>Past and Present Producers</th>
<th>Trade Names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha HCH</td>
<td>Not intentionally produced; see Lindane</td>
<td>Kepeone, GC-1189, Merex, ENT 16391, Curlone</td>
</tr>
<tr>
<td>Beta HCH</td>
<td>Not intentionally produced; see Lindane</td>
<td></td>
</tr>
<tr>
<td>Chlordane</td>
<td>Allied Chemical (USA); Life Sciences Products (USA); Hooker Chemical (USA); Nease Chemical (USA); De Laguarique (France); unnamed French and Brazilian companies</td>
<td></td>
</tr>
<tr>
<td>Endosulfan</td>
<td>China; India (All India Medical Corp, Bharat Pulverizing Mills, Excel Industries, Krishi Rasayan, Mewar Oil and General Mills); Germany (Bayer CropScience, Hoechst); Israel (Makhteshim Chemical Works); Italy (Dupont); South Korea; Mexico (Production Quimicos de Chihuahua); Taiwan (Mictionon Industries); UK (FBC); USA (FMC, Drexel, SureCo)</td>
<td>Benzoepin, Beosit, Bio 5462, Chlorthiepin, Crisultan, Cyclozan, Endocel, Endosol, EndosulfanE, Endosulfin, Endosulfan, Endosulfan 350EC, Endosulphan, ENT-23979, FMC 5462, Hildan, HOE 2671, Insectophene, Kops-Thiodan, Malix, NCI-C00566, NIA 5462, Niagara 5462, OMS 570, SD 4314, Thiofur, Thumul, Thiodan, Thionex, Farmoz, Nufarm, Tiovel</td>
</tr>
<tr>
<td>HBB</td>
<td>Michigan Chemical Corp (USA); White Chemical Corp (USA); Atotech (France); Berk Corp (UK); Chemische Fabrik Kalk (Germany)</td>
<td>Firemaster BP-6 Firemaster FF-1</td>
</tr>
<tr>
<td>Lindane</td>
<td>Companies in Albania, Argentina, Austria, Azerbaijan, Brazil, Bulgaria, China, Czech Republic, France, Germany (Bayer CropScience), Ghana, Hungary, India (KCIL, Kanoria, India Pesticides Ltd), Italy, Japan, Poland, Romania, Russia, Slovakia, Spain (Inquinos), Turkey, United Kingdom, and USA (Crompton, Gustafson). It appears that only Romania and India are current producing countries.</td>
<td>Benhexachlor, BHC, Exagama, Forlin, Gallowama, Gamaphex, Gammex, Index, Isotox, Lindafur, Lindagam, Lindagrain, Lindagranox, Lindalo, Lindamul, Lindano, Lindapoudre, Lindaterra, Novigan, Silvanol</td>
</tr>
<tr>
<td>OctaBDE</td>
<td>Companies in France, Israel, Japan,</td>
<td></td>
</tr>
<tr>
<td>Substance</td>
<td>Uses</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
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<td></td>
</tr>
<tr>
<td>Alpha HCH</td>
<td>None; waste product</td>
<td></td>
</tr>
<tr>
<td>Beta HCH</td>
<td>None; waste product</td>
<td></td>
</tr>
<tr>
<td>Chlordecone</td>
<td>Pesticide formerly used on banana root borer, fly larvicide, apple scab, powdery mildew, Colorado potato beetle, rust mite, wireworm, and household ant and roach traps.</td>
<td></td>
</tr>
<tr>
<td>Endosulfan</td>
<td>Insecticide for control of aphids, thrips, beetles, foliar feeding larvae, mites, borers, cutworms, bollworms, whiteflies, and leafhoppers. Used on cotton, tobacco, cantaloupe, tomatoes, squash, eggplant, sweet potato, broccoli, pears, pumpkins, corn, cereals, oilseeds, potatoes, tea, coffee, cacao, soybean, and other vegetables. Historically used to control termites and tsetse fly. Used in some countries in the past as a wood preservative.</td>
<td></td>
</tr>
<tr>
<td>HBB</td>
<td>Hexabromobiphenyl has been used as a fire retardant in acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business, machine housings and in industrial and electrical products and in polyurethane foam for auto upholstery.</td>
<td></td>
</tr>
<tr>
<td>Lindane</td>
<td>Lindane has been used as a broad-spectrum insecticide for seed and soil treatment, foliar applications, tree and wood treatment and against ectoparasites in both veterinary and human applications.</td>
<td></td>
</tr>
<tr>
<td>OctaBDE</td>
<td>Flame retardant primarily for ABS plastics used in office equipment and business machines. Other uses include nylon, low density polyethylene, polycarbonate, phenol-formaldehyde resins, and unsaturated polyesters.</td>
<td></td>
</tr>
<tr>
<td>PentaBDE</td>
<td>PentaBDE been used almost exclusively in the manufacture of flexible polyurethane (PUR) foam for furniture and upholstery in homes and vehicles.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Substance</th>
<th>Companies</th>
</tr>
</thead>
<tbody>
<tr>
<td>PentaBDE</td>
<td>Companies in China, EU, Israel (Dead Sea Bromine Group); Japan; and USA (Great Lakes Chemical now Chemtura)</td>
</tr>
<tr>
<td>PeCB</td>
<td>PeCB was produced intentionally to make paranitrochlorobenzene (quintozene), a pesticide. Currently, it is believed to come primarily from unintentional production from sources that include: PCBs, chlorinated solvents, pesticides, chemical manufacturing, aluminum casting, waste combustion including barrel burning, ore treatment for metal production of magnesium, copper, niobium, tantalum, titanium dioxide production, wood treatment plants, and hazardous waste incineration.</td>
</tr>
<tr>
<td>PFOS</td>
<td>Companies in Brazil (Milenia Agro Ciencias S.A.), China (Changjiang Chemical Plant), India (Indofine Chemical Co.), Italy (Miteni S.p.A., EniChem Synthesis S.p.A.), Japan (Midori Kaguka Co., Tohkem Products Corp., Tokyo Kasei Kogyo Co.), Russia (Scientific Industrial Association P &amp; M Ltd.) Switzerland (Fluka Chemical Co.), UK (BNFL Fluorochemicals Ltd., Fluorochem Ltd.), USA (3M)</td>
</tr>
<tr>
<td>SCCPs</td>
<td>Companies in Brazil, Czech Republic, Germany (Clariant, Hoechst, Huels), Japan, Slovakia, USA (Dover Chemical Corp.)</td>
</tr>
</tbody>
</table>

**USES**

Chlorowax 500C
packaging, and non-foamed PUR in casings and electronic equipment (EE). They are also used to some extent in specialized applications in textiles and in industry.

<table>
<thead>
<tr>
<th>PeCB</th>
<th>No current intentional use believed though PeCB has been found in the following uses: PCBs, dyestuff carriers, flame retardant, and pesticides (quintozene, endosulfan, chlorpyrifos-methyl, atrazine, and clopyrilid). PeCB has been used to make paranitrochlorobenzene (quintozene).</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFOS</td>
<td>PFOS uses include: fire fighting foams, carpets, leather/apparel, textiles/upholstery, paper and packaging, coatings and coating additives, industrial and household cleaning products, pesticides and other insecticides, photographic industry, photolithography and semiconductor manufacturing, hydraulic fluids, and metal plating.</td>
</tr>
<tr>
<td>SCCPs</td>
<td>SCCPs are used primarily in metalworking applications. Other uses include uses as flame retardants or plasticizers in PVC, paints, adhesives, sealants in buildings, PCB substitutes in gaskets, leather fat liquors, and flame retardants in rubber, car carpets, textiles, and other polymers. SCCPs used as flame retardants are added to rubber in a proportion of 1–10%.</td>
</tr>
</tbody>
</table>

**EFFECTS**

<table>
<thead>
<tr>
<th>Substance</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha HCH</td>
<td>Alpha-HCH has been shown to be neurotoxic, hepatotoxic, and to cause immunosuppressive effects and cancer in laboratory animals. Several epidemiological studies indicate that alpha-HCH might play a role in human breast cancer.</td>
</tr>
<tr>
<td>Beta HCH</td>
<td>Toxicological studies with beta-HCH have demonstrated neurotoxicity and hepatotoxicity. Also reproductive and immunosuppressive effects and effects on fertility were seen in laboratory animals. Several epidemiological studies indicate that beta-HCH might play a role in human breast cancer.</td>
</tr>
<tr>
<td>Chlordecone</td>
<td>The pesticide is both acutely and chronically toxic, producing neurotoxicity, immunotoxicity, reproductive, musculoskeletal and liver toxicity. Chlordecone is very toxic to aquatic organisms, with the most sensitive group being the invertebrates.</td>
</tr>
<tr>
<td>Endosulfan</td>
<td>Excessive and improper application and handling of endosulfan have been linked to congenital physical disorders, mental retardations and deaths in farm workers and villagers in developing countries in Africa, southern Asia and Latin America. Endosulfan was found among the most frequently reported intoxication incidents, adding unintentionally further evidence to its high toxicity for humans. In laboratory animals, endosulfan produces neurotoxicity effects, which are believed to result from over-stimulation of the central nervous system. It can also cause haematological effects and nephrotoxicity. Recent literature has indicated the potential for endosulfan to cause impaired development in amphibians, reduced cortisol secretion in fish, impaired development of the genital tract in birds and hormone levels, testicular atrophy and reduced sperm production in mammals.</td>
</tr>
<tr>
<td>HBB</td>
<td>Hepatotoxicity, effects on the thyroid, and endocrine disruption including effects on reproductive capacity in rats, mink and monkeys. There is epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer in exposed women.</td>
</tr>
<tr>
<td>Lindane</td>
<td>Hepatotoxic, immunotoxic, reproductive and developmental effects have</td>
</tr>
</tbody>
</table>

13
been reported for lindane in laboratory animals. The most commonly reported effects associated with oral exposure to gamma-HCH are neurological including seizures and convulsions in individuals who have accidentally or intentionally ingested lindane in insecticide pellets, liquid scabicide or contaminated food.

OctaBDE
Unfortunately, the available information on the toxicity and ecotoxicity of hexa to nonaBDE [which make up commercial OctaBDE] is very limited. Effects on mammals and birds include slight fetotoxicity, increased liver weights, and delayed skeletal ossification. Other observed effects include immunotoxicity and neurotoxicity. There is an increasing evidence suggesting similar toxicological profiles and therefore, equivalent hazards and concerns, between PBDEs and PCBs.

PentaBDE
Toxicological studies have demonstrated reproductive toxicity, neurodevelopmental toxicity and effects on thyroid hormones in aquatic organisms and in mammals. Information is lacking on the effects in humans of short-term and long-term exposure, although it is to be expected that vulnerable groups can be pregnant women, embryos and infants.

PeCB
PeCB is moderately toxic to humans. Animal studies reveal effects including decreased thyroxin, abnormal sperm, and histopathological effects on the kidneys. Pentachlorobenzene is very toxic to aquatic organisms and may cause long-term adverse effects in the aquatic environment.

PFOS
PFOS has demonstrated toxicity towards mammals in sub-chronic repeated dose studies at low concentrations, as well as rat reproductive toxicity with mortality of pups occurring shortly after birth. Environmental toxicity data for PFOS is predominantly found for aquatic organisms such as fish, invertebrates and algae, and for birds. PFOS is toxic to aquatic organisms with mysid shrimp and Chironomus tentans being the most sensitive organisms.

SCCPs
SCCPs can harm sensitive aquatic organisms at relatively low concentrations (i.e. below threshold criteria of 1 mg/L used to categorize substances on Canada’s Domestic Substances List). SCCPs affect the liver, kidney and thyroid in rats including increased liver, weight, altered liver enzymes, and enlarged thyroid. Rodent studies showed dose related increases in adenomas and carcinomas in the liver, thyroid, and kidney. There continues to be contention over the mechanisms of these tumors and whether they are relevant for human health. SCCPs were classified as a group 2B carcinogen (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC). There are no data on fertility or developmental effects for humans.

REFERENCES

<table>
<thead>
<tr>
<th>Substance</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha HCH</td>
<td>Draft Risk Profile May 2007</td>
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<tr>
<td></td>
<td><a href="http://www.pops.int/documents/meetings/poprc/drp/drp/DraftRiskProfile_a-">http://www.pops.int/documents/meetings/poprc/drp/drp/DraftRiskProfile_a-</a></td>
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<td>HCH.pdf</td>
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<td>Beta HCH</td>
<td>Draft Risk Profile May 2007</td>
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<td><a href="http://www.pops.int/documents/meetings/poprc/drp/drp/DraftRiskProfile_b-">http://www.pops.int/documents/meetings/poprc/drp/drp/DraftRiskProfile_b-</a></td>
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<td>HCH.pdf</td>
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<td>Chlordecone</td>
<td>Draft Risk Management Evaluation May 2007</td>
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<td><a href="http://www.pops.int/documents/meetings/poprc/drme/DraftRME_Chlordecone.pd">http://www.pops.int/documents/meetings/poprc/drme/DraftRME_Chlordecone.pd</a></td>
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<td>Substance</td>
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<td>--------------------------------------------------</td>
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<tr>
<td>Endosulfan</td>
<td>Proposal by the European Union August 2007</td>
</tr>
</tbody>
</table>
### B.1. Alpha-hexachlorocyclohexane – SUMMARY

**SUMMARY**

1. **Alpha Hexachlorocyclohexane (Alpha HCH)**

   Draft Risk Profile May 2007

<table>
<thead>
<tr>
<th>Composition</th>
<th>One of 5 stable HCH isomers in technical HCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Alpha-HCH by itself is neither intentionally produced nor placed on the market but produced as the main constituent of technical HCH which is used as organochlorine insecticide or chemical intermediate to manufacture enriched gamma-HCH (Lindane).</td>
</tr>
<tr>
<td>Releases</td>
<td>Historically, alpha-HCH was released during the manufacture of technical HCH and its use as a pesticide. Li and Macdonald (2005) estimated the global usage of alpha-HCH (based on data on technical HCH) at 6 millions tons, with 4.3 millions tons emitted into the atmosphere. Releases of alpha-HCH into the environment are also possible from hazardous waste sites (USEPA, 2006), stockpiles and residues of Lindane production, which are not always controlled or maintained safely (IHPA, 2006). Also, contaminated sites (e.g. from former production plants) may contribute to the environmental burden of alpha-HCH (Concha-Grana et al., 2006). Though no quantitative estimates of these releases exist, the amounts of HCH residuals in the form of by-products from Lindane production are assumed to range between 1.6 - 1.9 to 4.8 million tons.</td>
</tr>
<tr>
<td>Fate</td>
<td>Degradation is very slow especially at lower temperatures. Half-lives for alpha-HCH in Arctic lakes were up to 1.4 years, whereas in the Eastern Arctic Ocean enantioselective degradation resulted in a range of approximately 5 to 17 years. High levels are found in Arctic biota because of the bioaccumulation potential of alpha-HCH (as a product of bioconcentration and biomagnification) and the historically particularly efficient deposition processes of this substance in the Arctic waters. The efficient accumulation is an effect of the combination of the physico-chemical properties of alpha-HCH and the low temperature in the Arctic. In other words, alpha-HCH effectively accumulates in the Arctic ecosystem as a whole.</td>
</tr>
<tr>
<td>Effects</td>
<td>Alpha-HCH has been shown to be neurotoxic, hepatotoxic, and to cause immunosuppressive effects and cancer in laboratory animals. The International Agency for Research on Cancer (IARC) has classified alpha-HCH in group 2B, possibly carcinogenic to humans. Several epidemiological studies indicate that alpha-HCH might play a role in human breast cancer. Alpha-HCH is a known tumour promoting agent. Alpha-HCH may adversely affect human health in contaminated areas as well as in Arctic regions. Based on the available toxicity data of alpha-HCH, it can be concluded that current concentrations of alpha-HCH in food and human breast milk are a matter of concern. The estimated daily intake of alpha-HCH of Arctic indigenous people exceeds safe intake reference values, even though estimation is very conservative. Compared with a general accepted risk of one case per million, this risk seems unacceptably high. Nevertheless it should be emphasized that traditional foods have unique social, cultural, spiritual and economic value and therefore it is strongly recommended to avoid alpha-HCH levels of concern. Human exposure to</td>
</tr>
</tbody>
</table>
alpha-HCH results mostly from ingestion of contaminated plants, animals and animal products. Monitoring data from a wide range of biota including humans suggest that significant uptake from the environment occurs, which demonstrates the bioavailability of alpha-HCH. Monitoring data show its ubiquitous distribution in all environmental media.

<table>
<thead>
<tr>
<th>Exposure</th>
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<tbody>
<tr>
<td>Human exposure to alpha-HCH results mostly from ingestion of contaminated plants, animals and animal products. Monitoring data from a wide range of biota including humans suggest that significant uptake from the environment occurs, which demonstrates the bioavailability of alpha-HCH. Monitoring data show its ubiquitous distribution in all environmental media.</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Status</th>
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<tbody>
<tr>
<td>Technical HCH is listed in Annex II of the 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) under the Convention on Long-Range Transboundary Air Pollution which restricted alpha-HCH use to an intermediate in chemical manufacturing only. HCH (mixed isomers) is subject to the PIC Procedure of the Rotterdam Convention and is listed in Annex III of the Convention. In the European Union, the production and use of technical HCH as an intermediate in chemical manufacturing will be phased out by the end of 2007 at the latest (Regulation (EC) No 850/2004). HCHs are also among the priority substances (Decision No 2455/2001/EC) of the adopted EU Water Framework Directive 2000/60/EC. Hexachlorocyclohexane isomers, including the alpha-isomer, are on the List of Chemicals for Priority Action under the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic.</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Alternatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Will be discussed together with Lindane in Annex F evaluation if Alpha HCH advances.</td>
</tr>
</tbody>
</table>
Candidate for POPs List

$\alpha$-HCH

(alpha-hexachlorocyclohexane)
Background

The 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) addresses technical hexachlorocyclohexane (HCH, a mixture of isomers) as a substance for restriction on use under Annex II. The Aarhus Protocol is one of the protocols under the United Nations Economic Commission for Europe (UNECE) convention on Long Range Transboundary Air Pollution (LRTAP). The objective of the UNECE regional Protocol is to control, reduce or eliminate discharges, emissions and losses of persistent organic pollutants.

The Rotterdam Convention on the Prior Informed Consent also includes technical HCH, indicating that several countries have banned or severely restricted import and use of this mixture of isomers. The objective of this convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm.

Mexico proposed on June 29, 2005 that gamma-hexachlorocyclohexane (Lindane) be added to Annex A of the Stockholm Convention. The proposal presented data on the gamma isomer but mentioned as well that "other isomers of hexachlorocyclohexane should also be considered.

The POPs Review Committee (POPRC) evaluated Annex D information for Lindane at its first meeting, held in Geneva in November 2005, and decided that "the screening criteria have been fulfilled for Lindane". The Committee agreed that alpha and beta isomers could be included in the discussions, although any decision to propose inclusion of the chemical in the Convention would apply only to Lindane, the gamma isomer. As a consequence, Mexico is now proposing that alpha-HCH (and beta-HCH in another proposal) be added to Annexes A, B and/or C of the Convention to ensure that the global impacts of all three environmentally significant HCH isomers (alpha, beta and gamma) are addressed.

Data sources

d) These reviews and other references serve as a source of further information.

Introduction

Alpha-HCH is one of the five stable isomers of technical HCH, an organochlorine pesticide formerly used in agriculture. The modes of action of the HCH isomers differ quantitatively and qualitatively with regard to their biological activity in the central nervous system as the main target organ. Alpha-HCH is mainly stimulating to the central nervous system, but the final effect of the mixed isomers depends on the composition (IPCS, 2001). In general, HCHs are among the most studied pesticides with respect to their environmental fate and effects (Breivik et al., 1999).

1 Identification of the chemical

1.1 Names and registry numbers

Chemical name: alpha-hexachlorocyclohexane (alpha-HCH)

IUPAC name: (1a,2a,3b,4a,5b,6b)-Hexachlorocyclohexane

Synonym: 1,2,3,4,5,6-hexachlorocyclohexane, alpha isomer, (1alpha,2alpha,3beta,4alpha,5beta,6beta)-1,2,3,4,5,6-hexachlorocyclohexane, alpha-1,2,3,4,5,6-
Hexachlorocyclohexane; alpha-benzene hexachloride, alpha-BHC, alpha-HCH, alpha-lindane; benzene-trans-hexachloride, Hexachlorocyclohexane-Alpha (Chemfinder, 2007)

CAS number:

1.2 Chemical Structure

Alpha-HCH is a brownish to white crystalline solid (ATSDR, 2005). Alpha-HCH is the only chiral isomer of the eight isomers of 1,2,3,4,5,6-HCH. The configurations of its enantiomers are shown in Figure 1.

Figure1: Structure of alpha-HCH, modified from Buser et al. (1995)

![Chemical structure of alpha-HCH](image)

Chemical formula: $C_6H_6Cl_6$

Molecular weight: 290.83

Stability and persistence of HCH isomers are attributed to the orientation of the chlorine atoms on the molecule. Axial chlorine atoms may probably provide available sites for enzymatic degradation. Alpha-HCH exhibits 4 axially and 2 equatorially orientated chlorine atoms. Thus it is thought that the molecule is more susceptible to degradation than the beta-isomer (Philips et al., 2005).

1.3. Physico-chemical properties

The physico-chemical properties (e.g. semi-volatility, see table 1 for selected properties) of alpha-HCH allow for long-range transport and “cold condensation”, an enrichment of the substance in cold climates compared to concentrations near sources, on altitudinal and latitudinal scales described by Wania and Mackay (1996). Alpha-HCH can volatilize due to its vapour pressure and low octanol-air partition coefficient from soil surfaces. The Henry’s law constant is relatively low and decreases with temperature.

<table>
<thead>
<tr>
<th>Table 1: Selected physico-chemical properties</th>
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</thead>
<tbody>
<tr>
<td>Melting Point (°C)</td>
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<tr>
<td>Boiling Point (°C)</td>
</tr>
<tr>
<td>Density (g cm$^{-3}$ at 19 °C)</td>
</tr>
<tr>
<td>Water solubility (mg/l at 25 °C)</td>
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<tr>
<td>Vapour pressure (mmHg at 25 °C)</td>
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<tr>
<td>Henry’s law constant (atm m³ mol⁻¹)</td>
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<td>-------------</td>
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<tr>
<td>Log Kow</td>
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<tr>
<td>Log Koa (25°C)</td>
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<tr>
<td>Physical state</td>
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</table>

1. ATSDR (2005)
2. SRC PhysProp Database (2007)

1.4. Chemical production

HCH isomers are produced as a result of the photochemical chlorination of benzene during the manufacture of technical HCH, which has been widely used as a commercial pesticide. Technical HCH is a mixture of five HCH isomers: alpha-HCH (53-70%), beta-HCH (3-14%), gamma-HCH (11-18%), delta-HCH (6-10%) and epsilon-HCH (3-5%).

As the gamma-HCH isomer, also known as Lindane, is the isomer with the highest pesticidal activity, technical-HCH is subject to subsequent treatment (fractional crystallization and concentration) to produce 99% Lindane. This process is extremely inefficient with only a 10-15% yield, producing 6-10 tons of other isomers for each ton of Lindane (IHPA, 2006). Alpha-HCH is the major by-product of the reaction (60-70%), followed by beta-HCH (7-10%) (WHO, 1991).

2. Persistence

The most common HCH isomers found in the environment are alpha-, beta- and gamma-HCH. Alpha-HCH is the predominant isomer in ambient air and in ocean water (Walker, 1999).

Alpha-HCH is stable to light, high temperatures, hot water and acid but it can be dechlorinated at high pH. At pH 8 and 5°C, the estimated hydrolytic half-life of alpha-HCH is 26 years (Willet, 1998). The hydrolysis rates were found to be slower at lower temperatures with an estimated half-life for alpha-HCH of 63 years at pH 8 and 0°C (USEPA, 2006). Other studies estimated half-lives in eastern Arctic Ocean water of 6 years for (+) enantiomer and 23 years for (-) enantiomer of alpha-HCH. The half-lives of (+) and (-) enantiomers of alpha-HCH in a small Arctic lake were also estimated to be 0.6 and 1.4 years respectively (ATSDR, 2005).

Direct photolysis in the atmosphere is not expected to be an important environmental fate process for HCH. However, some authors have reported a photodegradation half-life of 91 hours for thin films of alpha-HCH. It has also been found that alpha-HCH is degraded in the atmosphere by reacting with photochemically produced hydroxyl radicals. Using an average hydroxyl radical concentration of 5x10⁵ molecule/cm³, the calculated half-life is about 115 days. In locations where the atmospheric hydroxyl radical concentration is very low, the average half-life of alpha-HCH has been estimated to be about 3 to 4 years (ATSDR, 2005).

Alpha-HCH also tends to associate with soils and sediments because of its low polarity. Biodegradation in soils of alpha-HCH has also been studied showing half-lives of 54.4 days for cropped plots and 56.1 days for uncropped plots (ATSDR, 2005). Another laboratory study reported half-lives of 125 and 48 days under aerobic and anaerobic conditions, respectively. A field experiment carried out in 1988 using soil treated with technical HCH revealed that although the concentration of alpha-HCH was the highest of the HCH isomers, the alpha-isomer disappeared more rapidly (WHO, 1991).
Bioaccumulation

The log octanol-water partition coefficient (log Kow) for alpha-HCH is 3.8, indicating that it has the potential to bioaccumulate. A wide range of bioaccumulation factors (BAF) for alpha-HCH has been reported in several studies.

Bioconcentration factors of 1 500 – 2 700 on a dry-weight basis, and 12 000 on a lipid basis, have been reported for microorganisms. Studies in invertebrates show bioconcentration factors ranging from 60 – 2 750 on a dry weight basis and up to 8 000 on a lipid basis. Other studies report bioconcentration factors in fish from 313 to 1 216 (WHO, 1991). A bioconcentration factor (BCF) of 1 100 was found using zebra-fish under steady-state conditions by Butte et al. (1991). Oliver et al. (1995) have reported bioconcentration factors ranging from 1 600 to 2 400 in a variety of aquatic organisms.

Potential for long range environmental transport

Many studies have reported alpha and gamma HCH throughout North America, the Arctic, Southern Asia, the Western Pacific, and Antarctica. HCH isomers are the most abundant and persistent organochlorine insecticide contaminants in the Arctic, and their presence in the Arctic and in the Antarctic, where they have not been used or produced, is evidence of their long-range transport.

There are observations that suggest that alpha-HCH and other HCH isomers are subject to “global distillation”, in which warm climates at lower latitudes favor evaporation into the atmosphere enabling the chemicals to be carried to higher latitudes. At high latitudes, cold temperatures favor deposition. This latitudinal gradient was found to be more striking for alpha-HCH in seawater (Walker, 1999).

Other explanations have been suggested for the abundance of alpha-HCH in the environment, i.e., the conversion of gamma-HCH into alpha-HCH through isomerization. Laboratory research indicates that photo- and bio-isomerization of gamma-HCH can occur, but field studies have not found evidence that these processes are the main sources of accumulated alpha-HCH in the environment (Walker, 1999).

Because air-water partitioning for alpha-HCH favors the water phase, especially for cold water, alpha-HCH could be moved northwards by air, accumulated in the water and slowly build into a large reservoir in the Arctic Ocean (Li et al, 2002). It has been found that alpha-HCH has a longer atmospheric lifetime by approximately 25% than gamma-HCH (Willet, 1998).

Adverse effects

No specific studies are available on the effects of alpha-HCH on humans. Oral LD50 values in rats have been found to range from 500 to 4 674 mg/kg bodyweight (WHO, 1991).

Liver and kidney damage as well as a significant decrease in body weight gain have been reported in animals fed alpha-HCH. Neurological effects have not been seen in animals treated with alpha-HCH. Genotoxicity data indicate that alpha-HCH has some genotoxic potential but the evidence for this is not conclusive (USEPA, 2006). Alpha-HCH has recently been shown to disrupt endocrine processes (Li et al, 2002).

Alpha-HCH appears to be carcinogenic in mice and rats following subchronic and/or chronic exposure (USEPA, 2006). The International Agency for Research on Cancer (IARC) has classified alpha HCH as a possible human carcinogen (ATSDR, 2005).
**Reasons for Concern**

Alpha-HCH is the most frequent isomer found in environmental compartments. Due to its physicochemical properties it has the potential to be transported long distances and it is persistent in the environment. Its proven carcinogenic potential should be of special concern.

Even though most countries have banned or restricted the use of technical HCH as a pesticide, replacing it in most cases by the use of Lindane (99% gamma-HCH), the production process to obtain a ton of pure gamma-HCH yields 6 – 10 metric tonnes of the other isomers that must be disposed of or otherwise managed. Up to 70% of these waste isomers is alpha-HCH. As Lindane is the only isomer in the mixture that has insecticidal properties, there is very limited to no commercial value for the other isomers obtained. Because of this waste isomer problem, the production of HCH/Lindane has been a worldwide problem for years.

Other HCH isomers, like alpha-HCH, can be as toxic and persistent a contaminant as Lindane, or even more so. The continued use of Lindane in the world is causing this important pollution source. Global action is therefore needed to halt the pollution caused worldwide by Lindane production.
RISK PROFILE

1. EXECUTIVE SUMMARY

After almost forty years of extensive use worldwide, there has been a gradual replacement of technical hexachlorocyclohexane (HCH) by lindane (gamma-HCH). No significant uses of technical HCH have been reported after 2000. However, releases into the environment may also occur from lindane production as well as from hazardous waste sites, landfills and contaminated sites. Because of its hazard profile and widespread abundance, technical HCH (including alpha-HCH as the main isomer) is subject to national and international regulations and prohibitions.

Alpha-HCH is susceptible to abiotic and biotic degradation at variable rates and degrees, depending on e.g. environmental media, site and climate. Alpha-HCH is expected to rapidly degrade in tropical conditions, whereas it accumulated in colder climates. Alpha-HCH is moderately persistent in soil. Based on values from aquatic compartments i.e. Arctic freshwater and sea water, it can be concluded that alpha-HCH shows high persistence in water in colder regions.

The physico-chemical properties of alpha-HCH allow the dispersal of the substance from its sources to the Arctic by a combination of long-range atmospheric transport and ocean currents. High levels of alpha-HCH have been detected in the Arctic Ocean, where it has built a large reservoir and is present in marine as well as in terrestrial species.

Alpha-HCH exposure levels in local areas have declined after worldwide prohibitions and restrictions. However regions with recent exposure and/or high pollution can still show elevated levels. A special concern also arises from exposure of hazardous waste sites and dumping grounds from disposed alpha-HCH residues from lindane production. Due to its persistence alpha-HCH can still be detected regularly at low background levels in the environment. Elevated levels have also been reported from the Arctic (levels in the Arctic Ocean are higher than in temperate oceans and lakes). Though alpha-HCH levels in air decreased more than twenty-fold from the 1980s onwards, there has been only a modest change in higher marine and terrestrial predators e.g. fur seals or polar bears.

Because alpha-HCH is present in the terrestrial and aquatic food chain, alpha-HCH may bioaccumulate and biomagnify in biota and Arctic food webs. The biomagnification factors (predator-prey comparison) for many of the examined species are > 1. Some animals, especially birds, but also mammals, have the potential to metabolize alpha-HCH. As this is an enantioselective biotransformation, a distinctive accumulation of (+) or (-) alpha-HCH can occur in mammals (depending on the species).

Alpha-HCH is the isomer with the highest neurotoxic potential beside gamma-HCH. Alpha-HCH has been classified as possibly carcinogenic to humans (group 2B) by the International Agency for Research on Cancer (IARC), based on inadequate evidence of carcinogenicity in humans and sufficient evidence for carcinogenicity to animals. Alpha-HCH causes liver hyperplasia and liver tumours in (laboratory) rodents. From animal experiments it is known that alpha-HCH affects the immune system; immunosuppressive effects were observed in humans exposed to technical HCH as well. Epidemiological studies indicate an elevated incidence of breast cancer after exposure to alpha-HCH as well as hormonal disorders leading to infertility and abortions. A possible association with intrauterine growth retardation and aplastic anaemia has been postulated.

Based on the hazard profile and the exposure scenarios it can be concluded that alpha-HCH may adversely affect wildlife and human health in contaminated regions.

The United States Environmental Protection Agency (EPA) estimated, based on daily intake rates for the arctic population, elevated cancer rates, though estimates are very conservative. It has to be considered that the liver is the target organ for all HCH-isomers, thereby leaving the risk of additive effects. Moreover the indigenous Arctic population as well as wildlife are exposed to a broad range of POPs and other pollutants leading to probably additive or possibly synergistic effects which are difficult to predict. Nevertheless Arctic public health authorities believe the significant social, cultural
and economic benefits of traditional foods outweigh the risks of contaminants such as HCH at present but give another reason for the quick control and elimination of all HCH isomers from traditional foods.

For these reasons global action on alpha-HCH is warranted.

Data sources

The draft risk profile is based on the following data sources:

- Information submitted by parties and observers according to Annex E of the Convention: specific and/or scientific information: Czech Republic, Germany, International POPs Elimination Network (IPEN), Japan, Switzerland, United States of America; general information: Algeria, Crop Life International, Kingdom of Bahrain, Mauritius, Mexico, Qatar, Republic of Lithuania and Turkey. This information is available on the Convention’s website. (http://www.pops.int/documents/meetings/poprc/prepdocs/annexEsubmissions/submissions.htm)

In addition to these information sources, a literature search of public data bases was conducted. The following databases were used: ECOTOXicology database (Ecotox, http://www.epa.gov/ecotox/) Hazardous Substances Data Bank (HSDB, http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB), Pubmed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?DB=pubmed), Environmental Fate Data Base (EFDB http://www.syres.com/esc/efdb_info.htm). In general search terms include the chemical name or CAS number and/or a combination of technical terms because of the multiplicity of entries. For the same reason, specific topical and updated articles were also considered.

2. STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS

Alpha-HCH is a constituent of technical HCH, which is regulated by at least two international agreements. The first one is the 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) under the Convention on Long-Range Transboundary Air Pollution. Technical HCH is listed in Annex II of the protocol which restricted its use to an intermediate in chemical manufacturing only.

The second agreement is the Rotterdam Convention on the Prior Informed Consent (PIC) Procedure for Certain Hazardous Chemicals and Pesticides in International Trade. HCH (mixed isomers) is subject to the PIC Procedure and is listed in Annex III of the Convention.

Canada, Mexico and the United States signed the North American Regional Action Plan (NARAP) on Lindane and Other Hexachlorocyclohexane (HCH) Isomers in 2006. The goal of the NARAP is to reduce the risks associated with the exposure of humans and the environment.

In the European Union, the production and use of technical HCH as an intermediate in chemical manufacturing will be phased out by the end of 2007 at the latest (Regulation (EC) No 850/2004). HCHs are also among the priority substances (Decision No 2455/2001/EC) of the adopted EU Water Framework Directive 2000/60/EC.

Hexachlorocyclohexane isomers, including the alpha-isomer, are on the List of Chemicals for Priority Action under the OSPAR Commission for the Protection of the Marine Environment of the Northeast
Atlantic. The objective is the prevention of pollution of the maritime area by continuously reducing discharges, emissions and losses of hazardous substances.

3 SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

3.1. Sources

3.1.1. Production

Alpha-HCH by itself is neither intentionally produced nor placed on the market. It is produced as the main constituent of technical HCH which is used as organochlorine insecticide or chemical intermediate to manufacture enriched HCH (lindane). Currently no production data on technical HCH have been reported, whereas manufacture of lindane still takes place (IHPA, 2006).

HCH is manufactured by photochemical chlorination of benzene which leads to the formation of mainly five stable HCH isomers. The yields of different isomers vary due to technical differences in the production process. The reported ranges are: alpha-HCH (55 - 80%), beta-HCH (5 - 14%), gamma-HCH (8 - 15%), delta-HCH (6 - 10%) and epsilon-HCH (1 - 5%) (Breivik et al., 1999). Further details on the production and reuse of HCH residuals can be found in UNEP/POPS/POPRC.2/17/Add.4 (Risk Profile on Lindane) and IHPA (2006).

The following countries which submitted information according to Annex E stated that there was currently no production or use of alpha-HCH: Czech Republic, Germany, Mauritius, Mexico, Norway, Qatar, Republic of Lithuania, Turkey, Switzerland, and the United States of America.

3.1.2 Trade and stockpiles

Technical HCH was rapidly introduced in the 1940s on a large scale on the market, due to its universal insecticidal properties. The promising market opportunities worldwide arose in the search for an inexpensive alternative to DDT (IHPA, 2006). However, due to the decreasing effectiveness of the gamma > alpha > beta-isomer in controlling insects (Baumann et al., 1980) technical HCH was gradually replaced by lindane (> 99% gamma-HCH). However, the manufacture of lindane has resulted in a huge amount of HCH residuals, which must be disposed of or otherwise managed. IHPA (2006) calculated 1.9 to 4.8 million tons of HCH residuals based on global lindane production, in the absence of exact data. These estimates are far beyond the values reported by Walker et al. (1999) who reported stockpiles of approximately 2 785 tons of technical HCH and 45 tons of unspecified HCH material in Africa and the Near East.

3.1.3 Uses

Around 10 million tons of technical HCH were released into the environment between 1948 and 1997 (Li et al., 1999). Breivik et al. (1999) estimated technical HCH usage at approximately 400 000 tons in Europe alone between 1970 and 1996. The data illustrate the large uncertainties of these estimates.

According to Li and Macdonald (2005) global usage of technical HCH was dominated by 10 countries headed by China, which consumed almost half of the total global quantity. The other countries were (in order of decreasing usage): Former Soviet Union, India, France, Egypt, Japan, United States, East Germany, Spain and Mexico. Usage of technical HCH was banned in most western countries and Japan in the 1970s but continued in China and Russia until 1983 and 1990. In 1990, India also banned technical HCH for agricultural use but kept it for public health uses (AMAP, 2004). Technical HCH usage steadily declined and now technical HCH is virtually no longer used worldwide. However, there are indications that the use of stockpiles, limited use for public health purposes and/or illegal use cannot be excluded (Zhulidov et al., 2000; Bakore et al., 2003; Qian et al., 2006).
3.1.4 Releases to the environment

There are several pathways of alpha-HCH for entering the environment. Historically, alpha-HCH was released during the manufacture of technical HCH and its use as a pesticide. Alpha- and beta-HCH have the same global emission patterns which, however, differ in scale. Li and Macdonald (2005) estimated the global usage of alpha-HCH at 6,000 thousand tons, with 4,300 thousand tons emitted into the atmosphere. After the 1940s emissions of alpha-HCH increased and peaked in the early 1970s. Due to the ban on the use of alpha-HCH in North America, in European countries and Japan, emissions decreased but reached again a peak in the 1980s because of frequent usage in Asian countries. After the 1980s, figures dropped due to further prohibitions and restrictions e.g. in China. Releases of alpha-HCH into the environment are also possible from hazardous waste sites (USEPA, 2006), stockpiles and residues of lindane production, which are not always controlled or maintained safely (IHPA, 2006). Also, contaminated sites (e.g. from former production plants) may contribute to the environmental burden of alpha-HCH (Concha-Grana et al., 2006). Germany (submitted Annex E information) reported that there are still a few isolated local sources i.e. landfills and dumps in the former GDR (East Germany) from applications of technical HCH. As a result, higher concentrations of alpha-HCH in fish of the river Elbe near the former production site were detected after heavy rainfalls and floods in 2003. However, quantitative estimates of releases from hazardous waste sites and landfills are not available.

3.2 ENVIRONMENTAL FATE

3.2.1 Persistence

3.2.1.1 Abiotic Degradation

Alpha-HCH is, in principle, degradable in environmental compartments by abiotic processes such as photodegradation or hydrolysis. Based on laboratory experiments from Ngabe et al. (1993), hydrolytic half-lives of alpha-HCH show strong temperature dependence. At 20°C, pH 8 the DT50 was 0.8 years whereas it decreased at lower temperature (5°C, pH 7.8) to 26 years. Based on these degradation rates Harner et al. (1999) calculated a DT50 of alpha-HCH in the Arctic Ocean of 63 years.

In general, HCH-isomers do not absorb light > 290 nm. Thus it is expected that photolysis plays a minor role in the removal of alpha-HCH. Deo et al. (1991) reported half-lives of alpha-HCH in aqueous solution exposed to sunlight of 4 to 6 days. While the mechanism of this degradation is uncertain, it was shown that alpha- as well as gamma-HCH break-down by indirect photolysis with photosensitizing agents that may transfer the excitation energy to HCH (ATSDR, 2005; USEPA, 2006). Regarding photodegradation on hard surfaces, a half-life equal to 91 hours on a thin film has been reported (ATSDR, 2005). However, the relevance of this result is questionable when taking into consideration the arguments mentioned above.

The measured atmospheric OH rate constant of $1.4 \times 10^{-13}$ cm$^3$/molecule-sec resulted in a corresponding half-life of 115 days (using an average hydroxyl radical concentration of $5 \times 10^5$ molecule/cm$^3$ according to the TGD (2003)).

In conclusion, abiotic processes i.e. hydrolysis may contribute to the removal of alpha-HCH from environmental compartments. However, degradation is very slow especially at lower temperatures. Photolysis in aqueous media and air is considered to play an insignificant role in the degradation of alpha-HCH.

3.2.1.2 Biotic Degradation

Degradation of alpha-HCH has been found to take place in pure cultures, soil slurries, soil (semi-)field studies, sediment and water.
Initially it was thought that HCH biodegradation in soil occurs under anaerobic conditions. However, several investigations show that alpha-HCH is aerobically degraded, in some cases even faster than anaerobically. Breakdown was also reported for methanogenic and sulfate reducing conditions (Phillips et al., 2005).

The anaerobic metabolic pathway of alpha-HCH leads via dechlorination to tetrachlorocyclohexane. Dichlorophenol and trichlorophenol as well as chlorobenzene and benzene were formed under methanogenic conditions, the last two as stable end products. These metabolites can be further mineralised aerobically or anaerobically (Bachmann et al., 1988; Phillips et al., 2005). In pure cultures as well as in flooded soil gamma-HCH is the most easily dechlorinated isomer followed by alpha-HCH under anaerobic conditions (Jagnow et al., 1977; MacRae et al., 1967).

Under aerobic conditions alpha-HCH was dehydrochlorinated to pentachlorocyclohexane in soil slurries. Further conversion to tetrachlorobenzene or trichlorobenzene may occur to yield dichlorobenzene (Deo et al., 1994). The aerobic degradation pathway of gamma-HCH was extensively studied with Shingobium sp. and results in several metabolites. It was suggested that alpha-HCH follows the same pathway than gamma-HCH. Complete mineralization of alpha-HCH was shown in laboratory studies under aerobic conditions (Phillips et al., 2005).

In general, climatic conditions as well as soil texture and organic matter altering substance sorption, water content, pH and bacterial growth influence degradation rates (IPCS, 1992). The moisture content of the soil enhances losses of alpha-HCH, which is attributed to higher volatility and/or microbial degradation (Chessells et al., 1988; Phillips et al., 2005). Bacteria capable of degrading HCHs at extreme temperatures (≤ 5 °C or > 40°C) have not yet been reported (Phillips et al., 2005).

Data on laboratory soil studies or field investigations are limited. Under various field conditions it is assumed that degradation rates are in the order of alpha > gamma > > beta. Singh et al. (1991) reported half-lives of around 55 days on cropped and uncropped plots in a sandy loam in India under subtropical conditions. This result is consistent with findings from Kaushik (1989) who reported an even shorter half-life for technical HCH under similar study conditions. Also, in temperate climate Doelman et al. (1990) observed in a semi-field study with contaminated soil > 50 % removal after 161 days, mainly attributed to a quick decline in the first few weeks, whereas degradation slowed down afterwards. Suzuki et al. (1975) also suggested that low residue levels may resist microbial and physico-chemical action. Low concentrations of alpha-HCH may persist in the environment indefinitely because of low affinity of enzymes or transport system responsible of HCH degradation (Phillips et al., 2005). Stewart and Chisholm (1971) observed in a long-term field study after an application of technical HCH, 4 % of the alpha-isomer after 15 years in a sandy loam in Canada. In addition, Chessells et al. (1988) showed that after a 20 year application history of technical HCH on sugar cane in Queensland, Australia, alpha-HCH with the highest initial concentration is substantially less prevalent in the field and the detected levels were twice as much as the levels of the gamma-isomer.

Abiotic processes are not enantioselective, but biodegradation may be. If nonracemic alpha-HCH residues in the environment or biota are measured, enzymes are involved. However, racemic residues do not exclude the possibility of biotic degradation (cp. Suar et al., 2005). Also for monitoring purposes enantiomeric fractions (EFs, calculated by the formula EF = ER/(ER+1), ER = enantiomeric ratio: (+) /(-) alpha-HCH, Kallenborn et al., 2001), have been quantified for the characterisation of residues. Hegeman and Laane (2002) investigated the enantiomeric distribution of alpha-HCH in different environmental compartments obtained from 618 measurements. In general, the abiotic compartments showed average EFs close to 0.5. In soil, the preference tended to be the degradation of the (-) alpha-HCH (EF > 0.5), whereas in water an opposite tendency was found. Kurt-Karakus et al. (2005) reported a range of EFs for alpha-HCH of 0.4 - 0.89 (mean 0.5) in global background soils which covered a greater range than the EFs in ambient air.
of North America (0.47 - 0.52), suggesting that post-deposition degradation had taken place. However, since EFs vary considerably with site, caution is needed when using enantiomeric signatures in the air as a marker of reemissions from (soil) surfaces.

Based on the $K_{oc}$ value and confirmed by field data, alpha-HCH is expected to have a low leaching potential (HSDB, 2006; Singh et al., 1991). However, groundwater pollution may occur in highly contaminated areas (Law et al., 2004).

Alpha-HCH is able to biodegrade in sea water/sediment samples (HSDB, 2006) and freshwater (Padma and Dickhut, 2002). Helm et al. (2002) estimated the half-lives for alpha-HCH in a high Arctic lake at 0.6 to 1.4 years. For the Eastern Arctic Ocean enantioselective degradation for the (+) alpha- and (-) alpha-HCH with half-lives of 5.9 and 23.1 years was observed. If breakdown with hydrolysis was taken into consideration, the overall half-lives were 5.4 and 16.9 years for the (+) and (-) alpha-isomer respectively (Harner et al., 1999).

Though sediment degradation rates are poorly known and thus the estimates are less certain, the half-lives for alpha-HCH in sediments of a high Arctic lake were assumed to be approximately 2 years (Helm et al., 2002).

### 3.2.2 Bioaccumulation

The octanol-water partition coefficient (log $K_{ow} = 3.8$) for alpha-HCH indicates a potential for bioaccumulation (ATSDR, 2005).

A wide range of bioconcentration factors (BCFs) have been reported in several studies. For green algae, bioconcentration factors varied from about 200 in *Chlorella pyrenoidosa* cells to 2 700 (dry weight basis) and 13 000 on a lipid basis, respectively in *Dunaliella*.

Studies of invertebrates show BCFs in the range of 60 (8 000 on a lipid basis) in *Artemia* to 2 700 in polychaetes, depending on the lipid content of the animals (IPCS, 1992).

The BCF for alpha-HCH according to the former OECD test guideline 305 E in zebra fish was equal to 1 100 under steady state conditions with uptake constants (k1) of 50 and clearance rate constants (k2) of 0.045. These values are similar to those of gamma-HCH (BCF 850, k1 = 50.8, k2 = 0.055) (Butte et al., 1991). Oliver et al. (1985) reported BCFs ranging from 1 100 to 2 800 in rainbow trout.

In general, studies from Arctic marine food webs show food web magnification factors (FWMFs), which represent the mean rate of increase per trophic level in the food chain, greater than 1. The BMFs (biomagnification factor, predator-prey comparison) of alpha-HCH in zooplankton and artic cod are greater than 1, showing a potential for biomagnification. BMFs of alpha-HCH in seabirds were generally less than 1 with the exception of dovekie and black guillemot. Ringed seals showed a BMF of 2.5 (Moisey et al., 2001). It is suggested that alpha-HCH isomer has the potential to biomagnify in aquatic food webs and may increase at lower as well as in upper trophic levels, especially in marine mammals (USEPA, 2006; Hoekstra et al., 2003a). The report of Hoekstra et al. (2003b) also confirms this presumption with a BMF of 9.85 in bowheads for alpha-HCH.

Fisk et al. (2001) reported on the influence of chemical and biological factors on the trophic transfer of POPs including alpha-HCH. In general, the highest BMF should be seen in homeothermes (birds and mammals) compared to poikilothermes (fish, invertebrates) attributed to their greater energy requirements. Within the homeothermes, seabirds usually have the highest BMFs, consistent with the greater energy demand in birds. But this is not applicable for alpha-HCH. Most seabirds appear to be able to induce the cytochrome P450 such as CYP2B, which are enzymes to metabolize alpha-HCH, so the ranking from highest to lowest biotransformation ability (usually for OCs: marine mammals > seabirds > fish > zooplankton) is not applicable for
this compound. The BMF of alpha-HCH in poikilothermes is 1.3 and equal to that in homeothermes (Hop et al., 2002).

As alpha-HCH is a chiral compound, the determination of the ER or EF is important in order to understand species-specific metabolism and biotransformation. No enantioselective biotransformation in rainbow trout for alpha-HCH was observed by Konwick et al. (2006) in a dietary study showing consistent EFs in the fish. In an experiment of Wong et al. (2002) alpha-HCH was racemic throughout the course of the experiment with rainbow trout, fed with treated food. These results are in contrast with reports of enantioselective biotransformation in other species. The EF in benthic invertebrates, zooplankton and fish was 0.45 as a maximum. Ringed seals showed an EF of 0.51, while the EFs in seabirds range from 0.65 (dovekie) to 0.97 in glaucous gulls (Moisey et al., 2001). This suggests that seabirds preferentially metabolize the (-) enantiomer. Associated with a BMF of < 1 in seabirds, it has been found that both enantiomers of alpha-HCH are metabolized in birds (dovekie and black guillemot seems to have a lower capacity).

The EF of 0.51, considered together with the BMF of 2.5 in seals, indicates that mammals are not able to biotransform alpha-HCH in great amounts (Moisey et al., 2001). Nevertheless, Wiberg et al. (2000) found residues of alpha-HCH with nonracemic ERs in seals as well as in polar bears. According to Hoekstra et al. (2003b) accumulation of the (+) enantiomer occurs in bowhead whale and beluga, but (-) alpha-HCH enriches in bearded seal. Ringed seal show a slight accumulation of the (+) enantiomer (Hoekstra et al., 2003b) but sometimes the alpha-HCH residues are racemic (Fisk et al., 2002). This indicates an enantiospecific biotransformation and accumulation of alpha-HCH in the food chain. When investigating the EFs in krill, cod and penguin eggs, Corsolini et al. (2006) also found enantioselective biotransformation with an increase by 14 % of (+) alpha-HCH from the lower to the higher trophic level (from krill to penguin). There are interspecies differences in the enantiomeric profile of alpha-HCH in marine mammals, too. The BMF for calanus to bowhead for example is high (near 10 with a (+) alpha-HCH fraction of 16 and 4.5 of (-) alpha-HCH) (Hoekstra et al., 2003b).

Moisey et al. (2001) showed different BMFs in doveky, depending on the prey. Summed up, biomagnification is affected by many parameters such as contamination in biota, and consequently of food (prey), the trophic level and the ability to biotransform alpha-HCH.

Not only in the arctic food web, but also in organs of fur seals from the Pacific coast of Japan and double crested cormorants from the great lakes, alpha-HCH was detected (with an alpha-HCH ER from 1 in the muscle to 1.58 in fat). High alpha-HCH ERs were found in the brain of the cormorants (> 3.6) (Iwata et al., 1998). Willet et al. (1998) inferred from high alpha-HCH concentrations in marine mammal brain that this compound can cross the blood/brain barrier. Ulrich et al. (2001) also found in studies with rats that the alpha-HCH ER in brain, ranging from 2.8 to 13.5, is not caused by an enantioselective metabolism but that selective retention might be responsible.

Braune et al. (1999) detected alpha-HCH residues in the fat of caribous. Residues of alpha-HCH could also be found in livers and the adipose tissue of arctic foxes. The alpha-HCH ER of 2.2 in the liver, and 1.1 in the adipose tissue, indicates a stereoselective bioaccumulation also in terrestrial mammals (Klobes et al., 1997).

In conclusion high levels are found in Arctic biota because of the bioaccumulation potential of alpha-HCH (as a product of bioconcentration and biomagnification) and the historically particularly efficient deposition processes of this substance in the Arctic waters. The efficient accumulation is an effect of the combination of the physico-chemical properties of alpha-HCH and the low temperature in the Arctic. In other words, alpha-HCH effectively accumulates in the Arctic ecosystem as a whole.
3.2.3 Long range environmental transport

Monitoring data on the environment including biota from remote regions such as the Arctic or Antarctica, where technical HCH has not been used, provide evidence of the long-range transport potential of alpha-HCH.

Also the physico-chemical properties in combination with its stability allow alpha-HCH to undergo long range transport in the atmosphere. Primary emissions from the source regions (mainly in Asia) and Arctic air concentrations have synchronously decreased, suggesting a rapid dispersion of alpha-HCH from its sources to remote regions (Li and Bidleman, 2003). Especially high concentrations compared to the source regions were reported for the Arctic Ocean. It is assumed that after long range transport alpha-HCH accumulated in the cold water due to its low Henry’s law constant and built a large reservoir (Li and Macdonald, 2005). HCHs including alpha-HCH are the most abundant pesticides in the Arctic air and water (Walker, 1999).

To understand pathways and the fate of alpha-HCH in the upper Arctic Ocean, Li et al. (2004) developed an Arctic Mass Balance Box Model. They concluded that the highest load of 6.670 tonnes was reached in 1982 mainly by gas exchange and ocean currents and decreased thenceforward by an average annual rate of approximately 270 tons/year. After 1990, ocean currents become the dominant input of alpha-HCH in the Arctic Ocean. However, the portion of alpha-HCH entering the Arctic atmosphere via long range transport from source regions played a prominent role (especially in the beginning). After the early 1990s alpha-HCH in the Arctic air came from both atmospheric transport and volatilization from the Arctic Ocean. It was suggested that a complete elimination of alpha-HCH mainly by degradation and ocean currents would require another two decades. In total 27 700 tons alpha-HCH were transported between 1945 and 2000 via long range transport to the Arctic Ocean.

According to model calculations with the OECD Pov and LRTP Screening Tool alpha-HCH has similar persistence and long-range transport properties compared to already identified POPs such as PCBs and organochlorine pesticides (submitted Annex E information by Switzerland).

Model input properties of the chemicals include partition coefficients in air-water and octanol-water as well as half-lives in air, water and soil and the Henry’s Law constant (based on figures contained in UNEP/POPS/POPRC2./INF/7). The model considers all environmental compartments quantitatively. The results of the model do not indicate absolute levels in the environment but help to compare possible POPs with identified POPs (reference chemicals: PCB congeners 28, 101, 180, HCB, carbon tetrachloride and alpha-HCH) according to their environmental persistence and potential for long range transport. Uncertainties in the chemical properties were investigated by Monte Carlo uncertainty analysis.

3.3 EXPOSURE

Exposure to alpha-HCH resulted from the use of technical HCH, and from the production and manufacture of technical HCH and lindane. Because of the persistence high exposure is also expected in contaminated areas due to extensive use, former production, disposal sites and stockpiles. Though usage of technical HCH has practically ceased worldwide monitoring data based on the ratio of the alpha-/gamma-isomer still suggest possible releases of technical HCH in certain areas (Zhang et al. 2003; Qian et al., 2006; Zhulidov et al., 2000).

Human exposure to alpha-HCH results mostly from ingestion of contaminated plants, animals and animal products. Inhalation of ambient air and consumption of drinking water are further sources of exposure, although to a minor extent. As shown by a French pilot study alpha-HCH was detected in indoor air and on the hands of the general population in the Paris area in 42 and 35 % of the samples. Levels were low and ranged up to 1.8 ng/m$^3$ in air and up to 8.5 ng/hand (Bouvier et al., 2006).
Monitoring data from a wide range of biota including humans suggest that significant uptake from the environment occurs, which demonstrates the bioavailability of alpha-HCH.

3.3.1 Environmental monitoring data from local areas

Generally environmental levels in local areas have dropped after restrictions and prohibitions of the usage of technical HCH (IPCS, 1992; see also table 2). However, monitoring data show its ubiquitous distribution in all environmental media e.g. in monitoring activities in the Czech Republic, in lichens of various locations in Switzerland (values given in table 2) or in a recently performed monitoring programme in Japan. Alpha-HCH had been detected in Japan in all but 7 fish specimens. The reported values are as follows: water 0.013 - 5.7 ng/l, sediment trace - 5.7 ng/g dw (dry weight), shellfish up to 1.8 ng/g ww (wet weight), fish up to 2.9 ng/g ww, bird 0.1 - 1.6 ng/g ww, air (warm and cold season) 0.02 - 3.2 ng/m$^3$ and 0.01 - 0.68 ng/m$^3$ (submitted Annex E information by Japan).

Environmental levels can still be high in the proximity of sources. HCH concentrations in contaminated soil of 40 - 225 mg/kg were found in the topsoil around a chemical plant in Albania (UNEP, 2003). Mean levels of 0.02 mg/kg were reported for soils from the Pearl River Delta in China, Russian soils near the Lena River contained 0.001 - 0.017 mg/kg HCH (UNEP, 2003). Levels of up to 12 000 mg/kg were detected in soil of a highly polluted area in Spain (Concha-Grana et al., 2006). Levels in biota vary, depending on the location (recent usage and/or high pollution) and species. Alpha-HCH is in most cases the dominant isomer in fish (Willett et al., 1999). E.g. concentrations of HCHs (mainly the alpha-isomer) in several fish species from India ranged between 6 to 68 ng/g ww. Fish samples collected from the Nile River near Cairo in 1993 showed a concentration of alpha-HCH of 0.5 ng/g ww (UNEP, 2003).

Alpha-HCH was also determined in eggs of Dalmatian Pelican (Pelecanus crispus) as well as in eels (Anguila anguila), the main pelican prey species collected in the wetlands of Amvrakikos Gulf in Greece for a two year period, 1992 and 1993. The concentration in pelican eggs was 7.9 ± 3.2 ng/g and 6.5 ± 2.5 ng/g ww in eels (UNEP, 2003). Concentrations of alpha-HCH in perch from the Latvian cost were up to 21 ng/g lw (lipid weight) (range 50 - 60), which were considered as background load. Elevated levels of up to 126 ng/g lw were attributed to a recent discharge of technical HCH (Olsson et al., 1999).

A local source of alpha-HCH was the usage of technical HCH by indigenous human populations in the Russian North against nuisance insects parasiting on domesticated reindeer (Li et al., 2004). However, no quantitative estimates of these exposure levels exist.

3.3.2 Exposure as a result of long range environmental transport

Highest measured levels of alpha-HCH have been reported for higher latitudes in air (e.g. Svalbard, Alert) as well as in seawater (Harner et al., 1999). As shown in table 2, alpha-HCH in air (e.g. from 94 pg/m$^2$ in 1992 to 12 pg/m$^2$ in 2003 in Norway) has decreased. AMAP (2004) also summarized that concentrations of HCHs in Arctic air have been low since the mid 1990s due to worldwide prohibitions and restrictions. Before, in the 1980s, levels as high as approximately 900 pg/m$^2$ were measured in Arctic air (Li et al., 2002). Seawater levels in the eastern Arctic Ocean were generally lower than in the western part (Harrer et al., 1999). Surface concentrations are highest in the Central Canadian Arctic Archipelago, intermediate in the Beaufort/Chukchi Seas and at the North Pole. In the 1990s levels in the Canadian Arctic Ocean were higher than anywhere else in the global marine environment (AMAP, 2004).
This spatial distribution is also reflected in the levels in biota. Hoekstra et al. (2002) found that bowhead whales exhibit a reversal in their blubber alpha-beta-HCH ratios on their migration route between the Bering to the Beaufort Sea. Levels in beluga blubber decreased from approximately 190 to 140 ng/g lw between 1982 and 1997 in the southeast Baffin Bay (AMAP, 2004). Levels of up to 196 ng/g ww were reported from Alaska (submitted Annex E information by IPEN) and of up to 344 ng/g ww from Arviat (Stern et al., 2005). Minke whales from Greenland had higher concentrations of the prevalent alpha-isomer in blubber (mean levels 40 - 55 ng/g ww), than individuals from the North Sea (below 30 ng/g) (AMAP, 2004). No decline of ΣHCHs in blubber of narwhal from the Canadian Arctic was observed between 1982 and 1999.

Concentrations in ringed seal of the Canadian Arctic showed no significant change of ΣHCHs concentration from the 1970s. The elevated residues of HCH isomers in marine mammals of the Canadian Archipelago are likely due to the high concentrations of HCH isomers in the water because HCH isomers are the most abundant organochlorines in the Arctic Ocean (NARAP, 2006).

No temporal trend for Arctic cod and dab from the costal waters of Iceland was found for the period from 1991 to 2000, whereas results from Norway revealed a significant decrease (from 23 to 4 ng/g lw) of alpha-HCH residues in Arctic cod liver between 1987 to 1998 (Sinkkonen and Paasivirta, 2000).

Alpha-HCH has been detected in the muscle and liver of Arctic foxes (1.5 and 3 ng/g ww) in Canada (AMAP, 2004). Levels in polar bear also reflect the spatial distribution of alpha-HCH being highest in Alaskan populations (in male polar bear up to 593 ng/g lw). No decline of alpha-HCH levels were reported for female polar bears in the Western Hudson Bay (concentrations up to 260 ng/g lw) from 1991 – 2002 (Verreault et al., 2005). Residues of alpha-HCH in East Greenland polar bears increased from 18 - 25 % during the 1990s (AMAP, 2004).

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**Table 2: Selected monitoring data of abiotic compartments and vegetation**

<table>
<thead>
<tr>
<th>Compart-ment</th>
<th>Country/region</th>
<th>Levels</th>
<th>Comments</th>
<th>References</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>Great Lakes, rural</td>
<td>&lt; 1 - 84 pg/m³</td>
<td>alpha-HCH, mean values, gas phase</td>
<td>Sun et al., 2006b</td>
<td>1992-2003</td>
</tr>
<tr>
<td></td>
<td>Great Lakes, Chicago</td>
<td>52 pg/m³</td>
<td>alpha-HCH, mean value, gas phase</td>
<td>Sun et al., 2006b</td>
<td>1996-2003</td>
</tr>
<tr>
<td></td>
<td>Niigata, Japan</td>
<td>92 pg/m³</td>
<td>Annual average, according to the authors a result of long-range transport</td>
<td>Murayama et al., 2003</td>
<td>2000-2001</td>
</tr>
<tr>
<td></td>
<td>Czech Republic (Kosetice)</td>
<td>38/21/17/22/13 pg/m³</td>
<td>Air and aerosol, annual mean concentrations</td>
<td>EMEP measurement, data online</td>
<td>1999-2003</td>
</tr>
<tr>
<td></td>
<td>Finland (Pallas)</td>
<td>24/28/18/15/17/18/9 pg/m³</td>
<td>Air and aerosol, annual mean concentrations</td>
<td>EMEP measurement, data online</td>
<td>1996-2003</td>
</tr>
<tr>
<td></td>
<td>Iceland (Storhofdi)</td>
<td>17/16/15/10/8/10/5/7 pg/m³</td>
<td>Air and aerosol, annual mean concentrations</td>
<td>EMEP measurement, data online</td>
<td>1995-2003</td>
</tr>
<tr>
<td></td>
<td>Norway (Lista)</td>
<td>94/94/76/69/52/61/50/37/25/19/17/17/12 pg/m³</td>
<td>Air and aerosol, annual mean concentrations</td>
<td>EMEP measurement, data online</td>
<td>1991-2003</td>
</tr>
<tr>
<td></td>
<td>Sweden (Aspvreten)</td>
<td>43/37/61/50/67/16 pg/m³</td>
<td>Air and aerosol, annual mean concentrations</td>
<td>EMEP measurement, data online</td>
<td>1995-2002</td>
</tr>
<tr>
<td></td>
<td>Ny-Aslund (Svalbard,</td>
<td>73 pg/m³</td>
<td>ΣHCHs, mostly alpha-HCH, highest</td>
<td>AMAP, 2004</td>
<td>1996-1988</td>
</tr>
<tr>
<td>Compart-ment</td>
<td>Country/region</td>
<td>Levels</td>
<td>Comments</td>
<td>References</td>
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<tr>
<td>Norway)</td>
<td>Barents Sea and eastern Arctic Ocean</td>
<td>11 - 68 pg/m(^3)</td>
<td>annual average value reported in 1996</td>
<td>Harmer et al. (1999)</td>
<td>1999</td>
</tr>
<tr>
<td>Arctic</td>
<td>23 +/- 10 pg/m(^3)</td>
<td>Uniform distribution, arithmetic mean, measurements from 4 Arctic sites</td>
<td>Su et al., 2006</td>
<td>2000-2003</td>
<td></td>
</tr>
<tr>
<td>Precipitation</td>
<td>Belgium (Knokke)</td>
<td>4.1 - 0.5 ng/l</td>
<td>annual mean concentrations</td>
<td>EMEP measurement data online</td>
<td>1996-2003</td>
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<td></td>
<td>Germany (Zingst)</td>
<td>1 - 0.3 ng/l</td>
<td>annual mean concentrations</td>
<td>EMEP measurement data online</td>
<td>1999-2003</td>
</tr>
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<td></td>
<td>Finland (Pallas)</td>
<td>&lt; 1 ng/l</td>
<td>precipitation + dry deposition annual mean concentrations</td>
<td>EMEP measurement data online</td>
<td>1996-2003</td>
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<td></td>
<td>Norway (Lista)</td>
<td>2.7 - 0.4 ng/l</td>
<td>annual mean concentrations</td>
<td>EMEP measurement data online</td>
<td>1991-2003</td>
</tr>
<tr>
<td></td>
<td>Sweden (Aspvreten)</td>
<td>2.7 - 0.4 ng/l</td>
<td>annual mean concentrations</td>
<td>EMEP measurement data online</td>
<td>1995-2002</td>
</tr>
<tr>
<td></td>
<td>Canada/Great Lakes</td>
<td>1 - 40 ng/L</td>
<td>81 samples</td>
<td>IPCS, 1992</td>
<td>1976-77</td>
</tr>
<tr>
<td>Soil</td>
<td>Russian Arctic</td>
<td>0.2 - 0.5 ng/g dw</td>
<td>(\Sigma) HCHs, predominantly alpha-HCH, soil including peat and litter</td>
<td>AMAP, 2004</td>
<td>2000-2001</td>
</tr>
<tr>
<td></td>
<td>Antarctica</td>
<td>&lt; 0.01 - 0.026 ng/g dw</td>
<td></td>
<td>Borghini et al., 2005</td>
<td>1999</td>
</tr>
<tr>
<td>Seawater</td>
<td>Northern Barents Sea, Eastern Arctic Ocean</td>
<td>910 (350 - 1630) pg/l</td>
<td>Sample period: July-September</td>
<td>Harmer et al., 1999</td>
<td>1996</td>
</tr>
<tr>
<td></td>
<td>North American Arctic Ocean</td>
<td>~ 7.5 µg/m(^3)</td>
<td></td>
<td>Li and Macdonald, 2005</td>
<td>1983</td>
</tr>
<tr>
<td></td>
<td>Canadian Archipelago and southern Beaufort Sea</td>
<td>3.5 (1.1 - 5.4) ng/L</td>
<td>Surface water, measurements in summer</td>
<td>Bidleman et al., 2007</td>
<td>1999</td>
</tr>
<tr>
<td>Freshwater, rivers</td>
<td>Russian north rivers</td>
<td>&lt; 1 - 69 ng/l</td>
<td>Seven-year weighted mean concentrations</td>
<td>AMAP, 2004</td>
<td>1190-1996</td>
</tr>
<tr>
<td>River and estuarine waters</td>
<td>Eastern and southern Asia and Oceania</td>
<td>up to max. 470 ng/l</td>
<td></td>
<td>Iwata et al., 1994</td>
<td>1989-1991</td>
</tr>
<tr>
<td>Sediment (Lake)</td>
<td>Southern Sweden</td>
<td>9.2 ± 6.3 ng/g dw</td>
<td>(\Sigma) HCHs, data from the Swedish Monitoring Program, 2002</td>
<td>AMAP, 2004</td>
<td>2002</td>
</tr>
</tbody>
</table>
| Vegetation | Taymir | 7 ng/g dw | Highest | AMAP, 2004 | 1991-
### 3.3.3 Food

Daily intake values of alpha-HCH for the general population in adult diets between 1986 and 1991 in the United States were reported to be 0.008 µg/kg. In the Total Diet Study conducted by FDA in 1990 on 935 food items, alpha-HCH was detected in 11 items (~1%). The average concentration of alpha-HCH in 234 ready-to-eat foods was 0.0010 µg/kg (no information on which basis the concentrations are expressed, ATDSR, 2005). In the USA, the age dependent average daily intake of alpha-HCH declined from 3.3 – 16.1 ng/kg bodyweight (bw; 1982 – 84) to 0.5 – 2.7 ng/kg bw (1986 – 91). In a Total Diet Study from Canada (1993 – 96), an average daily dietary intake of 0.37 ng/kg bw alpha-HCH was reported (Health Canada, 2003, in EFSA, 2006). Within the European countries, representative dietary intake studies are scarce. One was performed in the Czech Republic. The median daily intake values for alpha-HCH declined from 4.3 ng/kg bw in 1994 to 1.6 ng/kg bw in 2002 (EFSA, 2005). A local diet study carried out in Spain in the years 1990/91 estimated daily intakes below 0.1 µg alpha-HCH (Urieta et al., 1996).

Alpha-HCH has been found in cow’s milk in countries where HCH had been used recently. Mean levels of alpha HCH in cow’s milk of two different regions in India were 0.012 mg/kg lipid and 0.0045 mg/kg lipid, respectively (ATDSR, 2005). 140 bovine milk samples from 14 districts of Haryana, India (sampled within 1998 - 1999) were analysed for organochlorine pesticide residues. Four percent of the samples exceeded the maximum residue limit (MRL) of 0.05 mg/kg as recommended by WHO for alpha-HCH (Sharma et al., 2006). A monitoring study (192 samples) of cow’s milk from Mexico revealed 0.001 - 0.201 mg/kg alpha-HCH (ATDSR, 2005).

Fish and clam samples from India contained 0.01 – 0.02 mg/kg ww and 0.26 mg/kg ww alpha-HCH respectively (Nair and Pillai, 1992).

High levels of alpha-HCH in the food chain are documented for the arctic region (AMAP, 2004b).

### 3.3.4 Body burden

#### 3.3.4.1 General population

Median levels of alpha-HCH in 25 US-American patients were 0.04 ng/g in the whole blood and 1.1 ng/g (maximum 9.6 ng/g) in biopsy fat (ATDSR, 2005). A Spanish study reported mean alpha-HCH levels of 1.43 µg/g (maximum 6.75 µg/g) in fat samples of children living in farm areas (Olea et al., 1999).

Alpha-HCH has been detected in 1.7 % of the 4822 blood samples of German adults from 120 locations (detection limit: 0.1 µg/l) (German Environmental Survey 1998, Becker et al., 1998). Alpha-HCH was detected in blood serum from three of 186 (~1.6 %) Brazilian children (mean: 1.8 ppb) (ATDSR, 2005). Alpha-HCH has been detected in all samples (n = 142) of an eastern Romanian study in 2005 with a median concentration of 31 ng/g lipid (range 3 - 146 ng/g) (Dirtu et al., 2006). High concentrations were reported for India, due to agricultural use and malaria control. Blood serum contained up to 0.45 mg/l alpha-HCH, whereas adipose tissue...
contained up to 0.30 mg/kg. Breast milk contained 0.16 mg/l (mean) (Nair and Pillai, 1992).

Scheele et al. (1998) investigated levels of several organochlorine compounds including alpha-HCH in bone marrow of 29 adults from Germany (collected between 1980 and 1991). Compared to adipose tissue, with generally highest levels of organochlorine compounds, alpha-HCH concentrations were 10-fold higher in bone marrow (mean: 0.050 ppm on dry lipid weight; max: 0.476 ppm). Alpha-HCH has also been detected in semen (ATDSR, 2005).

### 3.3.4.2 Indigenous population

Blood plasma samples from different regions and ethnic groups of indigenous mothers of the Arctic were investigated for total HCH and beta-HCH (AMAP, 2004b). A more recent study about exposure and health effects from the Russian indigenous population also focuses on total HCH. Data on alpha-HCH are scarce. However, total HCH concentrations of blood samples of pregnant indigenous women below 40 years were 204.0 ng/g lipid and 384.2 ng/g lipid (geometric means) for women of 40 years and more, respectively.

### 3.3.5 Exposure of children

Children are at specific developmental stages more vulnerable to risks from chemical substances than adults. It is unclear if children are more susceptible than adults to health effects from exposure to alpha-HCH although it is known that the developing brain is sensitive to the effects of different POPs. The specific enrichment of alpha-HCH in the mammalian brain might be a reason of concern. Placental transfer of alpha-HCH in humans is well documented (ATDSR, 2005; Falcon et al., 2004; Shen et al., 2006). Alpha-HCH accumulates to a higher extent in human placenta than in breast milk.

Mean alpha-HCH levels in breast milk of a Finnish cohort (43 mothers, 1997 – 2001) were 0.19 ng/g lipid, whereas placenta mean concentrations of alpha-HCH were 3.47 ng/g lipid. In a Danish cohort (43 mothers, 1997 – 2001), mean concentrations of 0.51 ng/g lipid in breast milk and 1.53 ng/g lipid in placenta were detected. A specific metabolic activity of the placental tissue is suspected (Shen et al., 2007). It could be shown that in case of restrictions of use, alpha-HCH concentrations in breast milk decline continuously. In Germany alpha-HCH was still found in 28 % of the breast milk samples analysed in 1984/85 whereas it could not be detected in 1990/91 and 1995 samples (Ott et al., 1999). More than 2 000 individual human milk samples from women living in Western Germany collected and analysed between 1984 and 2001 indicated that alpha HCH concentration declined from > 0.01 mg/kg fat to levels below detectability (detection limit of 0.001 mg/kg fat) (Fürst, 2004). In the framework of the 3rd WHO human milk field study, HCHs were analysed in 16 human milk pools from ten European countries. In Bulgaria, Russia and Ukraine, alpha-HCH was detected in concentrations between 0.002 – 0.006 mg/kg lipid, whereas in the samples of Czech Republic, Germany, Ireland, Italy, Luxembourg, Norway and Spain alpha-HCH was not detectable (detection limit: 0.001 mg/kg lipid). In Nairobi, Kenya, 8.8% of 216 breast milk samples contained detectable alpha-HCH with a mean concentration of 0.013 mg/kg milk fat and a range of 0.002 – 0.038 mg/kg (Kinyamu et al., 1998). Breast milk samples from India contained 0.16 mg/l (mean) (Nair and Pillai, 1992). Another Indian study reports 0.045 mg/l alpha-HCH in breast milk (Nair et al., 1996)

It can be concluded that alpha-HCH concentrations in breast milk strongly depend on exposure and that in several East European and developing countries concentrations are still very high.

### 3.4 Hazard assessment for endpoints of concern

Compared to technical HCH and lindane, limited data are available for alpha-HCH. A limited number of subchronic and chronic oral toxicity studies exist. No studies of the
toxicity of alpha-HCH via inhalation and dermal application have been conducted. Studies on acute toxicity, developmental, teratogenic and reproductive effects of alpha-HCH are missing. There is a lack of dose-response data after oral exposure for all relevant species. For the present risk profile, the most important findings concerning the hazard assessment have been reviewed.

**Acute Toxicity/ Neurotoxicity**
Oral LD50 values range between 1000 and 4000 mg/kg bw for mice and between 500 and 4674 mg/kg bw for rats. The signs of poisoning were those of central nervous stimulation: excitation, hunched posture, rough fur, dyspnoea, anorexia, tremors, convulsions, and cramps (IPCS, 1992).

**Subchronic toxicity**
In a 90-day study on rats carried out with dose levels of 0, 2, 10, 50, or 250 mg alpha-HCH/kg diet, growth retarded and relative weight of organs (liver, heart, kidneys, and adrenals) increased at 250 mg/kg diet (equivalent to 12.5 mg/kg bw/day). At levels of 50 and 250 mg/kg, liver enzyme activities were modified and liver parenchyma cells enlarged. Liver weight increased at dose levels of 10 mg/kg diet (equivalent to 0.5 mg/kg bw/day) and reductions in white blood cell count were noted. Signs of immunosuppression (reduced serum levels of immunoglobulins G and M) were observed at 50 and 250 mg/kg diet. The NOAEL was 2 mg/kg alpha-HCH/kg diet (equivalent to 0.1 mg/kg bw/day) (IPCS, 1992).

**Chronic Toxicity**
When groups of 10 female and 10 male weanling Wistar rats were administered daily diets containing 0, 10, 50, 100, or 800 mg/kg alpha-HCH (in corn oil) for 107 weeks, the highest dose level resulted in growth retardation, increased mortality, and slight kidney damage. With daily doses of 100 or 800 mg/kg, liver enlargement and histopathological changes in the liver were found. However, there were no liver changes at 50 mg/kg diet (Fitzhugh et al., 1950).

**Genotoxicity**
Alpha HCH was not mutagenic to bacteria (*Salmonella typhimurium* strains TA 98, TA 100, TA 1535 and TA 1537) with and without metabolic activation and did not induce DNA damage in bacteria. However, alpha-HCH induced DNA-fragmentation in human and rat hepatocytes. Oral exposure to alpha-HCH resulted in mitotic disturbances including an increased mitotic rate and increased frequency of polyploid hepatic cells in mice (ATDSR, 2005).

**Carcinogenicity**
Studies of the carcinogenicity of alpha-HCH are limited. Several studies in mice were performed, but their value is limited. Nevertheless, it is clear from the results that alpha-HCH, at high dose levels, produces nodular hyperplasia and hepatocellular carcinomas in mice (the incidence varying according on the strain) and also in rats (low incidence). Studies on initiation promotion and mode of action indicate that the neoplastic response observed with alpha-HCH is most likely due to a non-genotoxic mechanism. Alpha-HCH has been shown to promote tumors in the liver of mice and rats (IPCS, 1992). The International Agency for Research on Cancer (IARC) classified alpha- HCH in group 2A: possibly carcinogenic to humans. USEPA categorized alpha-HCH as probable human carcinogen.

**Immunotoxicity**
Mice, treated with alpha-HCH (50 and 250 mg/kg/day- i.e. 0.5 and 2.5 mg/kg/bw/day) showed signs of immunosuppression (reduced serum levels of immunoglobulins G and M).

**Effects in Humans**
Adverse effects such as neurophysiological and neuropsychological disorders and gastrointestinal disturbances have been reported for workers exposed to technical HCH
during pesticide or fertilizer formulation. Workers suffered from paraesthesia of the face and extremities, headache and giddiness, malaise, vomiting, tremors, apprehension, confusion, loss of sleep, impaired memory and loss of libido. Serum enzyme and IgM levels were enhanced (ATDSR, 2005).

A German study on organochlorinated compounds in the peripheral blood of 486 women with hormonal disorders and/or infertility revealed that alpha-HCH concentrations were significantly higher in women with uterine fibroids, antithyroidal antibodies, luteal insufficiency and women highly susceptible to allergies. Obese women and women with a history of abortion had the highest HCH levels in blood (Gebhard, 1993).

In a pilot study with limited statistical power a possible association between exposure to organochlorines and the risk of childhood aplastic anaemia was shown. Alpha-HCH was significantly higher in children with aplastic anaemia than in those of controls (p < 0.05) (Ahamed et al., 2006).

The association between alpha-HCH exposure and intrauterine growth retardation (IUGR, < 10th percentile of birth weight for gestational age) was examined in India. Statistically significant associations (p < 0.05) between maternal blood levels of alpha-HCH and intrauterine growth retardation were found (Siddiqui et al., 2003).

**Environment**

Data on effects in non-target species are extremely limited. Alpha-HCH is acutely toxic to aquatic organisms. Effect concentrations in algae, zooplankton (brine shrimp and water flea) and fish of < 1 mg/l were reported (IPCS, 1992; ECOTOX database, 2007). A LC50 of approximately 1.4 mg/l was determined in an acute test in zebra fish (Oliveira-Filho and Paumgarten, 1997). In a long term study with snails (*Lymnaea stagnalis*) a 50 % reduction of reproduction was found at a concentration of 65 µg/l. In fish no histopathological changes or influence on growth and behaviour could be detected in long-term experiments at low concentrations (IPCS, 1992). Monitoring data on Arctic polar bears revealed a negative correlation with retinol concentrations and HCHs, which may impact a wide range of biological functions (AMAP, 2004).

**Risk characterisation**

USEPA performed a dietary risk assessment for Alaskan communities for alpha and beta-HCH in 2006. USEPA estimated alpha-HCH exposures for Alaskan communities in the range of 0.00057 - 0.0039 mg/kg bw/day for adults, 0.0021 - 0.051 mg/kg bw/day for children (age 1 - 6) and 0.00073 - 0.0050 mg/kg bw/day for children (age 7 - 12). The risk is expressed as a percentage of a maximum acceptable dose or reference dose (RfD). A level of concern is reached if the dietary risk exceeds 100 % RfD (USEPA, 2006). The RfD value of 0.001 mg/kg/day for chronic exposure is based on a LOAEL of 0.5 mg/kg/day established in a chronic study in rats and applying an uncertainty factor of 100 (ATSDR, 2005). RIVM calculated a chronic inhalation RfD of 0.00025 mg/m³ for alpha-HCH based on a NOAEL of 0.025 mg/m³ for observations of liver and kidney toxicity observed in an subchronic inhalation study in rats and applying an uncertainty factor of 100 (RIVM, 2001 in USEPA, 2006).

The acute dietary exposure estimates are not of concern according to USEPA (2006). USEPA's dietary risk assessment indicates that the chronic dietary exposure estimates for alpha-HCH are above the levels of concern for high-end dietary intake estimates. The cancer dietary risk estimates for alpha-HCH are also above the level of concern for both low and high-end dietary intake estimates. According to EPA, the risk values (% cRfD) are 57 - 390 for adult males, 67 - 460 for adult females, 210 - 5 100 for children (1 - 6 years) and 73 - 500 (7 - 12 years). The estimated cancer risk for adult males is 3.2x10⁻³ to 2.5x10⁻² and 4.2x10⁻³ to 2.9x10⁻² for adult females. It should be noted that these estimated incidences are at least four orders of magnitude higher than a general accepted cancer risk of 1x10⁻⁶. Even though this risk estimation is very conservative due to the basic maximum detected levels it can be concluded that the dietary risks are
of concern. Additionally, it has to be mentioned that the target organ of chronic toxicity is the liver and it can be expected that HCHs effects might be additive.

4. SYNTHESIS OF THE INFORMATION

Technical HCH, a mixture of five stable HCH-isomers, contains 55 – 80 % alpha-HCH and was used extensively worldwide as an organochlorine pesticide.

Though usage of technical HCH is negligible nowadays releases into the environment may still occur. Local sources include hazardous waste sites, contaminated sites, stockpiles and landfills or dumping grounds. Though no quantitative estimates of these releases exist, the amounts of HCH residuals in the form of by-products from lindane production are assumed to range between 1.6 - 1.9 to 4.8 million tons. In addition, many sites are expected to cause environmental pollution and are not maintained or controlled appropriately.

The physico-chemical properties of alpha-HCH facilitate long-range atmospheric transport and allow for "cold condensation" on a global scale. In addition, the low Henry's law constant contribute to achieve high levels in the Arctic Ocean. Moreover, it was shown that Arctic air concentrations mimicked global usage data directly until the early 1990s. Also, monitoring data from remote regions e.g. the Arctic and Antarctica suggested that detected levels, which were sometimes higher than in source regions, originate from long range transport.

Alpha-HCH is fairly degradable under favourable conditions (e.g. enhanced bioavailability, temperature, moisture content). Hydrolysis contributes to the overall removal of alpha-HCH in aqueous solution under alkaline pH, but under environmental conditions with minor importance. Alpha-HCH may undergo enantioselective degradation which depends on the site and medium. Reported half-lives and residues analyses in soil suggest moderate persistence. However, certain environmental conditions e.g. low concentrations or low temperatures resulted in longer half-lives. Half-lives for alpha-HCH in Arctic lakes were up to 1.4 years, whereas in the Eastern Arctic Ocean enantioselective degradation resulted in a range of approximately 5 to 17 years.

Alpha-HCH may bioaccumulate and biomagnify in biota and Arctic food webs. The BMFs as well as FWMFs in invertebrates, fish and terrestrial and marine mammals were greater than 1. Because of the individual potential to metabolize alpha-HCH, birds do not fit into this scheme. Most birds show BMFs < 1, independent of the trophic level. Especially in mammals, a enantiospecific accumulation of (+) or (-) alpha-HCH occurs (depending on the species). Combined with the lower potential for biotransformation alpha-HCH, - reaches high BMFs in mammals, with the highest concentrations in brain tissue (especially the (+) enantiomer). As all HCHs aim at the central nervous system, this has to be seen with caution. To date, however, no enantiomer-specific toxicity studies for alpha-HCH are available and the reasons for the enrichment and differences are largely unclear.

Alpha-HCH has been shown to be neurotoxic, hepatotoxic, and to cause immunosuppressive effects and cancer in laboratory animals.

The International Agency for Research on Cancer (IARC) has classified alpha-HCH in group 2B, possibly carcinogenic to humans. Several epidemiological studies indicate that alpha-HCH might play a role in human breast cancer. Alpha-HCH is a known tumour promoting agent.

Alpha-HCH may adversely affect human health in contaminated areas as well as in Arctic regions. Based on the available toxicity data of alpha-HCH it can be concluded that current concentrations of alpha-HCH in food and human milk are a matter of concern. The estimated daily intake of alpha-HCH of Arctic indigenous people exceeds safe intake reference values, even though estimation is very conservative. Compared with a general accepted risk of one case per million, this risk seems unacceptably high. Nevertheless it should be emphasized that traditional foods have unique social, cultural, spiritual and economic value and therefore it is strongly recommended to avoid alpha-HCH levels of concern.
CONCLUDING STATEMENT

Though most countries have banned or restricted the use of technical HCH as a pesticide, replacing it in most cases by the use of lindane, the lindane production process has produced huge amounts of HCHs residuals. These waste isomers are still a worldwide problem and contribute to the releases into the environment.

Releases into the environment have dramatically decreased over the past 30 years, but levels in the environment suggest that alpha-HCH may persist in the environment (at lower concentrations). Especially the cold Arctic Ocean, which is now eliminating alpha-HCH, was a sink which preserved the chemical from rapid degradation. Levels in Arctic biota do not thoroughly reflect the decreasing trend of the abiotic compartments.

Alpha-HCH is present in the terrestrial and the aquatic food chains and concentrations are a human health concern. High exposure is expectable in polluted areas and, as a result of long-range transport, in the Arctic region. In addition, humans and wildlife are exposed to various contaminants that can influence the toxicological effects of alpha-HCH in an additive or synergistic way. Based on the inherent properties, together with estimated daily intakes of alpha-HCH of Arctic indigenous people that exceeds safe intake reference values, and given the widespread occurrence of alpha-HCH in biota, including in remote areas far from likely sources, it is concluded that the substance is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.
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### B.2. Beta-hexachlorocyclohexane – SUMMARY

**Summary**

2. Beta Hexachlorocyclohexane (Beta HCH)

Draft Risk Profile May 2007

http://www.pops.int/documents/meetings/poppercdrp/DraftRiskProfile_b-HCH.pdf

<table>
<thead>
<tr>
<th>Composition</th>
<th>One of 5 stable HCH isomers in technical HCH at levels of 5 – 14%.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Beta-HCH by itself is neither intentionally produced nor placed on the market. It is produced as constituent of technical HCH used as organochlorine insecticide or chemical intermediate to manufacture enriched HCH (lindane). Currently no production data on technical HCH have been reported, whereas manufacture of lindane still takes place (IHPA, 2006).</td>
</tr>
<tr>
<td>Releases</td>
<td>Around 10 million tons of technical HCH were released to the environment between 1948 and 1997 (Li et al. 1999). According to Li and Macdonald (2005) global usage of technical HCH was dominated by 10 countries headed by China, which consumed almost half of the total global quantity. The other countries were (in order of decreasing usage): Former Soviet Union, India, France, Egypt, Japan, United States, East Germany, Spain and Mexico. Historically beta-HCH was released during the manufacture of technical HCH and its use as a pesticide. Li et al. (2003) estimated global emissions of beta-HCH from the usage of technical HCH between 1945 and 2000 at 850 000 tons, of which 230 000 tons were emitted into the atmosphere over the same period. As a result of the ban on technical HCH in northern countries, global emissions of beta-HCH have undergone a “southward tilt” (Li et al., 2003). Releases of beta-HCH into the environment are also possible from hazardous waste sites (USEPA, 2006), stockpiles and residues of lindane production, which are not always controlled or maintained safely (IHPA, 2006). Also, contaminated sites (e.g. from former production plants) may contribute to the environmental burden of beta-HCH (Concha-Grana et al., 2006).</td>
</tr>
<tr>
<td>Fate</td>
<td>Abiotic degradation processes do not play an important role in the fate of beta-HCH in the environment. Thus photolysis and hydrolysis are not significant. Under favourable conditions, beta-HCH is susceptible to biodegradation. However compared to the gamma- and alpha-HCH it is the most recalcitrant isomer. Laboratory and field data including a long-term soil study suggest that beta-HCH is persistent in soil, especially under low temperatures. It is mainly associated with particles and has a low leaching potential. The physico-chemical properties of beta-HCH allow the dispersal of the substance from its sources to the Arctic mainly by long-range environmental transport via ocean currents. Beta-HCH has been detected in the Arctic Ocean and is present in marine, terrestrial species, and humans.</td>
</tr>
<tr>
<td>Effects</td>
<td>Beta-HCH is acutely toxic to aquatic organisms and shows estrogenic effects in fish. Reduced fitness of offspring in birds as well as reduced retinol concentrations in polar bears is associated with beta-HCH and HCHs levels. Toxicological studies with beta-HCH have demonstrated neurotoxicity and hepatotoxicity. Also reproductive and immuno-suppressive effects and effects on fertility were seen in laboratory animals. Beta-HCH has been classified in</td>
</tr>
</tbody>
</table>
group 2B as possibly carcinogenic to humans by the International Agency on Research and Cancer (IARC). Several epidemiological studies indicate that beta-HCH might play a role in human breast cancer.

| Exposure | Beta-HCH exposure levels in local areas have declined after worldwide prohibitions and restrictions. However regions with recent exposure and/or high pollution can still show elevated levels. A special concern also arises from exposure of hazardous waste sites and dumping grounds from disposed beta-HCH residues from lindane production. Due to its persistence beta-HCH can still be detected at low background levels in all environmental media except in regions with recent usage and/or high pollution. Data from the abiotic environment in the Arctic are scarce partly due to low levels compared with the other HCH isomers. In contrast to this fact fairly high concentrations in Arctic biota including marine mammals and birds were detected with increasing levels. Beta-HCH is present in terrestrial and aquatic food chain. Beta-HCH may bioaccumulate and biomagnify in biota and Arctic food webs, especially in upper trophic levels. In humans accumulation in fat tissue and high concentrations in blood and in breast milk may occur. Beta-HCH transfers from mothers to embryos and nursing infants. Human exposure to beta-HCH results mostly from ingestion of contaminated plants, animals and animal products. High exposure is expected in contaminated areas due to extensive use, former production, disposal sites and stockpiles. Based on the hazard profile and the exposure levels in the environment including the food chain, it can be concluded that beta-HCH may adversely affect wildlife and human health in contaminated regions. Arctic public health authorities believe the significant social, cultural and economic benefits of traditional foods outweigh the risks of contaminants such as HCH at present but give another reason for the quick control and elimination of all HCH isomers from traditional foods. However based on levels found in the Arctic region, it can be also concluded that beta-HCH can lead to significant adverse human and environmental effects as a result of its long-range environmental transport. |

| Status | Technical HCH is listed in Annex II of the 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) under the Convention on Long-Range Transboundary Air Pollution which restricted alpha-HCH use to an intermediate in chemical manufacturing only. HCH (mixed isomers) is subject to the PIC Procedure of the Rotterdam Convention and is listed in Annex III of the Convention. Canada, Mexico, and the United States signed the North American Regional Action Plan (NARAP) on Lindane and Other Hexachlorocyclohexane Isomers in 2006 with the goal of reducing the risks associated with the exposure of humans and the environment to lindane and other HCH isomers. In the European Union the production and use of technical HCH as an intermediate in chemical manufacturing will be phased out by the end of 2007 at the latest (Regulation (EC) No 850/2004). HCHs are also one of the priority substances (Decision No 2455/2001/EC) of the adopted EU Water Framework Directive 2000/60/EC. Hexachlorocyclohexane isomers, including the beta-isomer, are on the List of Chemicals for Priority Action under the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic. |

| Alternatives | Will be discussed together with Lindane in Annex F evaluation if Beta HCH advances. |
Candidate for POPs List

$\beta$-HCH

beta-hexachlorocyclohexane
Background

The 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) addresses technical hexachlorocyclohexane (HCH, a mixture of isomers) as a substance for restriction on use under Annex II. The Aarhus Protocol is one of the protocols under the United Nations Economic Commission for Europe (UNECE) convention on Long Range Transboundary Air Pollution (LRTAP). The objective of the UNECE regional Protocol is to control, reduce or eliminate discharges, emissions and losses of persistent organic pollutants.

The Rotterdam Convention on the Prior Informed Consent also includes technical HCH, indicating that several countries have banned or severely restricted import and use of this mixture of isomers. The objective of this convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm.

Mexico proposed on June 29, 2005 that gamma-hexachlorocyclohexane (Lindane) be added to Annex A of the Stockholm Convention. The proposal presented data on the gamma isomer but mentioned as well that "other isomers of hexachlorocyclohexane should also be considered in this proposal".

The POPs Review Committee (POPRC) evaluated Annex D information for Lindane at its first meeting, held in Geneva in November 2005, and decided that "the screening criteria have been fulfilled for Lindane". The Committee agreed that the alpha and beta isomers could be included in the discussions, although any decision to propose inclusion of the chemical in the Convention would apply only to Lindane, the gamma isomer. As a consequence, Mexico proposed that beta-HCH (and alpha-HCH in another proposal) be added to Annexes A, B and/or C of the Convention to ensure that the global impacts of all three environmentally significant HCH isomers (alpha, beta and gamma) are addressed.

Data Sources


Introduction

Beta-HCH is one of the five stable isomers of technical HCH, an organochlorine pesticide formerly used in agriculture. The modes of action of the HCH isomers differ quantitatively and qualitatively with regard to their biological activity in the central nervous system as the main target organ. Beta-HCH is mainly depressant and the final effect of the mixed isomers depends on the composition (IPCS, 2001). In general HCHs are among the most studied pesticides with respect to environmental fate and effects (Breivik et al., 1999).

1 Identification of the chemical

1.1 Names and registry numbers

Chemical name: beta-hexachlorocyclohexane (beta-HCH)

IUPAC name: (1-alpha, 2-beta, 3-alpha, 4-beta, 5-alpha, 6-beta)-Hexachlorocyclohexane
Common synonyms: beta-1,2,3,4,5,6-Hexachlorocyclohexane: benzenehexachloride, beta-BHC, benzene-cis-hexachloride; beta-HCH; beta-Hexachlorocyclohexane; beta-Hexachlorocyclohexane; beta-isomer; beta-lindane; Hexachlorocyclohexane-Beta; trans-alpha-benzenehexachloride; beta-benzenehexachloride

CAS number: 319-85-7

1.2 Chemical Structure

Figure 1: Structure of beta-HCH, modified from Buser et al. (1995)

![Structure of beta-HCH](image)

Chemical formula: $C_6H_6Cl_6$

Molecular weight: 290.83

1.3 Chemical production

HCH isomers are produced as a result of the photochemical chlorination of benzene during the manufacture of technical HCH, which has been widely used as a commercial pesticide. Technical HCH is a mixture of five HCH isomers: alpha-HCH (53-70%), beta-HCH (3-14%), gamma-HCH (11-18%), delta-HCH (6-10%) and epsilon-HCH (3-5%).

As the gamma-HCH isomer, also known as Lindane, is the isomer with the highest pesticidal activity, technical-HCH is subject to subsequent treatment (fractional crystallization and concentration) to produce 99% Lindane. This process is extremely inefficient with only a 10-15% yield, producing 6-10 tons of other isomers for each ton of Lindane (IHPA, 2006). Alpha-HCH is the major by-product of the reaction (60-70%), followed by beta-HCH (7-10%) (WHO, 1991).

1.4 Physico-chemical properties

Beta-HCH is more soluble in water and octanol compared to other organochlorine pesticides. Its chemical structure seems to confer the greatest physical and metabolic stability (e.g. beta-HCH has a lower vapour pressure and a higher melting point than the alpha-isomer). The physico-chemical properties (a selection is given in table 1) of beta-HCH allow for “cold condensation”, an enrichment of the substance in cold climates compared to concentrations near sources, on altitudinal and latitudinal scales described by Wania and Mackay (1996).

The Henry’s law constant is a factor of 20 lower than for alpha-HCH and decreases significantly with water temperature which favours partitioning from air to water. Also
its relatively high log \( K_{oa} \) promotes partitioning from air to environmental organic phases. This is probably one reason why transportation pathways of alpha- and beta-HCH diverge in the environment (Li and Macdonald, 2005). Based on an extensive data analysis of the physico-chemical properties of alpha-, beta- and gamma-HCH Xiao et al. (2004) concluded that the lower volatility compared to the gamma- and alpha-isomer does not account for its different environmental behaviour but is caused by a higher solubility in water and octanol (data not shown).

<table>
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<tr>
<th>Table 1: Selected physico-chemical properties</th>
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<tr>
<td>Melting Point (°C)</td>
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<tr>
<td>Boiling Point (°C)</td>
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<tr>
<td>Density (g cm(^{-3}) at 19 °C)</td>
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<td>Water solubility (mg/l at 20 °C)</td>
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<td>Vapour pressure (mmHg at 20 °C)</td>
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<td>Henry’s law constant (atm m(^3) mol(^{-1}))</td>
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<td>Log Kow</td>
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<td>Log Koa (0°C)</td>
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<td>Physical state</td>
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\(^1\) ATSDR (2005)  
\(^2\) HSDB (2006)  
\(^3\) Shoeib and Harner (2002)

2 Persistence

In general, HCH isomers are resistant to abiotic processes like photolysis and hydrolysis (except at high pH), and microbial degradation is very slow (USEPA, 2006).

Beta-HCH is the most persistent isomer, with half-lives of 184 and 100 days on cropped and uncropped plots. It comprised 80-100% of the total HCH residues found in soil and vegetation on land surrounding an industrial landfill in Germany 10 years after the final HCH input (ATSDR, 2005). Other laboratory studies have calculated half-lives values of 91 and 122 days for aerobic and anaerobic soil indicating that persistence is dependent on environmental factors such as the action of soil microorganisms, evaporation rates, soil oxygen and organic matter content (WHO, 1991).

Beta-HCH has a much lower vapor pressure and a much higher melting point than the alpha-HCH. These properties are dictated by the great physical and metabolic stability conferred by the isomer structure (Willet, 1998).

Although photolysis is not expected to be an important environmental fate process for HCH, it may be degraded in the atmosphere by reacting with photochemically produced hydroxyl radicals. A photodegradation half-life for a thin film of beta-HCH equal to 152 hours has been reported (ATSDR, 2005).

3 Bioaccumulation

Beta-HCH is the predominant isomer in soils and animal tissues because its configuration favors storage in biological media and affords it greater resistance to hydrolysis and enzymatic degradation (Walker, 1999).

The log octanol-water partition coefficient (log Kow) for beta-HCH is 3.78, indicating that it has the potential to bioaccumulate. A bioconcentration factor (BCF) equal to 1 460 was found for beta-HCH using zebra-fish under steady-state conditions compared to BCFs equal to 1 100 for alpha-HCH and 850 for gamma-HCH (ATSDR, 2005). BCFs from 250 – 1 500 on a dry weight basis or 500 000 times on a lipid basis within 3-10 days have also been reported (WHO, 1991).

Several studies suggest that the relative proportions of HCH isomers vary dramatically across species in the Arctic marine food web. A study carried out in 2000 indicated that upper trophic level mammals may be able to efficiently eliminate Lindane and to a smaller extent alpha-
HCH, but not beta-HCH. As a result, beta-HCH tends to bio-accumulate to higher concentrations in upper trophic level fishes, birds and mammals (USEPA, 2006).

4 Potential for long range environmental transport

Air concentrations of beta-HCH have been measured regularly at the Alert and Tagish stations in Arctic Canada. The results indicate that concentrations of beta-HCH in the Arctic atmosphere are very low in comparison with the more volatile alpha- and gamma-HCH. However, the concentration of beta-HCH in Arctic surface water can be as high as 240 pg/L, approaching the concentration of gamma-HCH in the same media (Li et al, 2003).

Li et al (2002) reported that in contrast to alpha-HCH, beta-HCH appears to be less subject to direct atmospheric loading into the high Arctic as most of beta-HCH stays in the source region after application. This can be explained by differences in their Henry’s law constant and air/water partition coefficient that leads to enhanced affinity for particles, greater resistance to degradation and reduced volatility of beta-HCH.

According to Li et al (2002), rain scavenging is much more efficient for beta- than for alpha-HCH. In addition, the amount and frequency of precipitation is considerably higher in the North Pacific compared to the Arctic. These two aspects, when combined, suggest that beta-HCH enters the Arctic probably by mechanisms involving wet deposition or partitioning into the North Pacific surface water and subsequently entering the Arctic in ocean currents passing through the Bering Strait (Li et al, 2003).

The Bering and Chukchi Seas are the most vulnerable locations for beta-HCH loadings coming primarily from Asia via the Pacific (Li et al, 2002).

5 Adverse effects

Beta-HCH has moderate toxicity for algae, invertebrates and fish. The acute LC50 values for these organisms are of the order of 1 mg/L (WHO, 1991).

Studies of short-, intermediate- and long-term exposure to beta-HCH in diet have reported liver and renal effects in animals. A significantly decreased body weight gain has been seen in rats treated orally with 250 mg/kg beta-HCH. Neurological effects have also been reported in rats exposed to beta-HCH. Oral exposure of rats and mice to beta-HCH has resulted in degeneration of male reproductive organs and sperm abnormalities. The limited genotoxicity data indicate that beta-HCH has some genotoxic potential but the evidence is not conclusive (USEPA, 2006).

Beta-HCH may be the most toxicologically significant HCH isomer due to the recent reports of its estrogenic effects in mammalian cells, laboratory mammals and fish (Willet, 1998).

There are limited studies to estimate cancer risk from exposure to beta-HCH. However, EPA’s Integrated Risk Information System (IRIS) currently lists beta-HCH as a possible human carcinogen based on the incidence of hepatic nodules and hepatocellular carcinomas observed in male mice administered beta-HCH at a single dose level in the diet (USEPA, 2006).

REASONS FOR CONCERN

Beta-HCH is the most persistent isomer of hexachlorocyclohexane. Due to its physicochemical properties it has the potential to bioaccumulate. Its listing as a possible human carcinogenic should also be of special concern.

Even though most countries have banned or restricted the use of technical HCH as a pesticide, replacing it in most cases by the use of Lindane (99% gamma-HCH), the production process to obtain a ton of pure gamma-HCH yields 6 – 10 metric tonnes of the other isomers that must be disposed of or otherwise managed. As Lindane is the only isomer in the mixture that has insecticidal properties, there is very limited to no commercial value for the other
isomers obtained. Because of this waste isomer problem, the production of HCH/Lindane has been a worldwide problem for years.

Other HCH isomers, like beta-HCH, can be as toxic and persistent a contaminant as Lindane, or even more so. The continued use of Lindane in the world is causing this important pollution source. Global action is therefore needed to halt the pollution caused worldwide by Lindane production.
After almost forty years of extensive use worldwide, there has been a gradual replacement of technical hexachlorocyclohexane (HCH) by lindane (gamma-HCH). No significant uses of technical HCH have been reported after 2000. However releases into the environment may also occur from lindane production as well as from hazardous waste sites, landfills and contaminated sites. Because of its hazard profile and widespread abundance, technical HCH (including beta-HCH) is subject to national and international regulations and prohibitions.

Abiotic degradation processes do not play an important role in the fate of beta-HCH in the environment. Thus photolysis and hydrolysis are not significant. Under favourable conditions, beta-HCH is susceptible to biodegradation. However compared to the gamma- and alpha-HCH it is the most recalcitrant isomer. Laboratory and field data including a long-term soil study suggest that beta-HCH is persistent in soil, especially under low temperatures. It is mainly associated with particles and has a low leaching potential.

The physico-chemical properties of beta-HCH allow the dispersal of the substance from its sources to the Arctic mainly by long-range environmental transport via ocean currents. Beta-HCH has been detected in the Arctic Ocean and is present in marine, terrestrial species, and humans.

Beta-HCH exposure levels in local areas have declined after worldwide prohibitions and restrictions. However regions with recent exposure and/or high pollution can still show elevated levels. A special concern also arises from exposure of hazardous waste sites and dumping grounds from disposed beta-HCH residues from lindane production. Due to its persistence beta-HCH can still be detected at low background levels in all environmental media except in regions with recent usage and/or high pollution. Data from the abiotic environment in the Arctic are scarce partly due to low levels compared with the other HCH isomers. In contrast to this fact fairly high concentrations in Arctic biota including marine mammals and birds were detected with increasing levels.

Beta-HCH is present in terrestrial and aquatic food chain. Beta-HCH may bioaccumulate and biomagnify in biota and Arctic food webs, especially in upper trophic levels. In humans accumulation in fat tissue and high concentrations in blood and in breast milk may occur. Beta-HCH transfers from mothers to embryos and lactating infants.

Beta-HCH is acute toxic to aquatic organisms and shows estrogenic effects in fish. Reduced fitness of offspring in birds as well as reduced retinol concentrations in polar bears is associated with beta-HCH and HCHs levels.

Toxicological studies with beta-HCH have demonstrated neurotoxicity and hepatotoxicity. Also reproductive and immunosuppressive effects and effects on fertility were seen in laboratory animals. Beta-HCH has been classified in group 2B as possibly carcinogenic to humans by the International Agency on Research and Cancer (IARC). Several epidemiological studies indicate that beta-HCH might play a role in human breast cancer.

Human exposure to beta-HCH results mostly from ingestion of contaminated plants, animals and animal products. High exposure is expected in contaminated areas due to extensive use, former production, disposal sites and stockpiles.

Based on the hazard profile and the exposure levels in the environment including the food chain, it can be concluded that beta-HCH may adversely affect wildlife and human health in contaminated regions. Arctic public health authorities believe the significant social, cultural and economic benefits of traditional foods outweigh the risks of contaminants such as HCH at present but give another reason for the quick control and elimination of all HCH isomers from traditional foods. However based on levels found in the Arctic region, it can be also concluded...
that beta-HCH can lead to significant adverse human and environmental effects as a result of its long-range environmental transport.

For these reasons, global action on beta-HCH is warranted.

Data sources
The draft risk profile is based on the following data sources:

- Proposal submitted by Mexico for listing alpha and beta isomers in Annexes A, B and/or C to the Convention (UNEP/POPS/POPRC2./INF/8), 2006.
- Information submitted by parties and observers according to Annex E of the Convention: specific and/or scientific information: Czech Republic, France, Germany, International POPs Elimination Network (IPEN), Japan, Norway, Switzerland, United States of America, general information: Algeria, Crop Life International, Kingdom of Bahrain, Mauritius, Mexico, Qatar, Republic of Lithuania and Turkey. This information is available on the Convention’s website (http://www.pops.int/documents/meetings/poprc/prepdocs/annexEsubmissions/submissions.htm)

In addition to these information sources a literature search of public data bases was conducted. The following databases were used: ECOTOXicology database (Ecotox, http://www.epa.gov/ecotox/) Hazardous Substances Data Bank (HSDB, http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB), Pubmed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?DB=pubmed), Environmental Fate Data Base (EFDB http://www.syres.com/esc/efdb_info.htm. In general search terms include the chemical name or CAS number and/or a combination of a technical term because of the multiplicity of entries. For the same reason also specific topical and updated articles were considered.
The quoted above listed reports contained individual references, which were not listed again in this draft risk profile.

STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS

Beta-HCH is a constituent of technical HCH, which is regulated at least by two international agreements. The first one is the 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs) under the Convention on Long-Range Transboundary Air Pollution. Technical HCH is listed in Annex II of the protocol which restricted its use to an intermediate in chemical manufacturing only.

The second agreement is the Rotterdam Convention on the Prior Informed Consent (PIC) Procedure for Certain Hazardous Chemicals and Pesticides in International Trade. HCH (mixed isomers) is subject to the PIC Procedure and is listed in Annex III of the Convention.

Canada, Mexico, and the United States signed the North American Regional Action Plan (NARAP) on Lindane and Other Hexachlorocyclohexane Isomers in 2006. The goal of the NARAP is for the three member countries to cooperatively take actions to reduce the risks associated with the exposure of humans and the environment to lindane and other HCH isomers.

In the European Union the production and use of technical HCH as an intermediate in chemical manufacturing will be phased out by the end of 2007 at the latest (Regulation (EC) No 850/2004). HCHs are also one of the priority substances (Decision No 2455/2001/EC) of the adopted EU Water
Framework Directive 2000/60/EC.

Hexachlorocyclohexane isomers, including the beta-isomer, are on the List of Chemicals for Priority Action under the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic. The objective is the prevention of pollution of the maritime area by continuously reducing discharges, emissions and losses of hazardous substances.

SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

1. SOURCES

1.1. Production

Beta-HCH by itself is neither intentionally produced nor placed on the market. It is produced as constituent of technical HCH used as organochlorine insecticide or chemical intermediate to manufacture enriched HCH (lindane). Currently no production data on technical HCH have been reported, whereas manufacture of lindane still takes place (IHPA, 2006).

HCH is manufactured by photochemical chlorination of benzene which leads to the formation of mainly five stable HCH isomers. The yields of different isomers vary due to technical differences in the production process. The reported ranges are: alpha-HCH (55-80%), beta-HCH (5-14%), gamma-HCH (8-15%), delta-HCH (6-10%) and epsilon-HCH (1-5%) (Breivik et al., 1999). Further details on the production and reuse of HCH residuals can be found in UNEP/POPS/POPRC.2/17/Add.4 (Risk Profile on Lindane) and IHPA (2006).

The following countries which submitted information according to Annex E stated that there was currently no production or use of beta-HCH: Czech Republic, Germany, Mauritius, Mexico, Norway, Qatar, Republic of Lithuania, Turkey, Switzerland and the United States of America.

1.2. Trade and stockpiles

Technical HCH was rapidly introduced in the 1940s on a large scale on the market due to its universal insecticidal properties. The promising market opportunities worldwide arose in the search for an inexpensive alternative to DDT (IHPA, 2006). However due to the decreasing effectiveness of the gamma> alpha> beta-isomer in controlling insects (Baumann et al., 1980) technical HCH was gradually replaced by lindane (> 99 % gamma-HCH). However the manufacture of lindane has resulted in a huge amount of HCH residuals, which must be disposed of or otherwise managed. IHPA (2006) calculated 1.9 to 4.8 million tons of HCH residuals based on global lindane production, in absence of exact data. These estimates are far beyond the values reported by Walker et al. (1999) who reported stockpiles of approximately 2 785 tons of technical HCH and 45 tons of unspecified HCH material in Africa and the Near East.

1.3. Uses

Around 10 million tons of technical HCH were released to the environment between 1948 and 1997 (Li et al. 1999). Breivik et al. (1999) estimated technical HCH usage at approximately 400 000 tons technical HCH in Europe alone between 1970 and 1996. The data illustrate the large uncertainties of these estimates.

According to Li and Macdonald (2005) global usage of technical HCH was dominated by 10 countries headed by China, which consumed almost half of the total global quantity. The other countries were (in order of decreasing usage): Former Soviet Union, India, France, Egypt, Japan, United States, East Germany, Spain and Mexico. Usage of technical HCH was banned in most western countries and Japan in the 1970s but continued in China and Russia until 1983 and 1990. In 1990, India also banned technical HCH for agricultural use but kept it for public health uses (AMAP, 2004). Technical HCH usage steadily declined and is now virtually out of use worldwide. However, there are indications that the use of stockpiles, limited use for
public health purposes and/or illegal use cannot be excluded (Zhulidov et al., 2000; Bakore et al., 2003; Qian et al., 2006).

1.4 Releases to the environment

There are several pathways of beta-HCH for entering the environment. Historically beta-HCH was released during the manufacture of technical HCH and its use as a pesticide. Li et al. (2003) estimated global emissions of beta-HCH from the usage of technical HCH between 1945 and 2000 at 850 thousand tons, of which 230 thousand tons were emitted into the atmosphere over the same period. In 1980 the usage of beta-HCH was around 36 kg tonnes, and the calculated primary emissions were 9.8 kg tonnes (83 % attributed to the application and 17 % to soil residues due to prior applications). In 1990 figures dropped to 7.4 (usage) and 2.4 thousand tons (emissions). In 2000, emissions of beta-HCH from soil residues were 66 tonnes in the absence of direct usage of technical HCH. Also, as a result of the ban on technical HCH in northern countries, global emissions of beta-HCH have undergone a “southward tilt” (Li et al., 2003).

Releases of beta-HCH into the environment are also possible from hazardous waste sites (USEPA, 2006), stockpiles and residues of lindane production, which are not always controlled or maintained safely (IHPA, 2006). Also, contaminated sites (e.g. from former production plants) may contribute to the environmental burden of beta-HCH (Concha-Grana et al., 2006). Germany (submitted Annex E information) reported that there are still a few isolated local sources i.e. landfills and dumps in the former GDR (East Germany) from applications of technical HCH. As a result higher concentrations of beta-HCH in fish of the river Elbe near the former production site were detected after heavy rainfalls and floods in 2003. However, quantitative estimates of releases from hazardous waste sites and landfills are not available.

2. ENVIRONMENTAL FATE

2.1 Persistence

2.1.1. Abiotic Degradation

Investigations of the hydrolysis and photolysis of beta-HCH are extremely limited. Only one literature study regarding photodegradation has to date been available. A photodegradation half-life for a thin film of beta-HCH equal to 152 hours has been reported (ATSDR, 2005). The relevance of this result is questionable with respect to the chosen test design which does not comply with internationally accepted test guidelines on photolysis and, as pointed out by ATSDR (2005) no absorption bands were observed in the studied spectral region. In general photolysis is not expected to be an important environmental fate process for beta-HCH since no absorption of light > 290 nm takes place.

Based on the calculated atmospheric OH rate constant of $5.73 \times 10^{13}$ cm$^3$/molecule-sec (HSDB, 2003) the estimated half-life is 56 days (using an average hydroxyl radical concentration of $5 \times 10^5$ molecule/cm$^3$ according to the TGD (2003)).

USEPA (2006) concluded that in general HCH isomers are resistant to abiotic processes like photolysis and hydrolysis (except at basic pH).

2.1.2. Biotic Degradation

Beta-HCH is in principle biodegradable under oxic and anoxic conditions. However several studies have suggested that significant degradation does mainly occur under anaerobic conditions (Middeldorp et al., 1996). Degradation was observed in pure cultures, soil slurry, soil microcosm, field studies and via bioremediation techniques in the soils of contaminated sites (Phillips et al., 2005). Effectiveness of removal varied depending on the test design and environmental factors.
In general the metabolic pathway of beta-HCH occurs anaerobically via dechlorination to tetrachlorocyclohexane and dichlorocycohexadiene, an unstable metabolite. Chlorobenzene and benzene were formed as stable end products under methanogenic conditions. These metabolites can be further aerobically or anaerobically mineralised (Phillips et al., 2005). Compared to other HCH isomers laboratory data using radio-labelled beta-HCH have shown only minimal and incomplete mineralization (Sahu et al., 1995).

Beta-HCH is considered to be the most recalcitrant isomer due to its chemical structure (Decision POPRC-2/10, 2006). Under favourable laboratory conditions several strains of bacteria e.g. Bacillus brevis, Bacillus circulans, Dehalobacter sp. in conjuction with Sedimentibacter sp., isolated from HCH polluted sites, have been identified as beta-HCH degraders (Gupta et al., 2000; van Doesburg et al., 2005). But only a few were able to transform beta-HCH under aerobic conditions e.g. Sphingobium sp. (Sharma et al., 2006).

Research on the intrinsic stimulation and additives for soil bioremediation of beta-HCH polluted sites is under way (e.g. Kumar et al., 2005; MacRae et al., 1984) but to remove the isomer remains a difficult challenge (Phillips et al., 2005). Regarding the effects on the intrinsic soil microbial population of an uncontaminated soil, Bhatt et al. (2006) showed that the application of technical HCH disturbed the microbial community irreversibly.

In general climatic conditions as well as soil texture and organic matter altering substance sorption, water content, pH and bacterial growth influence degradation rates (IPCS, 1992). Phillips et al. (2005) stated that bacteria capable of degrading HCHs at extreme temperatures (< 5 °C or > 40°C) have not yet been reported.

Data on laboratory soil studies or field investigations are limited. Singh et al. (1991) reported half-lives of 100 and 184 days on cropped and uncropped plots in a sandy loam in India under subtropical conditions. The applied formulated HCH was immediately incorporated into the top layer of the soil. Soil samples were taken randomly from the plots in 0-15 cm depths. No quantitative information on losses of beta-HCH by volatilisation or leaching during the experiment is available in the cited study. In temperate climate Doelman et al. (1990) observed in a semi-field study with contaminated soil no degradation of the beta-isomer under anaerobic conditions. Stewart and Chisholm (1971) observed in a long-term field study after an application of technical HCH, 44 % of the beta-isomer after 15 years in a sandy loam in Canada. Approximately 30 % of beta-HCH (from applied technical HCH) was observed after 570 days in a field test in Japan on agricultural field plots (Suzuki et al., 1975). Also Chessells et al. (1988) showed that after a 20 year application history of technical HCH on sugar cane in Queensland, Australia, beta-HCH occurs in concentrations which are more than one order of magnitude higher compared to the other isomers.

Based on the \( K_{oc} \) value and confirmed by field data beta-HCH is expected to have a low leaching potential and also volatilisation from soil surfaces is considered not to be an important fate process (HSDB, 2006; Singh et al., 1991).

Beta-HCH was stable in a sediment/water study under laboratory conditions. In addition, isomerisation of alpha- to the beta-isomer was observed (Wu et al. 1997). Detailed information regarding isomerisation can be found in the risk profile on lindane (UNEP/POPS/POPRC.2/17/Add.4). Levels of the beta-isomer compared to alpha-, gamma- and delta-HCH were highest in porewater (1 423 ng/l) compared to concentrations in surface water (92.5
ng/l) and sediment (3.9 ng/g) of the Minjiang River Estuary, China (Zhang et al., 2003). No degradation half-lives in water or sediment are available; however, based on monitoring studies, it can be assumed that beta-HCH is persistent and does not undergo degradation easily.

2.2. Bioaccumulation

The octanol-water partition coefficient (log $K_{ow} = 3.78$) for beta-HCH indicates that it has a potential to bioaccumulate, especially in combination with its shown persistence in animal tissue (Walker et al., 1999).

The BCF according to the former OECD test guideline 305 E in zebra fish was equal to 1460, which was the highest BCF compared to determined values for alpha- (1100) and gamma-HCH (850) (Butte et al., 1991). According to the ECOTOX database this was also the highest reported BCF.

Several studies suggest that the relative proportions of HCH isomers vary dramatically across species in the Arctic marine food web (USEPA, 2006). Concentrations of beta-HCH increased with the trophic level especially in upper trophic levels (marine mammals) (USEPA, 2006; Hoekstra et al., 2003). Whereas it is assumed that organochlorine (OC) profiles in mammals are mainly influenced with regard to their ability to biotransform and excrete OCs, high detected levels of beta-HCH in various mammalian species are another indication of its recalcitrant nature and slow elimination. Hop et al. (2002) showed that beta-HCH biomagnifies differently in poikilotherms and homeotherms. Beta-HCH increased more among homeotherms (birds and mammals) with the trophic level. Fisk et al. (2001) reported the highest BMF (biomagnification factor) in birds compared to the other trophic levels, but migration and prey items are also considered to influence the variability of the BMFs. These data are in line with findings from Moissy et al. (2001). In general, studies from Arctic marine food webs show that BMFs for nearly all examined species as well as obtained food web magnification factors (FWMFs), which represent the mean rate of increase per trophic level in the food chain, are greater than 1. E.g. Fisk et al. (2001) reported a FWMF of 7.2 which is comparable to higher chlorinated PCBs. A FWMF of 2.9 was calculated by Hoekstra et al. (2003) for the marine food web in the Beaufort-Chukchi Sea. However in sub-Arctic waters, the White Sea, values for for beta-HCH were lower compared to the other food web studies. Differences in feeding habits and availability/levels of contaminants were suggested as being responsible by Muir et al. (2003).

Also, in the terrestrial food chain, beta-HCH may biomagnify. Data obtained from an investigation in south India showed that HCHs were the predominant OCs in biota. Elevated concentrations were measured in snails and subsequently their predators (e.g. little egret) showed BMFs above 1 (Senthilkumar et al., 2001). Also Wang et al. (2006) found beta-HCH as a major compound in molluscs (submitted Annex E information by IPEN).

Fish, marine and terrestrial mammals as well as birds are the major nutrition sources of several Arctic population groups and thus exposure through diet is much more likely than for most populations in the developed world. Levels of beta-HCH in breast milk among women from indigenous people on the Chukotka Peninsula, Russia (Chukotsky rayon, mean value 370 ng/g lipids) are highest compared to other northern towns of Russia and to levels in Canada (Nunavik, by 30 times) (AMAP, 2004). Also, concentrations of maternal blood sampled between 1994 and 1997 were highest in Russian mothers (Arctic non-indigenous population, serum concentration 223 µg/kg lipid), but elevated levels were also found in Iceland (23 µg/kg) and in the Canadian Arctic (AMAP, 2003). Native women in Alaska also showed elevated plasma levels (25 µg/kg) (Rubin et al., 2001 in Annex E information submitted by IPEN, no information on which basis the concentrations are expressed). Based on concentrations of beta-HCH in the human diets from several countries (0.56 – 1.21 µg/kg) and corresponding levels in adipose tissue (0.27– 0.9 mg/kg,
respectively), the mean bioaccumulation factor (on a lipid base) for beta-HCH in humans was 527 (range 310 - 744). When beta-HCH is administered repeatedly to rats, mice, and mini-pigs, there is increased storage in fat, especially in females (IPCS, 1992).

2.3. Long-range environmental transport

Many studies and monitoring data have detected beta-HCH regularly in the Arctic environment as well as in biota (e.g. AMAP, 2004; AMAP, 2003). Because technical HCH including beta-HCH was never extensively used in this remote area, this is evidence of its long range transport (UNEP/POPS/POPRC2./17/Add.4).

Based on monitoring data from Arctic air, beta-HCH appears to be less subject to direct atmospheric loading into the high Arctic. This can possibly be explained by differences in the Henry’s law constant and the air/octanol partition coefficient that enhance affinity to organic matter (Li et al., 2002). Thus rain scavenging is much more efficient for beta- than for alpha-HCH and besides, the frequency of precipitation is considerably higher in the North Pacific compared to the Arctic. This suggests that beta-HCH enters the Arctic probably by mechanisms involving wet deposition or partitioning into the North Pacific surface water and subsequently entering the Arctic in ocean currents passing through the Bering Strait (Li et al., 2003). Bering and Chukchi Seas are the most vulnerable locations for beta-HCH loadings (Li et al., 2002). Concentrations of beta-HCH around the Bering Strait in the 1990s reached approximately 1.2 ng/l (Li and Macdonald, 2005). Thus “cold condensation” also occurred for beta-HCH, but mainly in the Pacific Ocean and Bering Sea upstream of the Artic Ocean. Thus beta-HCH reached the Arctic later compared to alpha-HCH and differed in its spatial distribution (Li et al., 2002). This spatial and temporal distribution is also reflected in residue levels in marine and terrestrial mammals as well as in local residents (Li and Macdonald, 2005).

Measurement of beta-HCH in high mountains in the Czech Republic is another proof for its long range transport potential (submitted Annex E information by the Czech Republic).

According to model calculations with the OECD Pov and LRTP Screening Tool beta-HCH has similar persistence and long-range transport properties compared to already identified POPs such as PCBs and OCs (submitted Annex E information by Switzerland). Model input properties of the chemicals include partition coefficients in air-water and octanol-water as well as half-lives in air, water and soil and the Henry’s Law constant (based on figures contained in UNEP/POPS/POPRC2./INF/8). The model considers all environmental compartments quantitatively. The results of the model do not indicate absolute levels in the environment but help to compare possible POPs with identified POPs (reference chemicals: PCB congeners 28, 101, 180, HCB, carbon tetrachloride and alpha-HCH) according to their environmental persistence and potential for long range transport. Uncertainties in the chemical properties were investigated by Monte Carlo uncertainty analysis.

3. EXPOSURE

Direct exposure to beta-HCH resulted from the production (including manufacture of lindane) and use of technical HCH. Because of the persistence high exposure is also expected in contaminated areas due to extensive use, former production, disposal sites and stockpiles. Though usage of technical HCH has practically ceased worldwide monitoring data based on the ratio of the alpha/gamma-isomer still suggest possible releases of technical HCH in certain areas (Zhang et al. 2003; Qian et al., 2006; Zhulidov et al., 2000).

Exposure of the general public results mostly from the ingestion of contaminated plants, animals and animal products. Inhalation of ambient air and consumption of drinking water are further sources of exposure, although to a minor extent. Intake through indoor air may be considerable for people living in houses treated for pest-control purposes.
3.1. Environmental monitoring data from local areas

Generally environmental levels in local areas have dropped after restrictions and prohibitions of the usage of technical HCH (IPCS, 1992). However, monitoring data show its ubiquitous distribution in all environmental media. E.g. beta-HCH (up to 15 µg/kg dry substance) has been detected using passive monitoring in lichens in various locations (e.g. cities, industry, rural) in Switzerland (submitted Annex E information by Switzerland). Also, a recently performed monitoring programme in Japan revealed that beta-HCH had been detected in all specimens. The reported values (range) are as follows: water 0.031-3.4 ng/l, sediment 0.004-53 ng/g dry weight, shellfish 0.22-1.8 ng/g wet weight, fish trace-1.1 ng/g wet weight, bird 1.1 – 4.8 ng/g wet weight, air (warm and cold season) 0.53 – 110 pg/m$^3$ and 0.32 – 78 pg/m$^3$ (submitted Annex E information by Japan). The Czech Republic (Annex E information) reported that, with regard to HCHs, the most severe situation is in central and southern Moravia, where sediment particles are found in amounts of tens of ng/g and in some cases even in hundreds of ng/g (no information on which basis the concentrations are expressed was submitted).

However, heavily contaminated soils were found in the proximity of sources. HCH concentrations of 40 - 225 mg/kg were found in the topsoil around a chemical plant in Albania. Mean levels of 0.02 mg/kg were reported for soils from the Pearl River Delta in China, Russian soils near the Lena River contained 0.001-0.017 mg/kg HCH (UNEP, 2003).

Compared to the other HCH isomers, concentrations of beta-HCH in the air are low. Elevated levels were detected in higher mountains (Mount Everest Region) of 11.2 pg/m$^3$ compared to up to 1 pg/m$^3$ in the Arctic (Li et al., 2006). Seasonal changes in beta-HCH concentrations in Japan (mean 23 pg/m$^3$) in 2000 were probably caused by re-emissions from a terrestrial source (Murayama et al., 2003).

Unlike alpha- und gamma-HCH observed concentrations of beta-HCH in air at most locations near the Great Lakes in North America did not show significant trends between 1990 and 2003. The highest concentration was observed in Chicago with a maximum of 73 pg/m$^3$ (mean 12 pg/m$^3$, 1999-2003, gas phase, Sun et al., 2006a). Regarding the occurrence of beta-HCH in precipitation samples from the same region (mean concentrations 0.16 - 0.64 ng/l) a significant increase in beta-HCH concentrations at three Great Lakes stations over the last decade was observed (Sun et al., 2006b).

Levels in biota vary, depending on the location (recent usage and/or high pollution) and species. E. g. concentrations of HCHs (mainly the beta-isomer) in one fish species (*Java tilapia*) from India amounted to up to 2 000 ng/g wet weight (Senthilkumar et al., 2001). Fish samples collected from the Nile River near Cairo in 1993 showed a concentration of beta-HCH of 1.5 ng/g wet weight (UNEP, 2003). Alpha-HCH is in most cases the dominant isomer in fish (Willett et al., 1999).

Beta-HCH was also detected in eggs of Dalmatian Pelican (*Pelecanus crispus*) as well as in eels (*Anguila anguila*), the main pelican prey species collected in the wetlands of Amvrakikos Gulf in Greece for a two year period, 1992 and 1993. The concentration in pelican eggs was $16.4 \pm 5.4$ ng/g and $10.1 \pm 4.2$ ng/g wet weight in eels (UNEP, 2003).

Birds and bats can accumulate higher concentrations of beta-HCH. According to submitted Annex E information submitted by Norway Bustnes et al. (2006) concluded that beta-HCH levels in blood and eggs were higher in the endangered subspecies of the black-backed gulls in Norway. One explanation might be the migration route through the Black Sea where HCH levels are considerable high.
In a study of resident and migratory birds collected from South India, the organochlorine contamination pattern varied depending on the migratory behaviour. Resident birds living in the same region for their entire life span contained relatively greater concentrations of HCHs (14 - 8800 ng/g wet weight). Long distance migratory birds which have their breeding grounds in Europe, Russia, the Middle East, Papua New Guinea and Australia contained HCHs at levels of 19 - 5500 ng/g. Among various HCH isomers, beta-HCH was the predominant contaminant in all the bird species (UNEP, 2003). Similar levels were reported in a later investigation (Senthilkumar et al., 2001) which included the determination of HCHs concentrations (mainly the beta-isomer, up to 330 ng/g wet weight) in Indian bats, which were higher in 1998 than in 1995 and compared to other parts of the world.

A local source of beta-HCH was the usage of technical HCH in the Russian North against nuisance insects on domesticated reindeer by indigenous human populations (Li et al., 2004). However, no quantitative estimates of these exposure levels exist.

3.2. Exposure as a result of long-range environmental transport

The main transportation pathway of beta-HCH to the Arctic is assumed to be ocean currents (Li et al., 2002). Compared to levels of alpha-HCH in sea water, beta-HCH levels were lower - partly due to reduced emissions and different spatial and temporal distributions, e.g. beta-HCH reached its peak (approximately 0.3 ng/l) in the North American Arctic Ocean in 1994, around 10 years after the alpha-HCH levels had reached their peak. Enrichment of the upper waters of the North Pacific Ocean and Bering Sea (approximately 1.3 ng/l 1988-1999) caused higher concentrations in the Chukchi Sea and subsequent decreases towards the Arctic interior ocean (Li and Macdonald, 2005). Data on beta-HCH from surface water of the Canadian Archipelago in 1999 showed concentrations of 0.1 ng/l (Bidleman et al., 2007).

This spatial distribution is also reflected in the levels in biota. Hoekstra et al. (2002) found that bowhead whales exhibit a reversal in their blubber alpha-/beta-HCH ratios on their migration route between the Bering to the Beaufort Sea. Also elevated residues of HCH isomers in marine mammals of the Canadian Archipelago are likely from the high concentrations of HCH isomers in the water because HCH isomers are the most abundant organochlorines in the Arctic Ocean (NARAP, 2006).

Beta-HCH is not so abundant in the Arctic abiotic environment and therefore it has not been studied as well as the other HCH isomers, partly due to its low concentrations. Measured levels in the Arctic air (e.g. < 1 pg/m³ from six Arctic circumpolar located sites between 2000-2003, Su et al. (2006)) and in terrestrial as well as freshwater ecosystems were low (AMAP, 2004). HCHs also show a high degree of spatial variability in the levels of contamination across the Russian North (AMAP, 2004).

Levels in the Arctic terrestrial environment (including carnivores) are much lower than in the marine compartment and its predators. However, beta-HCH has been detected in the fat of male Arctic foxes (up to 810 ng/g wet weight) in Alaska (AMAP, 2004). The highest levels of HCH in polar bears were detected in the Beaufort Sea population (approx. 770 ng/g wet weight in fat). Beta-HCH accounted for 93 % of HCH residues.

The metabolism of beta-HCH is very limited in Arctic seabirds, and therefore beta-HCH is detected more readily than alpha- and gamma-HCH. But concentrations vary notably between species, depending on the trophic position and migration. Higher levels of beta-HCH were observed in the North American Arctic in closer proximity to Asia where HCH was recently used. Levels were below 1 ng/g in bird tissue and 30 ng/g wet weight in eggs (AMAP, 2004).

Regarding temporal trends, it was shown that beta-HCH levels in seabirds, ringed seals and polar bears increased, whereas belugas showed no difference from 1982
3.3. **Food**

Daily intake values of beta HCH for the general population in adult diets between 1986 and 1991 in the United States were reported to be below 0.001 µg/kg/day. In the Total Diet Study conducted by FDA in 1990 on 935 food items, beta-HCH was detected in 11 items. The average concentration of beta-HCH in 234 ready-to-eat foods was 0.0027 µg/kg (no information on which basis the concentrations are expressed, ATDSR, 2005). In the USA, the average daily intake of beta-HCH was <0.1-0.4 ng/kg body weight (bw) (depending on age) during the years 1982-1984 and was generally below 0.1 ng/kg bw during the years 1986-1991 (ATDSR, 2005). In a total diet study from Canada (1993-1996), an average daily dietary intake of 0.39 ng/kg bw beta-HCH was reported (EFSA, 2005). In fat-containing food products, levels ranged up to 0.03 mg/kg (fat) but in milk products levels up to 4 mg/kg (fat) were detected (WHO, 2003). In the United States and Canada levels in food are slowly decreasing. Within the European countries representative dietary intake studies are scarce. One was performed in the Czech Republic. The median intake values for beta-HCH declined from 8.4 ng/kg bw in 1994 to 2.1 ng/kg bw in 2002 (EFSA, 2005). A local diet study from Spain showed elevated daily intakes of 0.1 µg beta-HCH (Urieta et al., 1976).

Fish and clam samples from India contained 0.001 and 0.02 mg beta-HCH/kg wet weight respectively (Nair and Pillai, 1992).

Because of the global trade of feeding stuffs, feed ingredients and food products from regions with ongoing or recent use of HCHs, which are supposedly more contaminated, might be imported by countries where technical HCH has already been phased out.

High levels of beta HCH levels in food are documented for the Arctic Region (AMAP, 2004). Subsistence foods in Alaskan communities from the years 1990 to 2001 were analysed for total HCH in order to estimate dietary intakes by indigenous people. Highest concentrations were found in marine mammals, whale (391 ng/g) and seal (215 ng/g). High concentrations were documented for walrus (20 ng/g), whitefish (20 ng/g) and salmon (26 ng/g). Berries contained 10 ng/g and ducks 7 ng/g (USEPA, 2006).

3.4. **Body burden**

3.4.1 **General population**

Beta-HCH is the most prevalent HCH-isomer in fatty tissue. The half-life of beta-HCH after inhalation exposure in the body is 7.2 - 7.6 years (ATDSR, 2005). Human biomonitoring studies in the United Stated showed that median levels of beta HCH in post-mortem human adipose tissue samples decreased over time (0.45 ppm in 1970 to 0.16 ppm since 1981) (ATDSR, 2005).

A comparison between body compartments showed median levels of 0.13ng/g in whole blood and 18ng/g in adipose tissue (ATDSR, 2005).

According to the results of the National Reports on Human Exposure to Environmental Chemicals, beta-HCH serum concentrations in the US population have been declining since 1970. For all tested age groups (12 years and older), the 95th percentile of beta-HCH serum concentrations on a lipid-weight basis decreased from 68.9 in the years 1999-2000 to 43.3 ng/g in the years 2001-2002. Concentration levels (2001/2002) in females were higher (54.5 ng/g) than in males (29.2 ng/g). Highest concentration levels were found in the Mexican Americans (84.4 ng/g). Comparably low levels were found in the age group 12-19 years (8.44 ng/g) (CDC, 2005). Age-
related increases in the levels of beta-HCH have been observed in several studies and documented by the German Commission on Biological Monitoring (Ewers et al., 1999).

Comparably high concentrations were detected in human blood serum samples from Romania. Beta-HCH was detected in all samples (n = 142) with a median concentration of 923 ng/g lipid (range 38-11690 ng/g) (Dirtu et al., 2006). High concentrations were reported for India due to agricultural use and Malaria control activities. Blood serum samples from India contained up to 0.02 mg beta-HCH/l, whereas adipose tissue contained up to 0.18 mg/kg (Nair and Pillai, 1992).

3.4.2. Indigenous population

Beta HCH concentrations in blood plasma samples from different regions and ethnic groups of indigenous mothers of the Arctic were 0.04 - 0.11 µg/l (Canada), 0.07-0.56 µg/l (Greenland), 0.12 - 0.53 µg/l (Alaska), 0.31 - 3.1 µg/l Russian Arctic (maximum level: 11.6 µg/l), 0.16 - 0.21 µg/l (Iceland), 0.05 - 0.09 µg/l (Norway, Finland and Sweden) and 0.11 µg/l from the Faroe Islands (AMAP 2004; values given as geometric means, with the exception of Alaska which are given as arithmetic means). The highest concentrations in blood samples of the indigenous population were reported for the Russian Arctic.

Comparative investigations of the maternal blood and cord blood of indigenous mothers for beta-HCH in the Russian Arctic were highly dependent on the residential area. The mothers with the highest exposure (Chukotksky District) had blood concentrations (µg/l plasma, geometric mean and range) of 2.0 (0.6 - 7.6) µg/l whereas the cord blood contained 0.8 (n.d.-8.0) µg/l (AMAP, 2004:2).

3.5. Exposure of children

Children are at specific developmental stages more vulnerable against chemical substances than adults. It is not known if children are more susceptible than adults to health effects from exposure to beta-HCH. Placental transfer of HCH in humans has been well documented (ATDSR, 2005; Falcon et al., 2004; Shen et al., 2006). Beta-HCH is lipophilic and accumulates in adipose tissue and breast milk, which is another relevant exposure source for children. Several studies concerning beta-HCH in breast milk are listed in table 2. It could be shown, that in case of restrictions of use, concentrations are constantly declining. Within a continuous surveillance in Japan it was demonstrated that compared to 1970, when contamination of breast milk was highest, the beta-HCH levels fell to about 3 % in 1992 (Konishi et al., 2001). In China a considerable reduction within 10 years was observed, although concentrations are still high (~1 µg/g) (Wong et al., 2002). In Germany beta-HCH levels fell from 0.12 mg/kg milk fat in 1984 to 0.02 mg/kg milk fat in 2001 (Fürst, 2004), another German study showed a notable decrease in contamination of human milk between 1984/85 and 1995 as well (Ott et al., 1999). Declining beta-HCH levels were also found in studies from Sweden and Norway.

It can be concluded that beta-HCH concentrations in breast milk are highly exposure-dependent. Whereas in some areas concentrations are very low, i.e. 13 ng/g in Poland, in other areas i.e. Russia, Ukraine, Romania they are very high (up to > 800 ng/g). In general it can be expected that in several East European and developing countries concentrations are still very high. Especially high concentrations were reported for India and China (Wong et al., 2002). Extremely high levels were also reported for cotton pickers in Pakistan (UNEP, 2003).

Due to bioaccumulation in the Arctic marine food web, high concentrations were found in the breast milk of indigenous mothers of Arctic regions despite the absence of local sources. This is due the fact that indigenous mothers rely on the consumption
of local foods with high levels of beta-HCH.

Table 2: Concentrations of beta-HCH in breast milk

<table>
<thead>
<tr>
<th>Country/region</th>
<th>Levels (on lipid basis)</th>
<th>Comments</th>
<th>References</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Germany</td>
<td>0.12 mg/kg</td>
<td>Start of Monitoring program 1984</td>
<td>Fürst et al. in EFSA, 2005</td>
<td>1984</td>
</tr>
<tr>
<td>Germany</td>
<td>0.02 mg/kg</td>
<td>Continuous Monitoring since 1984</td>
<td>Fürst et al. in EFSA, 2005</td>
<td>2001</td>
</tr>
<tr>
<td>Spain</td>
<td>0.24 µg/g</td>
<td>51 samples</td>
<td>Hernandez et al. in Wong, 2002</td>
<td>1991</td>
</tr>
<tr>
<td>Canada</td>
<td>0.6-0.8ng/g</td>
<td>Lower concentration: population near Great lakes</td>
<td>Mes and Malcolm in ATDSR, 2005</td>
<td>1992</td>
</tr>
<tr>
<td>Canada</td>
<td>0.02 µg/g</td>
<td>497 samples</td>
<td>Newsome and Ryan in Wong, 2002</td>
<td>1992</td>
</tr>
<tr>
<td>Brazil</td>
<td>0.27 µg/g</td>
<td>40 samples</td>
<td>Paumgartten et al. in Wong, 2002</td>
<td>1992</td>
</tr>
<tr>
<td>Russia Murmansk</td>
<td>853 ng/g</td>
<td>15 samples</td>
<td>Polder et al. in Dirtu, 2006</td>
<td>1993</td>
</tr>
<tr>
<td>Russia Nonchegorsk</td>
<td>740 ng/g</td>
<td>15 samples</td>
<td>Polder et al. in Dirtu, 2006</td>
<td>1993</td>
</tr>
<tr>
<td>Ukraine</td>
<td>731 ng/g</td>
<td>200 samples</td>
<td>Gladen et al. in Dirtu, 2006</td>
<td>1993-94</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>71 ng/g</td>
<td>17 samples</td>
<td>Schoula et al. in Dirtu, 2006</td>
<td>1993-94</td>
</tr>
<tr>
<td>Kazakhstan</td>
<td>2.21µg/g</td>
<td>33-76 samples</td>
<td>Hooper et al., in Won, 2002</td>
<td>1994</td>
</tr>
<tr>
<td>Siberian Russia</td>
<td>40-142 µg/kg (geom.means)</td>
<td>Arctic Monitoring Assessment Programme</td>
<td>Klopop et al. 1998, 2000 in AMAP 2004</td>
<td>1994-95</td>
</tr>
<tr>
<td>Northern Russia</td>
<td>120 -401 µg/kg (geom.means)</td>
<td>Arctic Monitoring Assessment Programme</td>
<td>Polder et al. in AMAP 2004</td>
<td>1994-95</td>
</tr>
<tr>
<td>Australia</td>
<td>0.35µg/kg</td>
<td>60 samples</td>
<td>Quinsey et al in Wong, 2002</td>
<td>1995</td>
</tr>
<tr>
<td>Afrika, Uganda,</td>
<td>0.005-0.25 mg/kg</td>
<td>-</td>
<td>Ejobi et al. in ATDSR, 2005</td>
<td>1996</td>
</tr>
<tr>
<td>India</td>
<td>8.83 µg/kg</td>
<td>Delhi, Age group: 20-30 61 samples</td>
<td>Banerjee et al. in Wong, 2002</td>
<td>1997</td>
</tr>
<tr>
<td>India</td>
<td>0.022-0,078 mg/kg</td>
<td>Region under Malaria control</td>
<td>Dua et al. in ATDSR, 2005</td>
<td>1997</td>
</tr>
<tr>
<td>Pakistan</td>
<td>0-0.90 mg/kg</td>
<td>Cotton pickers</td>
<td>Masud and Parveen, 1998 in UNEP, 2003</td>
<td>1998</td>
</tr>
<tr>
<td>Nairobi, Kenya</td>
<td>0.0830-0.026 mg/kg</td>
<td>Urban population</td>
<td>Kinyamu et al.</td>
<td>1998</td>
</tr>
<tr>
<td>Japan, Osaka,</td>
<td>5.43 µg/g</td>
<td>Estimated use in Japan: 400 000 tons</td>
<td>Konishi et al. 2001</td>
<td>1972</td>
</tr>
<tr>
<td>Japan, Osaka,</td>
<td>0.21 µg/g</td>
<td>Ban of organochlorine compounds in 1970ies</td>
<td>Konishi et al. 2001</td>
<td>1998</td>
</tr>
<tr>
<td>Romania, lassay</td>
<td>640 ng/g</td>
<td>19 samples</td>
<td>Covaci et al. in Dirtu, 2006</td>
<td>2000</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>56 ng/g</td>
<td>43 samples</td>
<td>Cajka and Hajslova in Dirtu, 2006</td>
<td>2000</td>
</tr>
<tr>
<td>China, Hong Kong</td>
<td>15.96 µg/g</td>
<td>Uncontrolled agricultural use</td>
<td>Wong et al. 2002</td>
<td>1985</td>
</tr>
<tr>
<td>China, Hong Kong</td>
<td>0.95 µg/g</td>
<td>115 samples</td>
<td>Wong et al. 2002</td>
<td>1999</td>
</tr>
<tr>
<td>China, Guangzhou</td>
<td>1.11 µg/g</td>
<td>54 samples</td>
<td>Wong et al. 2002</td>
<td>2000</td>
</tr>
</tbody>
</table>
3.6. Information on Bioavailability

Beta-HCH is moderately associated with organic matter in the environment. Uptake by plants and residues in vegetation as well as by food and feed is well documented (Willet et al. 1998; ATSDR, 2005; EFSA, 2005). Though beta-HCH is not assumed to be very mobile in soil there have been cases of groundwater contamination in the past (HSDB, 2006).

In biota, beta-HCH is selectively accumulated in certain tissues (e.g. liver, muscle, fat) and affects several organs (Willett et al., 1998). It can be concluded that beta-HCH is bioavailable in the environment and in biota.

4. Hazard assessment for endpoints of concern

4.1 Human Health

Information on the toxicity of beta-HCH is mostly derived from experimental studies in animals. Compared to lindane, the data available are limited, especially concerning human data because occupational exposure occurs mainly with technical-grade HCH and lindane.

Studies of acute/short-term toxicity via the oral route, subchronic and chronic oral toxicity studies and a limited number of studies of reproductive effects are available. No studies of the toxicity of beta-HCH via inhalation and dermal application have been conducted. There is a lack of dose-response data after oral exposure in all relevant species. For the present risk profile, the most important findings concerning the hazard assessment have been reviewed. For further studies and details the more comprehensive toxicological profiles should be consulted (IPCS, 1992; ATDSR, 2005; EPA, 2006).

**Acute Toxicity/ Neurotoxicity**

The concentration range for lethal acute toxic effects is - according to IPCS (1992) - 150 mg/kg to > 16000 mg/kg in mice and 600 mg/kg to > 8000 mg/kg in rats. Symptoms of acute toxicity affect mainly the nervous system: excitation, hunched posture, rough fur, dyspnoea, anorexia, tremors, convulsions and cramps.

**Subchronic toxicity**

In a 13-week study in rats, the effects of oral exposure to beta-HCH (0, 2, 10, 50, 250 mg/kg diet) were investigated. In all dose groups, liver effects were observed. At the highest dose tested (250 mg/kg diet) half of the animals died following ataxia, progressive inactivity, and coma. Observed effects included growth inhibition, decrease of red and white blood cells, increase of liver enzymes and liver effects (increase in organ weight, centrilobular hepatocytic hypertrophy). A decrease in thymus weight (50 and 250 mg/kg) and atrophy of the testes were observed. The females showed atrophy of the ovaries with impaired oogenesis and focal hyperplasia as well as metaplastic changes of the endometrial epithelium, which was interpreted as a possible estrogenic action of beta-HCH (van Velson, 1986). A NOAEL of 2 mg/kg diet (equivalent to 0.1 mg/kg bw/day) was established (IPCS, 1992; EFSA, 2005).

**Chronic Toxicity**

A long-term study (52 weeks) in rats with 10, 100 and 800 mg/kg beta-HCH in their diet (i.e. 0.5, 5 and 40 mg/kg bw/day) led to liver enlargement and histological changes. Nearly all animals died.

A two-generation reproduction study of rats exposed to 10 mg/kg diet resulted in increased mortality and infertility. The NOAEL was 2 mg beta-HCH/kg diet (equivalent to 0.1 mg/kg
Genotoxicity
Beta-HCH was not mutagenic to bacteria (Salmonella typhimurium strains TA 98, TA 100, TA 1535 and TA 1537) with and without metabolic activation and did not induce DNA damage in bacteria. Positive results were seen in an in-vivo rat bone marrow chromosomal aberration study (EFSA, 2005).

Carcinogenicity
Studies of the carcinogenicity of beta-HCH are limited. Several studies in mice were performed, but their value is limited. On the one hand their duration, due to high mortality, was too short; on the other hand histopathological evaluations were missing. Studies in rats have been inadequate due to high mortality and small animal numbers.

One study in mice is adequate for an evaluation of the carcinogenicity of beta-HCH. 200 mg/kg beta-HCH in the diet (equivalent to 40 mg/kg bw/day) for 110 weeks led to liver enlargement, hyperplastic changes and an increase in benign and malignant tumours in the exposed mice.

In a 32 weeks study where 0, 100, 300, 600 mg/ per kg diet were given to mice, liver toxicity and atypical proliferation was observed in all dose groups (IPCS, 1992). In a 24-week study in mice - given 0, 50, 100, 200, 500 mg beta-HCH /kg diet - liver tumours and nodular hyperplasia in the highest dose group was observed (IPCS, 1992).

In a 26-month study, liver cancer in mice was observed at a daily dose of 34 mg/kg (ATSDR, 2005). Based on these data beta-HCH has been classified as possible human carcinogen by IRIS (Integrated Risk Information System).

Studies on the mode of action of carcinogenicity showed no clear initiating potential of beta-HCH. In one study the hepatocarcinogenic action of beta-HCH was shown with PCBs as promoting agent (ATSDR, 2005).

It was suggested that the neo-plastic response observed with beta-HCH most likely occurs due to a non-genotoxic mechanism (IPCS, 1992). Beta-HCH has been shown to have tumour-promoting activity.

The International Agency for Research on Cancer (IARC) classified beta- HCH in group 2B: limited evidence for carcinogenicity. A positive association has been observed between exposure to beta-HCH and cancer, for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence. USEPA has classified technical HCH and alpha-HCH as probable human carcinogens and beta-HCH as a possible human carcinogen (ATSDR, 2005).

Neuroendocrine mediated toxicity
Degenerative changes in male reproductive tissues and sperm abnormalities in rats and mice were described (ATDSR, 2005). In a 13-week study, 0, 50, 150 mg beta-HCH/kg diet were given to Wistar rats. At 150 mg/kg diet, atrophy of the testes in males and increase in uterine weights in females and significantly reduced weight gains were reported (IPCS, 1992). Several other studies showed effects such as decrease in sperm counts and sperm abnormalities as well as histological effects on the testes and uterus at high doses of beta-HCH exposure (USEPA, 2006).

Animal studies and a study with MCF-7 cells showed weak estrogenic effects of beta HCH.

Reproductive toxicity
Beta-HCH has been shown to increase fetal deaths within 5 days of birth at a dose of 20 mg/kg/day given to rat dams (USEPA, 2006).

Immunotoxicity
Mice, treated with beta-HCH (60 mg/kg/day) orally for 30 days showed decreased
lymphoproliferative responses to T-cell mitogens and decreased natural killer cytolitic activity. The NOAEL was 20 mg/kg/day (USEPA, 2006). Cortical atrophy of the thymus was observed at a dose of 22.5-25 mg/kg/day (van Velsen et al, 1986).

**Effects in Humans**

Adverse effects such as neurophysiological and neuropsychological disorders and gastrointestinal disturbances have been reported in workers exposed to technical HCH during pesticide or fertilizer formulation. Although beta-HCH is only a minor component of technical-grade HCH, it reached higher levels and persisted longer in the serum than either alpha- or gamma-HCH. 60-100 % of the total HCH measured in serum was beta-HCH (0.07-0.72 ppm ). Workers suffered from paresthesia of the face and extremities, headache and giddiness, malaise, vomiting, tremors, apprehension, confusion, loss of sleep, impaired memory and loss of libido. Serum enzyme levels were enhanced as well as IgM (ATDSR, 2005).

Beta-HCH levels were higher in the blood of women with miscarriages compared to a control group. Several other organochlorine pesticides were also higher in these women, and therefore it was not possible to establish a causal relationship (Gerhard, 1999).

A possible link between human exposure to HCH and breast cancer has been examined in several epidemiological studies. Most studies showed a weak - not statistically significant - correlation. A non-significant trend between beta-HCH in serum and cancer risk was observed during a 17-year follow-up of a Copenhagen cohort study (Hoyer et al., 1998). Blood levels of beta- HCH were higher in women with breast cancer (in the 31-50 age group) when compared to women without breast cancer (Mathur et al., 2002).

In another study a possible association between breast milk concentrations of various organochlorine pesticides including beta-HCH and cryptorchidism was investigated. Beta-HCH was measurable, but not statistically significantly higher in case milk than in control milk. A combined statistical analysis of the eight most abundant persistent pesticides, including beta-HCH, showed that pesticide levels in breast milk were significantly higher in boys with cryptorchidism (Damgaard et al., 2006).

**4.1.1. Risk characterisation**

In 2006 the United States Environmental Protection Agency (USEPA) performed a risk assessment that indicated potential risks from dietary exposure to the alpha and beta HCH isomers to communities in Alaska and others in the circumpolar Arctic region who depend on subsistence foods, such as caribou, seal and whale. The dietary profile (intake rates) is based on the subsistence food harvest amounts of nearly 180 communities from the Community Profile Database Version 3.11 dated 3/27/01 from the Alaska Department of Fish and Game Division of Subsistence (data from 1990 to 2001, USEPA, 2006).

USEPA estimated beta-HCH exposures for Alaskan communities in the range of 0.00043-0.0032 mg/kg bw/day for female adults, 0.0014-0.010 mg/kg bw/day for children (age 1-6) and 0.00048-0.0036 mg/kg bw/day for children (age 7-12). The risk is expressed as a percentage of a maximum acceptable dose or reference dose (RfD). A level of concern is reached if the dietary risk exceeds 100 % Rfd. The Rfd for acute oral toxicity is 0.05 mg/kg/day. The Rfd value for intermediate duration is based on a LOAEL of 0.18 mg/kg/day established in a subchronic study in rats and applying an uncertainty factor of 300 (ATSDR, 2005). On this basis USEPA established a chronic Rfd of 0.00006 mg/kg/day by assessing another uncertainty factor of 10 for chronic exposure. RIVM calculated a chronic oral Rfd of 0.0006 mg/kg/day for beta-HCH based on a NOAEL of 0.02mg/kg/day for observations of infertility in two semi-chronic oral studies on reproduction in rats and applying an uncertainty factor of 1000 (RIVM, 2001 in USEPA, 2006).

Levels of concern are reached if the dietary risk exceeds 100% Rfd. The acute dietary exposure estimates are not of concern according to USEPA (2006). USEPA's dietary risk assessment indicates that the chronic dietary exposure estimates for
beta-HCH are above the levels of concern for both low and high end dietary intake estimates. The cancer dietary risk estimates for beta-HCH are also above the level of concern for both low and high-end dietary intake estimates. According to USEPA, the risk values (% cRfD) are 620-4700 for adult males, 720-5300 for adult females, 2300-17 000 for children (1-6 years) and 800-6000 (7-12 years). The estimated cancer risk for adult males is 6.7x10^{-4} to 5.0x10^{-3} and 7.7x10^{-4} to 5.8x10^{-3} for adult females respectively. It should be noted that a general accepted cancer risk is 1x10^{-6}. Even though this risk estimation is very conservative due to the basic maximum detected levels it can be concluded that the dietary risks are of concern. Additionally, it has to be mentioned that the target organ of chronic toxicity is the liver and it can be expected that HCHs effects might be additive. It has to be considered that the RfD based on effects on fertility (RIVM, 2001 in USEPA, 2006) is remarkably lower and would be exceeded to an even greater extent.

Also, based on the study of Nair et al. (1996), levels of 0.198 mg beta-HCH/l in breast milk would lead to an intake of 0.1386 mg/l (700 ml intake) which is almost 100-fold higher than the safe intake of 0.0015 mg/child (5 kg) and only about three times lower than the LOAEL seen in animal studies (Pohl et al., 2000). Establishing the chronic RfD value of USEPA, a safe intake for a child with 5 kg would be even lower (0.0003 mg/kg) and would exceed the RfD 462-fold. Also in other regions intake levels with food and especially with breast milk are of high concern.

Anyway the unique social, cultural, spiritual and economic values of traditional foods have to be considered and strong efforts should be taken to minimize beta-HCH levels therein (CACAR, 2003).

### 2.4.2 Environment

Beta-HCH is acutely toxic to aquatic organisms. Compared to effect concentrations in algae and daphnia (IPCS, 1992), fish is the sensitive species. An LC50 of approximately 1.7 mg/l was determined in an acute test in zebra fish and neon (Oliveira-Filho and Paumgarten, 1997). IPCS (1992) reported an EC50 based on changes in fish behaviour of 47 μg/l and LC50 in guppy of 0.9 mg/l. In a prolonged toxicity study including histopathological changes, the NOEC in young guppy was 32 μg/l (Wester and Canton, 1991). Estrogenic activity of beta-HCH occurred in the form of alterations of vitellogenin production, testis atrophy, hermaphroditism in male and pituitary changes.

It seemed that beta-HCH is not very toxic to birds (IPCS, 1992) but that it may affect reproduction. In female birds with high concentrations of various OCs including beta-HCH, the body condition of the first and second chicken in the clutch was poorer (AMAP, 2004).

Monitoring data on effects in Svalbard polar bears revealed that there is a significant negative correlation between retinol and HCHs (AMAP, 2004). Retinol is essential as it is required in reproduction, embryonic and foetal development, as well as in vision, growth, differentiation and tissue maintenance.

### SYNTHESIS OF THE INFORMATION

Technical HCH, a mixture of five stable HCH-isomers, contains 5-14 % beta-HCH and was used extensively worldwide as organochlorine pesticide. Though usage of technical HCH is currently negligible, releases into the environment may still occur. Local sources include hazardous waste sites, contaminated sites, stockpiles, landfills, or dumping grounds. Though no quantitative estimates of these releases exist, the amounts of HCH-residuals in the form of by-products from lindane production were estimated to range between 1.6-1.9 to 4.8 million tons. In addition many of local sources are expected to cause environmental pollution and are not maintained or controlled appropriately.
The physico-chemical properties of beta-HCH allow on a global scale for “cold condensation”, but pathways of alpha- and beta-HCH diverge in the environment. Reasons are possibly greater physical and metabolic stability, higher water/octanol solubility, a lower Henry’s law constant and a relatively high octanol-air partition coefficient, which favours partitioning to organic phases.

According to available data beta-HCH can be considered to be persistent in the environment. Though beta-HCH is biodegradable by various microbial strains under favourable conditions degradation rates in field experiments are low indicating very slow decrease under environmental conditions. Residues of beta-HCH remained for years in treated plots in several studies. The only determined DT50 values were 100 and 184 days on cropped and uncropped soil under subtropical conditions.

Monitoring data from remote regions far from sources clearly indicate that beta-HCH has undergone long-range environmental transport. It is suggested that beta-HCH enters the Arctic by ocean currents passing through the Bering Strait after wet deposition and partitioning into the North Pacific Ocean.

Despite low levels of beta-HCH in the abiotic environment, levels in biota are relatively high. Beta-HCH has a BCF of 1 460 based on a laboratory study in fish. However, there are several field investigations in Arctic marine food webs available that suggest that beta-HCH may accumulate to high concentrations in upper trophic levels (i.e. marine mammals and birds). Thus BMFs as well as FWMFs were greater than 1. It has further been demonstrated that beta-HCH is found in breast milk of highly exposed indigenous mothers who consume a subsistence diet. Thus its high bioaccumulation potential is well documented.

Beta-HCH has been shown to be neurotoxic, hepatotoxic, to cause reproductive and immunosuppressive effects and effects on fertility and reproduction in laboratory animals.

Monitoring data on Arctic polar bears revealed a negative correlation with retinol concentrations and HCHs, which may impact a wide range of biological functions.

The International Agency for Research on Cancer (IARC) has classified beta-HCH in group 2B, possibly carcinogenic to humans. Beta-HCH may adversely affect human health in contaminated areas and as well in Arctic regions. Based on the available toxicity data of beta-HCH it can be concluded that current concentrations of beta-HCH in food and human milk in these regions are of concern. The estimated cancer risk calculated by EPA, though very conservative, seems very high (5.0x10\(^{-3}\) to 7.7x10\(^{-4}\)).

It has to be taken into consideration that the Arctic population and wildlife are also exposed against a wide range of other persistent toxic substances which may act in an additive or probably synergistic way. Nevertheless it should be emphasized that traditional foods have unique social, cultural, spiritual and economic value and therefore it is strongly recommended to avoid levels of concern.

**CONCLUDING STATEMENT**

Though most countries have banned or restricted the use of technical HCH as a pesticide, replacing it in most cases by the use of lindane, the production process gains huge amounts of HCHs residuals. These waste isomers have been a worldwide problem for years.

Beta-HCH is persistent and present in all environmental compartments; especially levels in the terrestrial as well as in the aquatic food chain give rise to concern to adversely affect human health. High exposure is expected in polluted areas as well as in the Arctic region, as a result of long-range environmental transport.

Based on the hazard profile and the above exposure scenarios, it can be concluded that beta-HCH may adversely affect wildlife and human health in contaminated regions. Based on levels found in the Arctic region, it can be also concluded that beta-HCH can lead to significant adverse human and environmental effects as a result of its long-range environmental transport.

For these reasons, global action on beta-HCH is warranted.
References


• Gupta A., Kaushik C.P., Kaushik A.: Degradation of hexachlorocyclohexane (HCH; α, β, γ and δ) by Bacillus circulans and Bacillus brevis isolated from soil contaminated with HCH. Soil Biology & Biochemistry 32 (11), 2000, pp. 1803-1805(3).


Li YF., Macdonald, RW.: Sources and pathways of selected organochlorine pesticides to the Arctic and the effect to pathway divergence on HCH trends in biota: a review. The Science of the Total Environment 342, 2005, p. 87-106.


### B.3. Chlordecone - SUMMARY

**SUMMARY**

3. Chlordecone

Draft Risk Management Evaluation May 2007  

Risk Profile UNEP/POPS/POPRC.2/17/Add2  

<table>
<thead>
<tr>
<th>Composition</th>
<th>Synthetic chlorinated organic compound also known as Kepone, GC-1189, Merex, ENT 16391, and Curlone. Chlordecone is closely related chemically to mirex, a pesticide which is already listed under the Stockholm Convention. The chemical structure of chlordecone differs from mirex in that the oxygen of the keto group in chlordecone is replaced by two chlorine atoms in mirex.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Based on the available information, Chlordecone is not anymore produced or used. Chlordecone has been used in various parts of the world for the control of a wide range of pests. In particular, Chlordecone has been used extensively in the tropics for the control of banana root borer. It has been used as a fly larvicide, as a fungicide against apple scab and powdery mildew and to control the Colorado potato beetle, rust mite on non-bearing citrus, and potato and tobacco wireworm on gladioli and other plants. Chlordecone has also been used in household products such as ant and roach traps. Chlordecone was also found to be present in technical grade mirex.</td>
</tr>
<tr>
<td>Releases</td>
<td>Given the specific pesticidal uses of Chlordecone, it can be expected that all amounts manufactured are ultimately released to the environment. The use of Chlordecone as a pesticide in Martinique and Guadeloupe until 1993, resulted in severe contamination of soil and surface water, which are being monitored at present. (Bocquene &amp; Franco, 2005, Beaugendre, 2005). Major releases of Chlordecone occurred to the air, surface waters, and soil surrounding a major American manufacturing site in Hopewell, Virginia. Releases from this plant ultimately contaminated the water, sediment, and biota of the James River, a tributary to the Chesapeake Bay (Quoted from US ATSDR, 1995).</td>
</tr>
<tr>
<td>Fate</td>
<td>Chlordecone is not expected to hydrolyse or biodegrade in aquatic environments, nor in soil. Direct photodegradation is not significant. Therefore, Chlordecone is considered to be highly persistent in the environment. Chlordecone is considered to have a high potential for bioaccumulation and biomagnification. Due to lack of monitoring data on chlordecone, the assessment of the potential for long-range transport of chlordecone was based on physico-chemical properties and application of long range transport models.</td>
</tr>
<tr>
<td>Effects</td>
<td>Chlordecone is readily absorbed into the body and accumulates following prolonged exposure. The pesticide is both acutely and chronically toxic, producing neurotoxicity, immunotoxicity, reproductive, musculoskeletal and liver toxicity at doses between 1 - 10 mg/kg bw/day in experimental animal studies. Liver cancer was induced in rats at a dose of 1 mg/kg body weight per day, and reproductive effects are seen at similar dose levels. The International</td>
</tr>
</tbody>
</table>
Agency for Research on Cancer has classified chlordecone as a possible human carcinogen (IARC group 2B). Moreover, chlordecone is very toxic to aquatic organisms, with the most sensitive group being the invertebrates.

### Exposure

The available information regarding environmental concentrations of Chlordecone is very limited and includes only areas in the vicinity of production (US) or use (Martinique). Recent monitoring data from the United States demonstrate the persistence of Chlordecone, known as Kepone in the United States. In Martinique, the widespread use of Chlordecone until 1993 has resulted in contamination of soils and surface water in most of the island (Bocquené & Franco, 2005).

### Status

Chlordecone is listed in Annex I of the Protocol to the Convention on Long-Range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants. The provisions of the Protocol oblige Parties to phase out all production and uses of Chlordecone. Chlordecone is also included in the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR Convention) as a substance of possible concern. Under the Convention on the Protection of the Marine Environment of the Baltic Sea Area (HELCOM Convention) Chlordecone is listed as selected substances for immediate priority action (Recommendation 19/5, Attachment, Appendix 3) and is scheduled for elimination (Annex I, part 2). HELCOM aims to move towards the target of the cessation of discharges, emissions and losses of hazardous substances by the year 2020. Under the Basel Convention off-specification or out-dated pesticides, without specific mention of Chlordecone, are classified as hazardous in Annex VIII. Chlordecone is currently not listed in the Rotterdam Convention on the Prior Informed Consent Procedure for certain hazardous Chemicals and Pesticides in international trade. Thailand has submitted a notification of Final Regulatory Actions for Banned or Severely Restricted Chemicals that has been verified to meet the requirements of Annex I of the Rotterdam Convention.

### Alternatives

A variety of chemical alternatives have been utilised including ethoprop, oxamyl, cyfluthrin, imidaclopidr, azadirachtin, bifenthrin, boric acid, carbaryl, capsaicin, cypermethrin, cyfluthrin, deltamethrin, diazinon, dichlorvos, esfenvalerate, imidacloprid, lamda-cyhalothrin, malathion, permethrin, piperonyl butoxide, pyrethrins, pyriproxyfen, resmethrin, s-bioallerthrin, tetramethrin, aldicarb, isophenphos, phenamiphos, cadusaphos, terbuphos. Alternatives to chlordecone also include non-chemical agroecological methods, such as preventative pest management through appropriate fertility and field sanitation practices that reduce pest pressure; the use and habitat enhancement of natural enemies; microbial preparations such as *Bacillus thuringiensis*; cultural practices such as crop rotation, intercropping, and trap cropping; barrier methods, such as screens, and bagging of fruit; use of traps such as pheremone and light traps to attract and kill insects. These and other agroecological methods are being extensively and successfully practised in many countries, eliminating the need for Chlordecone or other chemical interventions.
B.3.a. Chlordecone – DETAILED PROFILE

BASIC PROFILE

Introduction

Chlordecone is a synthetic chlorinated organic compound which has mainly been used as an agricultural insecticide, miticide and fungicide. Chlordecone has been identified as a persistent organic pollutant chemical under the Protocol on Persistent Organic Pollutants to the Convention on Long-range Transboundary Air Pollution. Under the provisions of the Protocol, Parties are obliged to phase out all production and uses of chlordecone. Chlordecone is chemically very similar to mirex, another pesticide which is already listed under the Stockholm Convention.

1. Identification of the chemical

1.1 Names and registry numbers

**CAS chemical name:**
1,1a,3a,4,5,5a,5b,6-decachloro-octahydro-1,3,4-metheno-2H-cyclobuta-[cd]-pentalen-2-one

**Synonyms:**
Decachloropentacyclo-[5,2,1,0^2,6,0^3,9,0^5,6]-decan-4-one,
Decachlorooctahydro-1,3,4-metheno-2H,5H-cyclobuta-[cd]-pentalen-2-one
Decachloroketone

**Trade names:**
GC 1189, Kepone, Merex, ENT 16391, Curlone

**CAS registry number:**
143-50-0

1.2 Structure

![Structural formula](webbook.nist.gov/chemistry)

Molecular formula: \( \text{C}_{10}\text{Cl}_{10}\text{O} \)

Molecular weight: 490.6

The chemical structure of chlordecone differs from mirex in that the oxygen of the keto group in chlordecone is replaced by two chlorine atoms in mirex.
2. Persistence

Chlordecone is highly persistent in the environment. According to the information given in the review reports*, the estimated half-life of chlordecone in soil is between one and two years. It is not expected to hydrolyse or biodegrade in the environment. Direct photodegradation is not significant. The primary process for the degradation of chlordecone in soil or sediments is anaerobic biodegradation.

- The half-life in soils exceeds the criterion value of six months. It is reported to be from 1 to 2 years (Refs. 1 and 2). By analogy with mirex, one report suggests that the half-life could be three years or longer (Ref. 3);
- A new scientific paper indicates that, in the James River (Virginia, United States of America), downstream of a facility that produced Kepone (chlordecone), the chemical is still detected in fish samples more than 20 years after the production had been phased out (Ref. 4);

3. Bioaccumulation

Based on the lipophilic nature of this compound (high octanol-water partition coefficient (logKow)), chlordecone has a tendency to both bioaccumulate and biomagnify in aquatic food chains. Bioconcentration factors of over 60,000 have been measured in Atlantic silverside, an estuarine fish species.

- The reported bioconcentration factors are summarized below (Ref. 5):
  - Unicellular algae: 230–800
  - Aquatic invertebrates: 5,127–11,425
  - Fish: 1,800–16,600
- There is additional information supporting the potential for bioaccumulation and biomagnification, including an excretion half-life in mammals of several months and the detection of high levels of the chemical in fish and birds (Refs. 3 and 5). This bioaccumulation is a consequence of the lipophilic nature of the chemical, for which the log Kow value is 4.50–6.00 (Refs. 2, 3 and 5);

4. Potential for long-range environmental transport

The vapour pressure of chlordecone is below $3 \times 10^{-5}$ mm Hg at 25°C. Chlordecone is not expected to be subject to direct photodegradation in the atmosphere. Its estimated half-life time in air is up to 50 years. Atmospheric transport of chlordecone particles was reported in the United States of America during production years.

- The vapour pressure of chlordecone ($2.25 \times 10^{-7}$ mm Hg at 25°C) (Ref. 6) is such that long-range transport in the atmosphere can be anticipated, and dissemination in particulate form has been observed. Modelling studies suggest life-times in air substantially in excess of the criterion value of two days (Ref. 2);

5. Adverse effects

Chlordecone is moderately toxic to laboratory mammals with single exposures. Acute toxic symptoms in all species tested included severe tremor. It can cause skin irritation. In long-term studies, lower doses caused tremor and other neurological symptoms, and also liver hypertrophy.
Chlordecone interferes with reproduction and is fetotoxic in experimental animals. It is not generally active in short-term tests for genetic activity. Chlordecone is carcinogenic in both sexes of mice and rats, producing hepatocellular carcinomas. The International Agency for Research on Cancer concludes that there is sufficient evidence that chlordecone is carcinogenic in mice and rats. In the absence of adequate data in humans, it is reasonable to regard chlordecone as if it presented a carcinogenic risk to humans.

Chlordecone is very toxic to aquatic organisms. The few data available on terrestrial ecosystems indicate low acute toxicity but some long-term effects on vertebrate reproduction.

- Workers exposed in their work place showed clinical signs of chlordecone poisoning (Ref. 3);
- There are extensive data showing potential for adverse effects on humans and ecosystems, including carcinogenicity and reproductive effects and very high toxicity for aquatic organisms (fish non-observed-effect concentration < 1 microgram per litre) (Ref. 5);

References
1. Regional reports of the regionally based assessments of persistent toxic substances. UNEP. 2002.

SPECIFIC REASONS FOR CONCERN

Chlordecone is a synthetic chlorinated organic compound, chemically very similar to mirex, and has mainly been used as an agricultural insecticide, miticide and fungicide. It was first produced in 1951 and introduced commercially in the United States in 1958 (trade names Kepone® and GC-1189). It was available in the United States until 1976. In France, chlordecone was marketed with a trade name Curlone from 1981 to 1993.

Historically, chlordecone has been used in various parts of the world for the control of a wide range of pests. It has been used extensively in banana cultivation against banana root borer, as a fly larvicide, as a fungicide against apple scab and powdery mildew and to control the Colorado potato beetle, rust mite on non-bearing citrus, and potato and tobacco wireworm on gladioli and other plants. Given the specific pesticidal uses of chlordecone, it can be expected that all amounts manufactured are ultimately released to the environment.

Chlordecone is not expected to hydrolyse or biodegrade in aquatic environments, nor in soil. Direct photodegradation is not significant. Therefore, Chlordecone is considered to be highly persistent in the environment. With BCF-values in algae up to 6,000, in invertebrates up to 21,600 and in fish up to 60,200 and documented examples of biomagnification, chlordecone is considered to have a high potential for bioaccumulation and biomagnification.

The available data are not conclusive when it comes to long-range atmospheric transport of chlordecone in gaseous form. However, atmospheric transport of particle-bound substances and transport of sediment particles in ocean currents as well as biotic transport could also contribute to long-range environmental transport of chlordecone. Due to lack of monitoring data on chlordecone, the assessment of the potential for long-range transport of chlordecone was based on physico-chemical properties and application of long range transport models.
Chlordecone is readily absorbed into the body and accumulates following prolonged exposure. The pesticide is both acutely and chronically toxic, producing neurotoxicity, immunotoxicity, reproductive, musculoskeletal and liver toxicity at doses between 1 - 10 mg/kg bw/day in experimental animal studies. Liver cancer was induced in rats at a dose of 1 mg/kg body weight per day, and reproductive effects are seen at similar dose levels. The International Agency for Research on Cancer has classified chlordecone as a possible human carcinogen (IARC group 2B). Moreover, chlordecone is very toxic to aquatic organisms, with the most sensitive group being the invertebrates.

Production and use of chlordecone may have ceased over the last decades in developed countries but it is assumed that it is still produced or used as an agricultural pesticide in some developing countries. As chlordecone can move in the atmosphere far from its sources, single countries or groups of countries alone cannot abate the pollution it causes. Regional action has already been considered necessary and chlordecone is totally banned under the Convention on Long-range Transboundary Air Pollution Protocol on Persistent Organic Pollutants. Due to its harmful properties and the risks posed by its probable production and use, global action is warranted under the Stockholm Convention to eliminate this pollution."
RISK PROFILE

OVERVIEW

According to the Risk Profile, Chlordecone was first produced in 1951 and introduced commercially in the United States in 1958 under the trade names Kepone® and GC-1189 (Epstein, 1978; Huff and Gerstner 1978 quoted from UNEP 2006). Chlordecone was in use in the USA until 1976 (IARC, 1979). Chlordecone was also found to be present in technical grade mirex (EPA 1978b quoted from UNEP 2006; IARC 1979a quoted from UNEP 2006) (Quoted from US ATSDR, 1995).

Between 1951 and 1975, approximately 3.6 million pounds (1.6 million kg) of Chlordecone were produced in the United States (Epstein, 1978). (Quoted from US ATSDR, 1995) Chlordecone production was discontinued in the USA in 1976.

Diluted technical grade Chlordecone (80% active ingredient) was exported from the USA to Europe, particularly Germany, in great quantities from 1951 to 1975 (Epstein, 1978) where it was converted to Kelevan which is a derivative of Chlordecone and used for the same purposes. In the environment, it oxidizes to Chlordecone and could therefore also be considered with Chlordecone for listing in the Stockholm Convention. Approximately 90-99% of the total volume of Chlordecone produced during this time was exported to Europe, Asia, Latin America, and Africa. (DHHS 1985; EPA 1978b quoted from UNEP 2006) (Modified from US ATSDR, 1995). There is no information, indicating that Kelevan is being produced or used at present.

Chlordecone was marketed in France as a formulation, Curlone, by De Laguarique from 1981 to 1993. The formulation was used in Martinique and Guadeloupe following hurricane Allen in 1979 and David in 1980 which led to considerable pest infestations. Chlordecone for this formulation was synthesised in Brazil. The authorisation for Curlone was withdrawn by the French Ministry of Agriculture in 1990. Use was continued until September, 1993. (Beaugendre, 2005) In Canada, no product containing Chlordecone has been registered as a pest control product since 2000.

Chlordecone has been used extensively in the tropics for the control of banana root borer (Anonymous, 1978a quoted from UNEP 2006; Langford, 1978 quoted from UNEP 2006). It is regarded as an effective insecticide against leaf-cutting insects. Historically, Chlordecone has been used in various parts of the world for the control of a wide range of pests. It can be used as a fly larvicide, as a fungicide against apple scab and powdery mildew (Information Canada, 1973 quoted from UNEP 2006), and to control the Colorado potato beetle (Motl, 1977), rust mite on non-bearing citrus, and potato and tobacco wireworm on gladioli and other plants (Suta, 1978). Chlordecone has also been used in household products such as ant and roach traps at concentrations of approximately 0.125% (IARC 1979a quoted from UNEP 2006).

PHYSICAL AND CHEMICAL PROPERTIES

The physical and chemical properties of Chlordecone are listed in Table 1.1. It demonstrates that the variation is high between data sources for physical properties like vapour pressure and water solubility. This is confirmed by the fact that the Henry’s Law Constant varies by one order of magnitude, depending on the type of data used for the calculation. The source of used data are generally considered to be reliable: the data quality have been assessed in the (inter)national consensus documents (IARC, IPCS HSG, IPCS EHC and US ATSDR) and the quality of the data published by Hansch et. al. and Howard has been evaluated (Pedersen et. al., 1995).

<table>
<thead>
<tr>
<th>Property</th>
<th>Unit</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td>C₁₀Cl₁₀O</td>
<td>490.6</td>
<td>IARC, 1979¹</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>g/mole</td>
<td></td>
<td>Kilzer, I et. al., 1979²</td>
</tr>
<tr>
<td>Appearance at normal temperature and pressure</td>
<td>Pa</td>
<td>Tan-white crystalline solid</td>
<td>IARC, 1979¹</td>
</tr>
<tr>
<td>Vapour Pressure</td>
<td>Pa</td>
<td>3.0x10⁻⁵ (25 °C)</td>
<td>Kilzer, I et. al., 1979²</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt; 4.0x10⁻⁵ (25 °C)</td>
<td></td>
</tr>
</tbody>
</table>

85
Water solubility | mg/L | 0.35-1.0x
---|---|---
1-2 | HSG 41, IPCS, 1990
2.7 (25 °C) | EHC 43, IPCS, 1990
3.0 | Kilzer, l et. al., 1979

Melting point | °C | 350; (decomposes)
Boiling point | °C | No data

Log K_{OW} | | 4.50
| | Howard, 1991
| | Hansch et al., 1995

Log K_{aw} | | -6.69
| | Scheringer et al., 2006

Log K_{oc} | | 3.38-3.415
| | Howard, 1991
| | Calculated

Henry's Law Constant | Pa m^3/mol | 2.53x10^-3 (20 °C)
| | 2.0x10^-2
| | Calculated

Atmospheric OH Rate | cm^3/molecule-sec | \approx 0 (25 °C)

Data sources
This Risk Profile is mainly based on information from the following review reports:

- Report prepared for the Assemblée Nationale describing the history of production and use of Chlordecone in Martinique and Guadeloupe (Beaugendre, 2005).
- Arctic Monitoring and Assessment Programme (http://www.amap.no/)

SOURCES

Production
Chlordecone has been produced by reacting hexachlorocyclopentadiene and sulfur trioxide under heat and pressure in the presence of antimony pentachloride as a catalyst. The reaction product is hydrolyzed with aqueous alkali and neutralized with acid; Chlordecone is recovered via centrifugation or filtration and hot air drying (Epstein 1978) (Quoted from US ATSDR, 1995).

Chlordecone was first produced in 1951, patented in 1952, and introduced commercially in the United States by Allied Chemical in 1958 under the trade names Kepone® and GC-1189 (Epstein 1978; Huff and Gerstner 1978). The technical grade of Chlordecone, which typically contained 94.5%
Chlordecone, was available in the United States until 1976 (IARC 1979). Chlordecone was also found to be present in technical grade mirex at concentrations up to 2.58 mg/kg and in mirex bait formulations at concentrations up to 0.25 mg/kg (EPA 1978b; IARC 1979a) (Quoted from US ATSDR, 1995).

**Trade and stockpiles**
Between 1951 and 1975, approximately 3.6 million pounds (1.6 million kg) of Chlordecone were produced in the United States (Epstein 1978). (Quoted from US ATSDR, 1995) Chlordecone production was discontinued in the USA in 1976. However, a year later it was reported that a French company was considering the establishment of production facilities in France (Anonymous, 1978b), but no further information on this proposal is available. (Modified from EHC 43, (IPCS, 1984)). No current data are available regarding import volumes of Chlordecone. By 1976, technical Chlordecone was not exported from the United States and the compound was no longer produced there. Diluted technical grade Chlordecone (80% active ingredient) was exported to Europe, particularly Germany, in great quantities from 1951 to 1975 by the Allied Chemical Company (Epstein 1978) where the diluted technical product was converted to an adduct, Kelevan. Kelevan is a derivative of Chlordecone and used for the same purposes. In the environment, it oxidizes to Chlordecone and could therefore also be considered with Chlordecone for listing in the Stockholm Convention. Approximately 90-99% of the total volume of Chlordecone produced during this time was exported to Europe, Asia, Latin America, and Africa. (DHHS 1985; EPA 1978b) (Modified from US ATSDR, 1995) There is no information, indicating that Kelevan is being produced or used at present.

Chlordecone was marketed in France as a formulation, Curlone, by De Laguarique from 1981 to 1993. The formulation was used in Martinique and Guadeloupe following hurricane Allen in 1979 and David in 1980 which led to considerable pest infestations. Chlordecone for this formulation was synthesised in Brazil. The authorisation for Curlone was withdrawn by the French Ministry of Agriculture in 1990. Use was continued until September, 1993. (Beaugendre, 2005) In Canada, no product containing Chlordecone has been registered as a pest control product since 2000.

**Uses**
Chlordecone has been used extensively in the tropics for the control of banana root borer (Anonymous, 1978a; Langford, 1978). This was its only registered food use. It is regarded as an effective insecticide against leaf-cutting insects, but less effective against sucking insects (Information Canada, 1973). Historically, Chlordecone has been used in various parts of the world for the control of a wide range of pests. It can be used as a fly larvicide, as a fungicide against apple scab and powdery mildew (Information Canada, 1973), and to control the Colorado potato beetle (Motl, 1977), rust mite on non-bearing citrus, and potato and tobacco wireworm on gladioli and other plants (Suta, 1978).

Chlordecone has also been used in household products such as ant and roach traps at concentrations of approximately 0.125% (IARC 1979a). The concentration used in ant and roach bait was approximately 25%. (Epstein 1978) (Modified from EHC 43 (IPCS, 1984) and US ATSDR, 1995).

**RELEASES TO THE ENVIRONMENT**
Given the specific pesticidal uses of Chlordecone, it can be expected that all amounts manufactured are ultimately released to the environment. The use of Chlordecone as a pesticide in Martinique and Guadeloupe until 1993, resulted in severe contamination of soil and surface water, which are being monitored at present. (Bocquene & Franco, 2005, Beaugendre, 2005).

Major releases of Chlordecone occurred to the air, surface waters, and soil surrounding a major American manufacturing site in Hopewell, Virginia. Releases from this plant ultimately contaminated the water, sediment, and biota of the James River, a tributary to the Chesapeake Bay (Quoted from US ATSDR, 1995).

**ENVIRONMENTAL FATE**
The partitioning of Chlordecone in the environment will be governed by its high log \(K_{ow}\) (5.41 or 4.50) and relatively low water solubility (1-3.0 mg/L) resulting in sorption to particulate matter (dust, soil and sediment) and organic material (living organisms).
The combination of these properties and the vapour pressure (3.0-4.0\times10^5 \text{ Pa}) of Chlordecone, results in a relatively low potential for volatilisation as the Henry’s Law Constant is between 2.0\times10^{-2} and 5.45\times10^{-3} \text{ Pa m}^3/\text{mole (25 °C)}, depending on the type of data used for the calculation (Table 1.1.). In the EHC 43 (IPCS, 1984), the volatilisation of Chlordecone is evaluated based on laboratory and field observations that indicate that Chlordecone does not volatilise to any significant extent (Dawson, 1978). However, the release of copious quantities of Chlordecone dust from production facilities has represented a major source of environmental and human contamination. Airborne Chlordecone has been known to spread 60 miles from a point source (Feldmann, 1976), and the potential exists for further dispersion of fine particles (Lewis & Lee, 1976 (Abbreviated from EHC 43 (IPCS, 1984).))

The US ATSDR (1995), concluded that Chlordecone released to the environment partitions to soil and sediment. Small amounts may remain dissolved in water and Chlordecone released to the atmosphere is eventually deposited on soil or surface waters.

**Persistence**

In the EHC 43 (IPCS, 1984), early reports that did not include any evidence of Chlordecone degradation in the natural environment (Dawson, 1978; Geer, 1978) were quoted as well as a more recent study, in which microbial action had been shown to transform Chlordecone into monohydro- and possibly dihydrochlordecone (Orndorff & Colwell, 1980a).

EHC 43 (IPCS, 1984), concluded that Chlordecone is an extremely stable compound and is not expected to degrade in the environment to any significant extent. However, there have been reports of trace amounts of monohydrochlordecone being found (Carver et al., 1978, Orndorff & Colwell, 1980b), but the mechanism of its formation is not clear. Solar irradiation of Chlordecone in the presence of ethylenediamine results in 78% degradation after 10 days (Dawson, 1978) quoted from EHC 43 (IPCS, 1984). However, ethylenediamine is not usually present in the atmosphere, so at the time, there was no information available regarding the photolytic stability of Chlordecone under environmental conditions.

The more recent review (US ATSDR, 1995), concludes that Chlordecone is not expected to be subject to direct photodegradation in the atmosphere. Furthermore, it is concluded that Chlordecone is resistant to aerobic degradation, although some anaerobic biodegradation does occur and that Chlordecone is very persistent in the environment. Chlordecone will strongly bind to organic matter in water, sediment, and soil. When bound to organic-rich soil, Chlordecone is highly immobile; however, when adsorbed to particulate matter in surface water, Chlordecone can be transported great distances before partitioning out to sediment. The primary process for the degradation of Chlordecone in soil or sediments is anaerobic biodegradation (Abbreviated from US ATSDR, 1995).

Information regarding the persistence of Chlordecone dating after 1995 is scarce, but the use of Chlordecone until 1993 in the Caribbean island of Martinique has resulted in severe contamination and monitoring studies have been initiated. Bocquene & Franco (2005) reported concentrations in samples from 2002 in water (particulate matter) and sediment in rivers of up to 57 \mu g/kg and 44 \mu g/kg, respectively. They quoted other investigations for reporting concentrations in river water, sampled in 2000-2001 in the range 1.20 - 2.13 \mu g/L.

Even though Chlordecone was prohibited from main land France, an exemption was granted that allowed the use of it in the French West Indies until September, 1993. A recent study showed that it is still detected in different ecosystems of Martinique (Coat, S. et. al., 2006). Stocks of Chlordecone may have been used in Martinique after 1993, but it is expected that the use ceased several years ago. However, residues are still measurable in both river water and sediment, where the prevailing anaerobic conditions in the latter allow for the only known biotic degradation of Chlordecone. This is all the more remarkable as the climate in this area is optimal not only for crops and pests but also for biodegradation.

**Conclusion**

Chlordecone is not expected to hydrolyse or biodegrade in aerobic aquatic environments or in soil; however, there is some evidence of degradation under anaerobic condition. Direct photodegradation is not significant. Based on all available data Chlordecone is considered to be highly persistent in the environment.
Bioaccumulation
Because of the lipophilic nature of this compound (high octanol-water partition coefficient (log $K_{ow}$ 4.50-5.41), Chlordecone has a potential for both bioaccumulation and, with little or no metabolic depuration, also biomagnification in aquatic food chains.

Table 2.1 summarises bioconcentration factors (BCF) selected from the US EPA database Ecotox (US EPA, 2006). The results included are based on measured concentrations and, for organisms different from algae, derived from tests based on flow through exposure. Thereby, the results should reflect the bioconcentration obtained under well defined, constant exposure concentrations. For fish, the results of a series of tests of four days duration were not included, because it is not considered to be likely that equilibrium had been reached¹. Two additional studies from EHC 43 (IPCS, 1984) are also included.

Table 2.1 BCF values for Chlordecone.

<table>
<thead>
<tr>
<th>Species</th>
<th>Test Duration</th>
<th>Exposure Concentration µg/L</th>
<th>BCF</th>
<th>Reference¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Green algae (Chlorococcum sp., Dunaliella tertiolecta)</td>
<td>24 h</td>
<td>100</td>
<td>230-800</td>
<td>Walsh et. al., 1977</td>
</tr>
<tr>
<td>Green alga (Chlorococcum sp.)</td>
<td>48 h</td>
<td>40</td>
<td>6,000</td>
<td>Bahner et. al., 1977</td>
</tr>
<tr>
<td>Diatoms (Thalassiosira guillardi, Nitzschia sp.)</td>
<td>24 h</td>
<td>100</td>
<td>410-520</td>
<td>Walsh et. al., 1977</td>
</tr>
<tr>
<td>Crustacean (Callinectes sapidus)</td>
<td>96 h</td>
<td>110-210</td>
<td>6.2-10.4</td>
<td>Schimmel, 1977</td>
</tr>
<tr>
<td>Crustacean (Palaemonetes pugio)</td>
<td>96 h</td>
<td>12-121</td>
<td>425-933</td>
<td>Schimmel, 1977</td>
</tr>
<tr>
<td>Crustacean (Palaemonetes pugio, Americamysis bahia)</td>
<td>21-28 d</td>
<td>0.023-0.4</td>
<td>5,127-13,473</td>
<td>Bahner et. al., 1977</td>
</tr>
<tr>
<td>Crustacean (Palaemonetes pugio)</td>
<td>16 d</td>
<td>0.041</td>
<td>12,094</td>
<td>Fisher &amp; Clark, 1990</td>
</tr>
<tr>
<td>Oyster (Crassostrea virginica)</td>
<td>19-21 d</td>
<td>0.03-0.39</td>
<td>9,278-9,354</td>
<td>Bahner et. al., 1977</td>
</tr>
<tr>
<td>Midge (Chironomus tentans)</td>
<td>14 d</td>
<td>11.8-169.2</td>
<td>21,600</td>
<td>Adams et. al., 1985</td>
</tr>
<tr>
<td>Fish (Brevoortia tyrannus)</td>
<td>1-18 d</td>
<td>0.14-1.55</td>
<td>2,300-9,750</td>
<td>Roberts &amp; Fisher, 1985</td>
</tr>
<tr>
<td>Fish (Menidia menidia)</td>
<td>1-28 d</td>
<td>0.08-0.8</td>
<td>21,700-60,200</td>
<td>Roberts &amp; Fisher, 1985</td>
</tr>
<tr>
<td>Fish (Cyprinodon variegatus)</td>
<td>28 d</td>
<td>&lt; 0.02-1.9</td>
<td>3,100-7,115</td>
<td>Bahner et. al., 1977; Hansen et. al., 1977</td>
</tr>
<tr>
<td>Fish (Leiostomus xanthurus)</td>
<td>30 d</td>
<td>0.029-0.4</td>
<td>2,340-3,217</td>
<td>Bahner et. al., 1977</td>
</tr>
<tr>
<td>Fish (Pimephales promelas)</td>
<td>56 d</td>
<td>0.004</td>
<td>16,600</td>
<td>Huckins et. al., 1982²</td>
</tr>
<tr>
<td>Fish (Cyprinodon variegatus)</td>
<td>Life cycle</td>
<td>0.041</td>
<td>1,800-3,900</td>
<td>Goodman et. al., 1982²</td>
</tr>
</tbody>
</table>

¹: All quoted from the Ecotox database (US EPA, 2006), except for two quoted from EHC 43 (IPCS, 1984)

The information on bioaccumulation from food is limited, but the EHC 43 (IPCS, 1984) report includes two relevant studies; one on food exposure and the other on an estuarine food chain. When chlordecone was fed to juvenile spot for 28 days, the body burden of chlordecone increased additively and equilibrium was not attained (Stehlik & Merriner, 1983). The estuarine food chain study (Bahner et al., 1977) was composed of green algae, oysters, mysids, grass shrimps, sheepshad minnows and spot. The transfer from algae to oysters was very low; but a clear transfer from shrimp to mysids and from mysids to spot, indicated that much of the chlordecone was being transferred through the

¹ In OECD Test Guideline 305, the prescribed duration of the exposure phase is 28 days.
trophic levels. Clearance was slow in shrimp and fish, with tissue levels of chlordecone decreasing by 30-50% in 24-28 days.

US ATSDR (1995), described the bioaccumulation of chlordecone together with that of mirex, stating that they are both highly lipophilic and therefore, have a high bioconcentration potential. They bioaccumulate in aquatic food chains with virtually no degradation of the compounds by exposed organisms (de la Cruz and Naqui, 1973; Epstein, 1978; Huckins et al., 1982; Huggett and Bender, 1980; Kenaga, 1980; Lunsford et al., 1987; Naqvi and de la Cruz, 1973; Nichols, 1990; Oliver and Niimi, 1985 and 1988; Roberts and Fisher, 1985)\(^2\).

Only limited information is available on uptake and bioaccumulation of chlordecone in terrestrial food chains (Naqvi and de la Cruz, 1973), and little uptake of chlordecone by plants was observed (Topp et al., 1986).

**Conclusion**

With BCF-values of up to 6,000 in algae, of up to 21,600 in invertebrates and of up to 60,200 in fish, and with documented examples of biomagnification, chlordecone is considered to have a high potential for bioaccumulation and biomagnification.

**Potential for Long-Range Environmental Transport**

The potential for long-range environmental transport can be documented through monitoring data from remote regions (e.g. the Arctic) and/or through physical-chemical characteristics of the molecule, which are promoting such transport. The most well known mechanism of long-range transport is atmospheric transport of substances in the vapour phase. However, atmospheric transport of particle-bound substances and transport of sediment particles in ocean currents as well as biotic transport could also contribute (e.g. AMAP 2004).

One prerequisite for long-range atmospheric transport is persistence to degradation, and Chlordecone is considered to be highly persistent in the environment (see Section 2.2.1). Chlordecone does not volatilise to any significant extent (see section 2.2). The partitioning of Chlordecone in the environment will be governed by its high log \(K_{ow}\) (5.41 or 4.50) and relatively low water solubility (1-3.0 mg/L) resulting in sorption to particulate matter (dust, soil and sediment) and organic materials and living organisms. Therefore, the long range transport is expected to take place through these pathways.

The US ATSDR (1995), states that atmospheric transport of dust containing Chlordecone particles was reported during production years based on results from high volume air sample filters from Hopewell: At approximately 200 yards from the Chlordecon production plant, the contents ranged from 3.0-55 micrograms/m\(^3\), depending on weather conditions and date of collection. At more distant sites in May 1975, levels ranged from 1.4-21 ng/m\(^3\). Specifically, in South Richmond, 15.6 miles north west from Hopewell, the level was 1.41 ng/m\(^3\). At Byrd airport, 14.12 miles north of Hopewell, the level was 1.93 ng/m\(^3\). In Petersburg, 8.19 miles south west from Hopewell, the level was 20.7 ng/m\(^3\). (Epstein, 1978). They conclude further, that airborne Chlordecone has been known to spread 60 miles from a point source (Feldmann, 1976), and that the potential exists for further dispersion of fine particles (Lewis & Lee, 1976) (US ATSDR, 1995).

Transport in aquatic environments is illustrated by results of measurements in clams and oysters from the James River at sampling locations from 8-64 miles from Hopewell, Virginia that contained 0.2-0.8 mg/kg of Chlordecone (Epstein, 1978).

However, no records are available regarding concentrations of Chlordecone in areas at long distances from sites of production or use. Therefore, the assessment of the potential for long-range transport of Chlordecone must be based on physical properties. For this - apart from persistence - the vapour pressure and the Henry’s Law Constant are considered to be the most relevant properties. For a comprehensive evaluation of the potential for long-range atmospheric transport, knowledge of the vapour pressure at high as well as at low temperatures (e.g. 25 °C and 0 °C) is required. This information is, however, available for only a few substances (AMAP, 2004), so the vapour pressure at 25 °C is used as a measure of the volatility of the substance.

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\(^2\) These references describe both Mirex and Chlordecone.
As a rule of thumb, substances with vapour pressures $>1.33 \times 10^{-2}$ Pa will be entirely in the vapour phase and substances with vapour pressures $<1.0 \times 10^{-4}$ Pa will be particulate (US ATSDR, 2004).

A way of evaluating the characteristics and effects of a substance for which not enough information exists is to compare it with better known substances of similar characteristics. This approach (known as “the benchmark approach”) was proposed by Scheringer (1997) and Beyer et al., (2000), has been recently used in some recent studies concerning persistence and environmental transport of pollutants (see, e.g., Vulykh et al., 2006, and Klasmeier et al., 2006). As a measure of values of properties that would qualify for long-range atmospheric transport, the currently listed POPs are used. However, information regarding physical-chemical properties for chemicals often varies widely between sources and the quality of data cannot be compared without specific review of the individual studies. This is demonstrated by the available data on the physical-chemical properties of Chlordecone presented in Table 1.1. The two values for the vapour pressure are rather uniform (0.3 and $0.4 \times 10^5$ Pa) but the water solubility found in literature varies by an order of magnitude (0.35–3.0 and the lowest value is considered to be unreliable.\(^3\)

The comparison of Chlordecone with already listed POPs is presented in Table 2.2. As a starting point for this comparison, the highest and lowest values for Chlordecone (Table 1.1) were used. For already listed POPs, information was sought on the UNEP-POPs homepage. Among the currently listed POPs, most of the relevant properties were available for aldrin, chlordane, dieldrin, DDT, hexachlorobenzene, mirex, toxaphene, endrin and heptachlor. Missing information (water solubility of mirex) was sought in US ATSDR (1995) and AMAP (2004). The US ATSDR (1995), quotes values of 0.2 and 0.6 mg/L, while the AMAP (2004), quotes Mackay for very low water solubility: $6.5 \times 10^{-5}$ mg/L. In order to avoid introduction of what seems to be an outlier in the comparison, the value for water solubility of mirex from US ATSDR (1995) was used.

The water solubility and vapour pressure as well as Henry’s Law Constants calculated from these values of the currently listed POPs are summarised in Table 2.2 together with information on Chlordecone from Table 1.1.

Table 2.2 Water solubility (WS), vapour pressure (VP) and (calculated) Henry’s Law Constant (HLC) (at 25 °C) for Chlordecone and currently listed POPs.

<table>
<thead>
<tr>
<th>Substance</th>
<th>WS mg/L</th>
<th>VP Pa</th>
<th>HLC Pa m(^3)/mol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlordecone-min</td>
<td>1.0</td>
<td>0.00003</td>
<td>0.0049(^1)</td>
</tr>
<tr>
<td>Chlordecone-max</td>
<td>3.0</td>
<td>0.00004</td>
<td>0.02(^2)</td>
</tr>
<tr>
<td>POP-min</td>
<td>0.0012 (DDT)</td>
<td>0.000025 (DDT)</td>
<td>0.04 (endrin)</td>
</tr>
<tr>
<td>POP-max</td>
<td>3.0 (toxaphene)</td>
<td>27 (toxaphene)</td>
<td>3726 (toxaphene)</td>
</tr>
<tr>
<td>POP-2(^{nd}) max</td>
<td>0.5 (dieldrin)</td>
<td>0.04 (heptachlor)</td>
<td>267 (heptachlor)</td>
</tr>
</tbody>
</table>

1: Calculated from maximum water solubility and minimum vapour pressure
2: Calculated from minimum reliable water solubility and maximum vapour pressure

Table 2.2 shows that the water solubility of Chlordecone is at the level of the most water soluble among the currently listed POPs (toxaphene and dieldrin), while the vapour pressure is comparable to that of DDT. The highest of the two Henry’s Law Constants that were calculated for Chlordecone is of the same order of magnitude as that of endrin. It should be noted that in presenting the data in Table 2.2 it is not inferred that a chemical (in this case Chlordecone) is considered to meet the long range environmental transport criterion just because it fits within the range of values of currently listed POPs.

Further to this, it should be mentioned that the latest AMAP report on POPs (AMAP, 2004) describes the possibilities of particle borne transport for substances, which have Henry’s Law Constants (HLC) close to that of Chlordecone (HLC = 0.0049 or 0.056). Based on HLC-values from AMAP (2004), it is concluded that semi-volatile compounds such as lindane ($\gamma$-HCH) (HLC = 0.000149) and chlordane (HLC = 0.342) are distributed between airborne particles and the gaseous phase, depending on the

\(^3\) Availability of high quality data regarding physical-chemical properties could support more firm conclusions.
temperature. These can be washed out via precipitation and temporarily deposited in seawater or soil and can absorb to water, plant and soil surfaces from the gaseous phase. During favourable warm weather conditions, these compounds evaporate again into the atmosphere and undergo further atmospheric transport. This remobilization is also called the ‘grasshopper effect’. The role of stormy weather situations in remobilization of semivolatile compounds into the atmosphere is obvious but still scarcely investigated (AMAP, 2004).

Besides, certain physical-chemical properties of Chlordecone, such as the partition coefficients log $K_{\text{ow}}$ (octanol-water partition coefficient) and log $K_{\text{aw}}$ (air-water partition coefficient), are similar to those of some toxaphene components which, added to its persistence in air and water, would mean that coupled long range transport in atmosphere and oceans may take place (i.e. the substance is exchanging between atmospheric gas phase and oceanic dissolved phase and can be transported in either phase). (Wania, F. 2006, personal communication). Chlordecone has a very low Henry’s law constant and a high mass fraction is found in water, and therefore it can be inferred that transport with ocean currents contributes to the long-range transport of Chlordecone.

In a recent modeling study, Scheringer et. al. (2006), investigated the persistence and long range transport potential of these potential POPs, including chlordecone and hexabromobiphenyl, using an OECD screening tool which based the evaluation of overall environmental persistence and transport potential on the results of several of the currently available multimedia environmental fate models (see also Klasmeier et. al., 2006, and Fenner et. al., 2005 for a more detailed explanation). They concluded that the four POP candidates have persistence and long range transport potential properties similar to those of several known POPs in this evaluation of the air emission scenario.

Furthermore, they included the uncertainty regarding the data quality in an uncertainty analysis, which indicated that the result is valid although there are considerable uncertainties in the chemical properties of the four POP candidates. It should be noted that environmental fate modeling results strongly depend on the assumptions made, specifically when essential data such as environmental half-lives are not known. In addition, results for substances like Chlordecone, which are strongly bound to particles and are of very low volatility, are highly dependent on the medium to which they are emitted, i.e., to air, to water, or to soil. The emission to air scenario always yields the highest transfer efficiency, and that value is displayed in the Scheringer et. al. (2006) plots. Transfer efficiency will likely differ by several orders of magnitude when evaluated under soil and water emission scenarios.

### Conclusion

In summary, the above discussion shows that the available data on Chlordecone are not conclusive when it comes to long-range atmospheric transport in gaseous form. However, atmospheric transport of particle-bound substances and transport of sediment particles in ocean currents, as well as biotic transport, could also contribute to long-range environmental transport of Chlordecone. Coupled atmosphere-ocean transport also seems quite possible.

Due to a lack of monitoring data on Chlordecone the assessment of the potential for long-range transport of Chlordecone must be based on physico-chemical properties and modelling data. The modelling study of Scheringer et. al., 2006, shows clearly that long range environmental transport is possible (and possibly more than actually estimated), even considering the uncertainties surrounding the physico-chemical properties.

In accordance with paragraph 7 (a) of Article 8 of the Convention, and taking into account that a lack of full scientific certainty should not prevent a proposal from proceeding, Chlordecone is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects such that global action is warranted.

## EXPOSURE

### Environmental concentrations

The available information regarding environmental concentrations of Chlordecone is very limited and includes only areas in the vicinity of production (US) or use (Martinique).

The US ATSDR (1995), illustrates the presence of Chlordecone in the environment following production of the substance. In 1977, 12 years after production of Chlordecone began and 2 years
after the production ceased, average concentrations of Chlordecone in estuarine water (dissolved) were <10 ng/L (ppt) (Nichols 1990). In October 1981, 6 years after production ceased, Chlordecone water concentrations ranged from not detectable to 0.02 µg/L (ppb) (Lunsford et. al., 1987). Groundwater monitoring data are lacking, but because Chlordecone binds tightly to organic matter in soil, leaching into groundwater is not expected to occur extensively (Abbreviated from US ATSDR, 1995).

Recent monitoring data from the United States demonstrate the persistence of Chlordecone, known as Kepone in the United States. The substance is included in the U.S. EPA National Lake Fish Tissue Study to estimate the national distribution of selected residues in fish tissue from lakes and reservoirs in the lower 48 states. There were a total of 881 samples collected and analyzed between 2000 and 2005. For Chlordecone, there were 152 hits (17.25%), ranging from 12.3 and 2008 ppb. (Jensen, 2006).

In Martinique, the widespread use of Chlordecone until 1993 has resulted in contamination of soils and surface water in most of the island (Bocquené & Franco, 2005). These authors reported an investigation from 2002 of the presence of a series of pesticides in the water at the mouth of seven rivers. They measured Chlordecone in particulate matter or sediment of six of the seven rivers at concentrations up to 57 µg/kg in particulate matter, and up to 44 µg/kg in sediment.

Bocquené & Franco (2005), quoted other investigations in which concentrations of Chlordecone in the range 1.20 to 2.13 µg/L were measured in rivers of Martinique in 2002-2001. They also stated that Chlordecone was “ubiquitous” in river water used for drinking water.

Further to this, the report prepared for L’Assemblée Nationale (Beaugendre, June 2005), described the history of the use of Chlordecone in Guadeloupe and Martinique, and mentioned several monitoring programmes which are expected to result in reports at the end of 2005. However, these reports have not been available when drafting this document.

**Human exposure**

In the US ATSDR (1995), the experience from production of Chlordecone is summarised as follows: Chlordecone has not been detected in human adipose tissue or in blood samples from the general population, although historically it was detected in human milk samples collected in the south-eastern United States (EPA 1978c). Information is available regarding Chlordecone levels in blood of occupationally exposed workers and their families during 1974-1975 employed at the Hopewell, Virginia site. (Cannon et. al., 1978; Epstein 1978; Knishkowy & Baker 1986; Taylor et. al., 1978). (Quoted from US ATSDR, 1995) Further data on human exposure is quoted in section 2.4.1.

Information regarding human exposure resulting from direct use (application) of Chlordecone in the Caribbean Islands is not available. However, monitoring data in agricultural soils, crops, freshwater fish, littoral fish and shellfish indicates that human exposure more than 10 years after the use of chlordecone has ceased in Martinique and Guadeloupe, is still possible. In soils having received Chlordecone, residues in crop are proportional to soil contamination and may exceed the recommended national residues limits (50 µg/kg to 200 µg/kg). This concerns mainly root vegetables such as radish (max. measured concentration: 0.055 µg/kg), sweet potatoes (max. measured concentration: 0.300 µg/kg), taro root (max. measured concentration: 0.230 µg/kg), but also aerial part of plants, such as sugar cane (max. measured concentration: 0.690 µg/kg), or pineapple (max. measured concentration: 0.160 µg/kg). In addition, workers are directly exposed to contaminated soils. Concentrations in fisheries products (freshwater and estuarine water) have also been found to exceed in some occasions national residues limits up by a factor of 100 (max. measured concentration: 20 mg/kg). National provisions have been taken in order to prohibit fisheries activities in contaminated area (Cabidoche et. al., 2006).
HAZARD ASSESSMENT FOR ENDPOINTS OF CONCERN

Toxicity

Toxicokinetics in experimental animals and in man
The US ATSDR (1995) and EHS 43 (IPCS, 1984) both record that Chlordecone is well absorbed following oral, dermal and inhalation exposure. Toxicokinetic data are mainly available from studies in experimental animals (e.g. Blanke et. al., 1978; Boylan et. al., 1979; Cohn et. al., 1978; Egle et. al., 1978; Fujimori et. al. 1982a; Guzelian et. al., 1981; Hall et. al. 1988; Hewitt et. al., 1986b; Kavlock et. al. 1980; Plaa et. al., 1987; Richter et. al., 1979; Shah et. al., 1987; Skalsky et. al., 1980; as reported in IPCS, 1984). Following absorption, it is widely distributed in the body, with accumulation in the liver and to a lesser extent in fat, brain and kidneys, both in experimental animal studies and in humans (as reported in US ATSDR (1995) and EHS 43 (IPCS, 1984). Following administration of a single oral dose to rats at 40 mg/kg body weight, the highest concentrations were found in the adrenal glands and liver, followed by the fat and lung (Egle et. al., 1978, quoted from IPCS; 1984). Chlordecone has been reported to be slowly metabolised via reductive biotransformation to Chlordecone alcohol in the rat (Blanke et. al., 1978, as reported in EHS 43). Elimination from the body is slow, with a half-life of the order of several months and Chlordecone disappears more slowly from the liver than from other tissues (Egle et. al., 1978, quoted from IPCS, 1984). Elimination is mainly via the faeces, a total of 66% of the dose in the Egle study being removed in the faeces and 2% in the urine in the 84 days following administration (Egle et. al., 1978, quoted from IPCS, 1984).

EHC 43 reports that Chlordecone was detected in high concentrations in the liver (range 13.3-173 mg/kg), whole blood (range 0.6-32 mg/litre), and subcutaneous fat (range 2.2-62 mg/kg) of 32 male workers (Cohn et. al., 1976, adapted from IPCS (1984)). In occupationally-exposed workers, serum Chlordecone concentrations ranged from 120 to 2109 µg/litre, and dropped to 37 - 486 µg/litre 6-7 months after exposure had ceased (Adir et. al., 1978, reported in IPCS (1984)). The half-life of Chlordecone in these workers was estimated to be 63-148 days. Reductive biotransformation to Chlordecone alcohol has also been reported in humans (Blanke et. al., 1978, as reported in EHS 43). Chlordecone was eliminated, primarily in the faeces, at a mean daily rate of 0.075% of the estimated total store in the body (Cohn et. al., 1976, quoted from IPCS, 1984).

Toxicity of Chlordecone in animal studies
Chlordecone is of high acute toxicity in experimental animal studies, with an LD$_{50}$ of approximately 100 mg/kg in the rat and ranging from 65 mg/kg in the rabbit to 250 mg/kg in the dog (taken from IPCS, 1984, Table 2). Acute toxicity effects include tremors indicative of a neurotoxic effect on the nervous and/or musculoskeletal systems, investigated by many authors as reported in US ATSDR (1995). The neurotoxic effects of Chlordecone have been reported in chickens (Naber & Ware, 1965), quail (McFarland & Lacy, 1969), fish (Couch et. al., 1977), hamsters (Martinez et. al., 1976), mice (End et. al., 1979), rats (Epstein, 1978), and man (Martinez et. al., 1978). Acute oral administration of Chlordecone is also associated with reproductive effects (Khera et. al., 1976; Uzodinma et. al., 1984a; Yarbrough et. al., 1981) and hepatotoxicity in some studies (Fujimori et. al., 1983; Mehendale 1977b, 1981b; Teo & Vore 1991) (quoted from US ATSDR (1995)).

Repeated exposure to Chlordecone also causes reproductive, neurological, musculoskeletal and liver toxicity at doses as low as 10 mg/kg bw/day, although effects in other organs including kidney, thyroid, adrenals, and testes have also been reported (US ATSDR, 1995, IPCS, 1984). A Lowest-Observed-Adverse-Effect-Level (LOAEL) of 1.17 mg/kg bw/day was recorded in a 3 month feeding study in rats and signs of toxicity included focal necrosis in liver, enlargement of the adrenal gland, tremor, hyperactivity and exaggerated startle response (Cannon and Kimbrough, 1979, as quoted in US ATSDR, 1995). Histopathological changes in the liver, reduction in thyroid follicular size and colloid content and increase in epithelial cell height were reported in a 21-month gavage study in the rat, with a LOAEL of 0.07 mg/kg bw/day in males (Chu et al, 1981, as quoted in US ATSDR, 1995). Renal effects (proteinuria and increased severity of glomerulosclerosis) were seen in a 2-year feeding study in rats, with a NOAEL of 0.05 mg/kg/day (Larson et al., 1979b, as quoted in US ATSDR, 1995). Oral Chlordecone treatment caused decreased spleen and thymus weights, leukocyte counts, natural killer cell activity, and mitogenic responsiveness (EPA 1986c; Smialowicz et. al., 1985; Swanson and Wooley, 1982); decreased natural killer cell activity (Smialowicz et. al., 1985); and significant increase in plaque-forming cells (Chetty et. al., 1993c) (as reported in ATSDR, 1995). The NOAEL was 5 mg/kg bw/day and the LOAEL was 10 mg/kg bw/day.
Hepatocarcinogenicity (hepatocellular carcinoma) of Chlordecone has been demonstrated in rats and mice (males and females) (NCI 1976, Reuber, 1978, 1979, as quoted in IPCS, 1984 and US ATSDR, 1995). Tumours have been observed at doses as low as 1 mg/kg bw/day in the rat and in mice at a dose of 2.6 mg/kg bw/day (NCI, 1976, as quoted in US ATSDR (1995). The International Agency for Research on Cancer (IARC) concluded in 1987 that there was sufficient evidence that Chlordecone is carcinogenic in mice and rats and possibly carcinogenic to humans (Group 2B). Chlordecone is not genotoxic in in vitro microbial and mammalian cell gene mutation assays, in a clastogenicity test and in the dominant lethal assay (Mortelmans et al., 1986; Probst et al., 1981; Schoeny et al., 1979, Tong et al. 1981; Williams 1980, Khera et al., 1976; Simon et al., 1986, as reported in ATSDR (1995), although it has been reported to interfere with cell-to-cell communication (Tsushima et al., 1982, Caldwell and Loch-Caruso, 1992, as reported in US ATSDR (1995), suggests that it produces liver tumours by an epigenetic, tumour-promoting mechanism involving both hepatic toxicity and hypertrophy, including cytochrome P-450 induction.

Oral administration of Chlordecone to animals causes decreased fertility or fecundity and litter size, reduced sperm count and testicular atrophy (Khera et al., 1976; Linder et al. 1983; Uzodinma et al., 1984a; Yarbrough et al. 1981, as reported in US ATSDR (1995). A LOAEL of 0.83 mg/kg/day was recorded for sperm effects in a 90 day feeding study in rats, while effects on seminal vesicles and prostate were apparent at 1.67 mg/kg bw/day (Linder et al., 1983) (Quoted from US ATSDR (1995)).

Chlordecone is also a developmental toxicant. As reported in US ATSDR (1995) and EHC 43 (IPCS, 1984), gestational exposure of rats and mice to low doses of Chlordecone resulted in increased stillbirths and decreased postnatal viability, reduced fetal or neonatal weight and/or skeletal ossification and a low incidence of malformations such as renal pelvis dilatation, undescended testes, enlarged cerebral ventricles, clubfoot, fused vertebrae or ribs, and encephalocele. Chlordecone administered at levels of 2, 6, and 10 mg/kg bw/day to rats and 2, 4, 8, and 12 mg/kg body weight per day to mice on days 7 - 16 of gestation caused 19% maternal mortality in rats at the highest dose and fetuses exhibited reduced weight, reduced degree of ossification, oedema, undescended testes, enlarged renal pelvis, and enlarged cerebral ventricles. (Chernoff & Rogers, 1976, as reported in IPCS, 1984). Lower dose levels induced reductions in fetal weight and degree of ossification. Male rats born to treated dams did not show any reproductive impairment. The reproductive performance of mice fed 0, 10, 30, or 37.5 mg Chlordecone/kg diet was impaired in terms of offspring and litter size (Huber, 1965, as reported in IPCS, 1984). No litters were produced by females fed 40 mg/kg, but litter production did resume within 7 weeks following withdrawal of the Chlordecone, although litters were still smaller than those of untreated controls (quoted from IPCS (1984)). Anovulation and persistent vaginal estrus were observed in female mice given Chlordecone at a dose level of 2 mg/kg bw/day) (Swartz et al., 1988, as quoted in US ATSDR, 1995), and similar changes were observed in female offspring of maternal rats given 15 mg/kg/day of Chlordecone on gestation days 14-20 (Gellert and Wilson, 1979, as quoted in US ATSDR, 1995), although no effects on vaginal patency or fertility were observed in female offspring of maternal mice given 20 mg/kg/day during gestation days 8-12 or 14-18 (Gray and Kavlock 1984, as quoted in US ATSDR, 1995).

Toxicity of Chlordecone in humans
Available human data support the conclusion that Chlordecone has a similar toxicity profile in humans to that seen in experimental animal studies. As reported in US ATSDR (1995), a high incidence of nervous system toxicity was seen in a single group of workers exposed to Chlordecone during its manufacture (Cannon et al., 1978; Martinez et al., 1978; Sanbom et al., 1979; Taylor 1982, 1985; Taylor et al., 1978, taken from US ATSDR (1995)). Exposure of this population occurred by a combination of inhalation, oral, and dermal exposures, although the dermal route was suggested to be the predominant route. The toxicity was manifested as tremors, visual difficulties, muscle weakness, gait ataxia, in coordination, headache, and increased cerebrospinal fluid pressure (US ATSDR (1995)).

Prolonged exposure to high concentrations of Chlordecone in the workplace has been suggested to cause oligospermia and decreased sperm motility among male workers, although fertility was not impaired (Guzelian 1982a; Taylor 1982, 1985; Taylor et al., 1978, taken from US ATSDR (1995). A correlation between blood levels, atmospheric levels and sperm effects has however been difficult to prove conclusively (US ATSDR (1995). Epidemiological evidence for carcinogenicity of Chlordecone in exposed humans following inhalation exposure to Chlordecone is extremely limited (US ATSDR, 1995, IPCS, 1984). Liver biopsy samples taken from 12 workers with hepatomegaly resulting from
intermediate- or chronic-duration exposures to high concentrations of Chlordecone showed no evidence of cancer (Guzelian et. al., 1980, taken from US ATSDR (1995). However, conclusions from this study are limited by the very small number of workers sampled (US ATSDR, 1995).

**Effects on endocrine systems**

The effects of Chlordecone on reproduction indicate that this pesticide has effects on endocrine systems. It has been evaluated under the EU-Strategy for Endocrine Disrupters (EU-EDS) and has been placed in category 1 (evidence of endocrine-disrupting activity in at least one species using intact animals), in the priority list of chemicals established under the EU-Strategy. This categorisation is based on evidence of ED activity in a number of experimental systems including the mouse uterotrophic assay, increased uterine weight in rats given multiple injections of Chlordecone postnatally and receptor binding assays, indicative of an oestrogenic effect (as reported in BKH report, 2000, US ATSDR, 1995).

**Conclusion on effects assessment and toxicity of Chlordecone**

Chlordecone is readily absorbed into the body and accumulates following prolonged exposure. The pesticide is both acutely and chronically toxic, producing neurotoxicity, immunotoxicity, reproductive, musculoskeletal and liver toxicity at doses between 1 - 10 mg/kg bw/day in experimental animal studies. Liver cancer was induced in rats at a dose of 1 mg/kg body weight per day and in mice at a dose of 2.6 mg/kg bw/day, and reproductive effects are seen at similar dose levels. The International Agency for Research on Cancer has classified Chlordecone as a possible human carcinogen (IARC group 2B).

Table 2.3 summarises the outcomes of key toxicological studies on Chlordecone, including the NOAEL/LOAEL derived in each study. The studies included in this Table have been selected from the very large database on toxicological studies on Chlordecone, on the basis of the importance of the endpoint investigated (e.g. reproductive toxicity, carcinogenicity, other key target organ toxicity), robustness of the reported studies and the dose level (NOAEL/LOAEL) at which effects were reported. These studies were considered to be particularly relevant for characterisation of the toxicological risks of these compounds, and some of these studies have been used by US ATSDR to define Minimal Risk Levels (MRLs) for Chlordecone (US ATSDR, 1995).

Table 2.3 Summary of key toxicological studies on Chlordecone.

<table>
<thead>
<tr>
<th>Species</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL (mg/kg bw/day)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat Fischer 344</td>
<td>Short-term/acute toxicity 10 day repeat dose gavage study</td>
<td>65% loss in body weight, changes in clinical chemistry parameters</td>
<td>10 mg/kg bw/day (LOAEL) 5 mg/kg bw/day (NOAEL)</td>
<td>EPA, 1986 (as quoted in US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat Fischer 344</td>
<td>Short-term/acute toxicity 10 day repeat dose gavage study</td>
<td>Reductions in spleen and thymus weights, numbers of neutrophils, and natural killer cell activity, secondary to generalized toxicity</td>
<td>10 mg/kg bw/day (LOAEL) 5 mg/kg bw/day (NOAEL)</td>
<td>EPA, 1986; Smialowicz et. al., 1985, (as quoted in US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat Fischer 344</td>
<td>Short-term/acute toxicity 10 day repeat dose gavage study</td>
<td>Increased startle response</td>
<td>2.5 mg/kg bw/day (LOAEL) 1.25 mg/kg bw/day (NOAEL)</td>
<td>EPA, 1986c (as quoted in US ATSDR, 1995).</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Species</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL (mg/kg bw/day)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat (Sherman)</td>
<td>3 month feeding</td>
<td>Focal necrosis in liver, enlargement of the adrenal gland, hyperplasia and hypertrophy of cortical cells, tremor, hyperactivity, exaggerated startle response</td>
<td>1.17 mg/kg bw/day (LOAEL)</td>
<td>Cannon and Kimbrough 1979 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat, Wistar</td>
<td>2 year feeding</td>
<td>Renal effects (proteinuria and increased severity of glomerulosclerosis)</td>
<td>0.25 mg/kg bw/day (LOAEL)</td>
<td>Larson et. al., 1979b (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat Sprague-Dawley</td>
<td>21 month gavage</td>
<td>Histopathological changes in liver, reduction in follicular size and colloid content and increase in epithelial cell height in thyroid</td>
<td>0.07 mg/kg bw/day (LOAEL), in males</td>
<td>Chu et. al., 1981 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat, Wistar</td>
<td>3 month feeding</td>
<td>Testicular atrophy</td>
<td>0.5 mg/kg bw/day (LOAEL) 0.25 mg/kg bw/day (NOAEL)</td>
<td>Larson et. al., 1979b (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat (Osborne-Mendel) and mouse (B3C6F1)</td>
<td>80 week feeding</td>
<td>Hepatocellular adenoma and carcinoma</td>
<td>1.2 mg/kg bw/day (LOAEL, rat) and 2.6 mg/kg bw/day (LOAEL, mouse)</td>
<td>NCI, 1976, Reuber, 1978, 1979 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat</td>
<td>Multiple injections of Chlordecone to neonatal rats</td>
<td>Uterotrophic response - uterine weights increased in a dose-related manner</td>
<td>10 mg/kg bw/day (LOAEL, Gellert, 1978) &lt; 6 mg/kg bw/day (LOAEL, Hammond et. al., 1979)</td>
<td>Gellert 1978, Hammond et. al., 1979 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat, Hotzman strain, ovarectomized immature females</td>
<td>Rats injected x 3 with 0 - 45 mg/kg bw/day Chlordecone ± 0.01, 0.1, 1 or 10 mg/kg bw/day estradiol benzoate</td>
<td>Uterotrophic response. Effect was additive to that of estradiol benzoate over the dose range studied</td>
<td>Dose of 20 mg/kg bw/day Chlordecone appeared to be threshold for embryo implantation functions</td>
<td>Johnson, 1996</td>
</tr>
<tr>
<td>Rat</td>
<td>90-day feeding</td>
<td>Decrease in sperm motility and viability, decreased sperm, decrease in the weight of seminal vesicles and prostate</td>
<td>0.83 mg/kg bw/day LOAEL for sperm effects 1.67 ma/ka</td>
<td>Linder et. al., 1983 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Species</td>
<td>Study type</td>
<td>Effect</td>
<td>LOAEL/NOAEL (mg/kg bw/day)</td>
<td>Reference</td>
</tr>
<tr>
<td>--------------</td>
<td>--------------------------</td>
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<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Mouse, Balbc</td>
<td>130 day feeding study</td>
<td>8% decrease in litter size and 19% increase in pair-days to litter (constant oestrus)</td>
<td>1.3 mg/kg bw/day. (LOAEL)</td>
<td>Huber, 1965 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rats and mice</td>
<td>2, 6, and 10 mg/kg bw/day by gavage to rats and 2, 4, 8, and 12 mg/kg bw/day to mice on days 7 - 16 of gestation.</td>
<td>Reduced foetal weight, reduced degree of ossification, oedema, undescended testes, enlarged renal pelvis, and enlarged cerebral ventricles. Reductions in fetal weight and degree of ossification at lower dose levels. Maternal mortality at top dose. In the mouse, fetotoxicity was observed only at the highest dose level and consisted of increased fetal mortality and clubfoot.</td>
<td>2 mg/kg bw/day. (LOAEL, rat)</td>
<td>Chernoff &amp; Rogers, 1976). (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Balbc mice</td>
<td>160 day feeding study</td>
<td>Increased ovulation, persistent oestrus</td>
<td>2 mg/kg bw/day. (LOAEL)</td>
<td>Swartz et al., 1988 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
<tr>
<td>Rat</td>
<td>Reproductive toxicity</td>
<td>Increased ovulation, persistent oestrus in female offspring of maternal rats given Chlordecone on gestation days 14-20</td>
<td>15 mg/kg/day (LOAEL)</td>
<td>Gellert and Wilson, 1979, as quoted in US ATSDR, 1995</td>
</tr>
<tr>
<td>Humans</td>
<td>Occupational exposure</td>
<td>Histories of tremors, unfounded nervousness or anxiety, and visual difficulties. Also skin rashes</td>
<td>Mean blood levels of Chlordecone in workers reporting adverse effects were 2.53 ppm Skin rashes reported in workers with blood Chlordecone levels in excess of 2 µg/L</td>
<td>Cannon et al., 1978 (as quoted in IPCS, 1984 and US ATSDR, 1995).</td>
</tr>
</tbody>
</table>

**Ecotoxicity**

A summary of results of aquatic ecotoxicity tests with Chlordecone from the Ecotox database (US EPA, 2006) is given in Table 2.4.
In addition to this, the EHC 43 (IPCS, 1984), summarised a series of experiments investigating the bioavailability of Chlordecone, noting that it is strongly adsorbed on sediment. Exposure of aquatic organisms is therefore partly via the water phase and partly via sediment. D'Asaro & Wilkes (1982) examined the effects of sediments previously exposed to Chlordecone at a known concentration, and of James River sediments contaminated with Chlordecone, on an estuarine community established in aquaria supplied with non-filtered sea water. Mysid shrimps showed a dose-related mortality rate, when exposed to sediments previously equilibrated at 0.1, 1.0, or 10 µg Chlordecone/L. Mysids were not affected by James River sediment. Put concentration in sediments, if available Oysters showed dose-dependent reduced shell growth when exposed to Chlordecone-equilibrated sediments, and also responded adversely to river sediment. Lugworms Arenicola cristata died after 28 days of treatment with sediment exposed to 10 µg Chlordecone/L, though numbers were not affected by lower doses. Both lugworms and oysters concentrated Chlordecone from the sediment. (Quoted from EHC 43, (IPCS, 1984)).

Table 2.4 Summary of key ecotoxicological studies on Chlordecone.

<table>
<thead>
<tr>
<th>Taxonomic group and species</th>
<th>End point</th>
<th>Duration</th>
<th>Result mg/L</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Algae Chlorococcum sp., Dunaliella tertiolecta, Nitzschia sp., Thalassiosira pseudonana</td>
<td>EC&lt;sub&gt;50&lt;/sub&gt; growth inhibition</td>
<td>7 days</td>
<td>0.35 - 0.60 (formulation)</td>
<td>Walsh et al., 1977</td>
</tr>
<tr>
<td>Algae Chlorococcum sp., Dunaliella tertiolecta, Nitzschia sp., Thalassiosira pseudonana</td>
<td>EC&lt;sub&gt;50&lt;/sub&gt; growth inhibition</td>
<td>7 days</td>
<td>350 – 600 (formulation)</td>
<td>Hansen et al., 1977</td>
</tr>
<tr>
<td>Crustaceans Daphnia magna</td>
<td>EC&lt;sub&gt;50&lt;/sub&gt; immobility</td>
<td>48 hours</td>
<td>0.120 - 0.690</td>
<td>Barera &amp; Adams, 1983; Adams &amp; Heidolph, 1985; Ziegenfuß et al., 1986</td>
</tr>
<tr>
<td>Crustaceans Americamysis bahia, Callinectes sapidus, Palaemonetes pugio</td>
<td>LC&lt;sub&gt;50&lt;/sub&gt;</td>
<td>96 hours</td>
<td>0.01 - 0.210</td>
<td>Nimmo et al., 1977, 1981; Hansen et al., 1977; Schimmel, 1977; US EPA, 1976</td>
</tr>
<tr>
<td>Crustacean Daphnia magna</td>
<td>NOEC reproduction</td>
<td>21 days</td>
<td>0.0283</td>
<td>McKee &amp; Knowles, 1986</td>
</tr>
<tr>
<td>Crustacean Daphnia magna</td>
<td>NOEC growth</td>
<td>21 days</td>
<td>0.025</td>
<td>Adams &amp; Heidolph, 1985</td>
</tr>
<tr>
<td>Crustacean Americamysis bahia</td>
<td>MATC growth</td>
<td>28 days</td>
<td>0.000026 - 0.00034</td>
<td>Nimmo et al., 1981</td>
</tr>
<tr>
<td>Insect Chironomus tentans</td>
<td>LC&lt;sub&gt;50&lt;/sub&gt;</td>
<td>48 hours</td>
<td>0.17 - 2.3</td>
<td>Adams et al., 1985; Ziegenfuß et al., 1986</td>
</tr>
<tr>
<td>Fish 9 species</td>
<td>LC&lt;sub&gt;50&lt;/sub&gt;</td>
<td>96 hours, flow through</td>
<td>0.0066 - 0.512</td>
<td>Roberts &amp; Bendl, 1982; Roberts &amp; Fisher, 1985; Schimmel, 1977</td>
</tr>
</tbody>
</table>
In a publication from SETAC a collation of critical tissue residues (CTR) was presented and evaluated (Jarvinen et al., 1999). The database contains 32 entries for Chlordecone, with data originating from different studies (see Table 2.5). Some of the tissue residues were from studies where no effects were observed, so they may not represent the real CTR. Critical tissue residue values obtained in studies where effects were identified represent 15 CTR values for three fish species. For fathead minnow two studies are available with values of 1.7 and of 3.8-5.4 mg/kg ww. For sheepshead minnow 12 CTRs are available, ranging from 0.13 to 17 mg/kg ww with an average of 5.9 mg/kg ww. Furthermore, one CTR of 2.7 mg/kg ww for spot is available.

**Conclusion**

In summary, Chlordecone is very toxic to aquatic organisms. The most sensitive group is the invertebrates, which is not surprising for a substance with insecticidal properties. Even if the lowest effect concentration (0.000026 mg/L) was considered to be an outlier, the lowest effect concentrations would be well below 1 mg/L with the results of short term tests (mortality) in the range of 0.01 to 0.69 mg/L and those of long term tests (reproduction and growth) at 0.0025 and 0.0028 mg/L.

**Table 2.5  Collation of critical tissue residues (CTR)**

<table>
<thead>
<tr>
<th>Species</th>
<th>Life Stage</th>
<th>Exprte</th>
<th>Expo of Concentration</th>
<th>Results (g/g) (wet)</th>
<th>effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insect</td>
<td>NOEC development</td>
<td>14 days</td>
<td>17.9 mg/kg sediment</td>
<td>Adams et al., 1985</td>
<td></td>
</tr>
</tbody>
</table>

1: All are as quoted in Ecotox, US EPA 2006
<table>
<thead>
<tr>
<th>Species</th>
<th>Life Stage</th>
<th>Exprte</th>
<th>Expo of Concentration</th>
<th>Results (g/g (wet) )</th>
<th>effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Adult Water</td>
<td>1.9 µg/L</td>
<td>11 - 12</td>
<td>Survival Reduced 80%</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Adult Water</td>
<td>7.8 µg/L</td>
<td>17</td>
<td>Survival Reduced 100%</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Adult Water</td>
<td>0.16 µg/L</td>
<td>0.65 - 0.90</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo Adult fish</td>
<td>11-12 µg/g</td>
<td>11</td>
<td>Survival Reduced 25%</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo Adult fish</td>
<td>2.5 - 3.6 µg/g</td>
<td>4.7</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Larvae-Juvenile Water; Adult fish</td>
<td>1.9 µg/L; 11-12 µg/g</td>
<td>8.4</td>
<td>Survival Reduced 63%</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Larvae-Juvenile Water</td>
<td>2.0 µg/L</td>
<td>7.8</td>
<td>Survival Reduced 40%</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Larvae-Juvenile Adult fish</td>
<td>0.8 µg/L</td>
<td>2.0</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Larvae-Juvenile Adult fish</td>
<td>11-12 µg/g</td>
<td>0.13</td>
<td>Growth Reduced –</td>
<td></td>
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<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Larvae-Juvenile Water</td>
<td>0.08 µg/L</td>
<td>1.1</td>
<td>Growth Reduced –</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo-Adult Water</td>
<td>0.78 µg/L</td>
<td>5, 6.8*</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo-Adult Water</td>
<td>0.39 µg/L</td>
<td>2.2, 3*</td>
<td>Growth Reduced –</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo-Adult Water</td>
<td>0.12 µg/L</td>
<td>0.86, 1.2*</td>
<td>Growth - No effect</td>
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<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo-Adult Water</td>
<td>0.78 µg/L</td>
<td>5, 6.8*</td>
<td>Reproduction – Reduced</td>
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<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo-Adult Water</td>
<td>0.39 µg/L</td>
<td>2.2, 3*</td>
<td>Reproduction - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo, 2nd generation Adult Fish + Water</td>
<td>0.78 µg/L</td>
<td>2.3</td>
<td>Survival Reduced –</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Embryo, 2nd generation Adult Fish + Water</td>
<td>0.39 µg/L</td>
<td>1.3</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Fry, 2nd generation Adult Fish + Water</td>
<td>0.78 µg/L</td>
<td>2.3</td>
<td>Survival - No effect</td>
<td></td>
</tr>
<tr>
<td>Sheepshead minnow, Cyprinodon variegatus (Sw)</td>
<td>Fry, 2nd generation Adult Fish + Water</td>
<td>0.12 µg/L</td>
<td>0.41</td>
<td>Growth Reduced –</td>
<td></td>
</tr>
<tr>
<td>Species</td>
<td>Life Stage</td>
<td>Exprt</td>
<td>Expo of Concentration</td>
<td>Results g/g (wet)</td>
<td>effect</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>------------------</td>
<td>-------</td>
<td>------------------------</td>
<td>-------------------</td>
<td>---------------</td>
</tr>
<tr>
<td>variegatus (Sw)</td>
<td></td>
<td>Water</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sheepshad minnow, Cyprinodon variegatus (Sw)</td>
<td>Fry, 2nd generation</td>
<td>Adult Fish + Water</td>
<td>0.074 µg/L</td>
<td>0.30</td>
<td>Growth - No effect</td>
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<tr>
<td>Spot, Leiostomus xanthurus (Sw)</td>
<td>Juvenile</td>
<td>Diet</td>
<td>3.3 µg/g (wet wt)</td>
<td>2.7</td>
<td>Survival - Reduced</td>
</tr>
<tr>
<td>Spot, Leiostomus xanthurus (Sw)</td>
<td>Juvenile</td>
<td>Diet</td>
<td>3.3 µg/g (wet wt)</td>
<td>0.7</td>
<td>Survival - No effect</td>
</tr>
<tr>
<td>Spot, Leiostomus xanthurus (Sw)</td>
<td>Juvenile</td>
<td>Water; Diet</td>
<td>0.04 µg/L; 0.101 µg/g (wet wt)</td>
<td>0.144</td>
<td>Growth, No effect</td>
</tr>
</tbody>
</table>
RISK MANAGEMENT EVALUATION

Data sources

The draft Risk Management evaluation is primarily based on information that has been provided by parties to the Convention and observers.

In addition, France provided a report prepared for the Assemblée Nationale describing the history of production and use of Chlordecone in Martinique and Guadeloupe (Beaugendre, 2005) and a report on organochlorine pollution in the same region (Cabidoche et al., 2006). Specific national and international risk management reports for Chlordecone have not been identified.

Status of the chemical under international conventions

Chlordecone is listed in Annex A of the Protocol to the Convention on Long-Range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants. The provisions of the Protocol oblige Parties to phase out all production and uses of Chlordecone. Chlordecone is included in the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR Convention) as a substance of possible concern. Under the Convention on the Protection of the Marine Environment of the Baltic Sea Area (HELCOM Convention) Chlordecone is listed as selected substances for immediate priority action (Recommendation 19/5, Attachment, Appendix 3) and is scheduled for elimination (Annex I, part 2). HELCOM aims to move towards the target of the cessation of discharges, emissions and losses of hazardous substances by the year 2020.

Under the Basel Convention off-specification or out-dated pesticides are classified as hazardous in Annex VIII without further specification.

Any national or regional control actions taken

Regulation at European level

In the European Union Chlordecone is listed in Annex I to Regulation (EC) No 850/2004 on persistent organic pollutants as scheduled for elimination and with complete prohibition of production and use.

The issue of Chlordecone in waste is addressed at European level in Regulation 850/2004/EC. As amended by regulation 1195/2006/EC POP such as Chlordecone in waste have to be destroyed if concentration limits of 50 mg/kg are exceeded.

Regulation at national level

At national level legal control actions taken have been reported by Germany, Canada, the USA, Switzerland, Thailand and Japan.

In Canada production, sale, and use of Chlordecone is currently prohibited for all pesticide uses under the Pest Control Products Act (PCPA). Any stocks that existed at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable, after which their sale or use became a violation of the PCPA.

Therefore, there is no commercial reason to maintain stockpiles. In addition, Canada has established post-registration monitoring and compliance programs to ensure compliance with federal and provincial legislation. Although there is no Convention obligation to do so, federal, provincial and territorial hazardous waste programs address small quantities of retired material in the possession of consumers and have collected and safely disposed of pesticide products that are no longer registered. No further control measures are required.

5 The chemically related compound mirex is already included in the Stockholm convention. Both mirex and Chlordecone are included in the UNECE 1998 Aarhus Protocol on Persistent Organic Pollutants (POPs). Both are included in OSPAR as substances of possible concern.
In the USA all uses of Chlordecone under the authority of the Federal Insecticide, Fungicide, and Rodenticide Act have been cancelled by the USEPA in 1977. Under the Resource Conservation and Recovery Act (RCRA), chlordecone is listed as a hazardous waste when it is a discarded commercial chemical product off-specification species, container residue, and spill residue thereof (EPA 1980b quoted from US ATSDR 1995). US ATSDR 1995 contains an overview of further regulations and Guidelines applicable to Chlordecone in the USA. There is no record of any Chlordecone or Chlordecone-containing products registered to the U.S. FDA.

In Switzerland Chlordecone has been prohibited in 1986. In Mauritius Chlordecone is listed as a prohibited agricultural chemical in the Dangerous Chemicals Control Act (DCCA). The law prohibits import, manufacture, use or possess Chlordecone.

In Japan Chlordecone is included in a list of 300 substances (or group of substances) chosen by the Ministry of the Environment where the accumulation of the information for “environmental risk” is needed from the detection rates in environment and point of view of combined effects. These are substances which might have harmful effect on human health and ecosystem via water environment (i.e. “environmental risk”), but the effects are not significant, or the “environmental risk” is unknown.

In Thailand production, import, export or possession of Chlordecone for use in households and public health programs is prohibited based on the Hazardous Substances Control Act B.E. 2535 (1992). No documented evidence of action taken at national level has been reported by Zambia.

Identification of possible control measures

Legal prohibition of production and use of Chlordecone or Chlordecone-containing products have been stated as major control measures by all responding parties (Annex F responses 2007).

In addition, Canada states the elimination of stocks and their environmental sound disposal as additional type of control measure taken. “Any stocks that existed at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable, after which their sale or use became a violation of the Pest Control Products Act (PCPA). Therefore, there is no commercial reason to maintain stockpiles. Canada has established post-registration monitoring and compliance programs to ensure compliance with federal and provincial legislation. Although there is no Convention obligation to do so, federal, provincial and territorial hazardous waste programs address small quantities of retired material in the possession of consumers and have collected and safely disposed of pesticide products that are no longer registered.”

Mauritius stressed the issue of import control to prevent uses in countries which did not produce Chlordecone.

In the report submitted by France, the issue of soil decontamination by Chlordecone has been addressed. According to the report common techniques of soil decontamination such as solvent extraction and incineration are cost intensive. Microbiological degradation is not promising as it shows only low degradation rates and leads to degradation products with similar toxicity to Chlordecone itself. The authors of the study indicate that phytoremediation might be an economically viable option for the decontamination of soil which is polluted with Chlordecone. Chlordecone is taken up by specific plants from the soil. However it is noted that according to the current state of knowledge, phytoremediation requires large time scales (several centuries) to achieve similar decontamination rates as in solvent extraction (Cabidoche et al., 2006).

Besides this no further control measures have been reported or have been deemed necessary by contracting parties or observers.

As Chlordecone is an intentionally produced pesticide, the most evident and efficient control measure would be the prohibition of all production and uses of Chlordecone and Chlordecone-containing products. Alternatively, in accordance with Article 3(1), legal and administrative measures (e.g. withdrawal or denial of pre-production and pre-marketing authorisation of pesticide products) necessary to eliminate Chlordecone would have the same impact. As no remaining uses of
Chlordecone have been identified, listing of Chlordecone in Annex A without any specific exemptions could be the primary control measure under the Convention.

Listing of Chlordecone in Annex A would also mean that the provisions of Article 3 on export and import and of Article 6 on identification and sound disposal of stockpiles and waste would start to apply.

Alternatives

Information on alternative pesticides has been reported from Canada and USA. France has provided information on related to Guadeloupe and Martinique. It should be noted that the chemical alternatives mentioned below are not concluded as safe or recommended by the POP Review Committee.

Description of alternatives

According to Environment Canada several alternatives to the pesticide uses of Chlordecone are currently registered and in use in Canada. However, the table refered to, was not provided (Annex F responses, Canada 2007).

In the USA the following alternatives are registered for use to control specific pests (NPIRS, 2007, referenced in the Annex F responses, USA, 2007):

- Banana root borer: ethoprop, oxamyl.
- Tobacco wireworms: cyfluthrin, imidacloprid.
- Ants and/or cockroaches:
  - azadirachtin, bifenthrin, boric acid, carbaryl, capsaiacin, cypermethrin, cyfluthrin, deltamethrin, diazinon, dichlorvos, esfenvalerate, imidacloprid, lamda-cyhalothrin, malathion, permethrin, piperonyl butoxide, pyrethrins, pyriproxyfen, resmethrin, s-bioallerthrin, tetramethrin.

An assessment of the described alternatives has not been provided by the USEPA.

According to a French study on the use of Chlordecone in the French Antilles (Beaugrande et al., 2005) the farmers used the following substances as substitutes after the use of Chlordecone had stopped:

- Aldicarbe
- Isophenphos
- Phenamiphos
- Cadusaphos
- Terbuphos

The exemptions for the use of Chlordecone were not justified anymore as appropriate substitutes for Chlordecone were available.

According to another French study on organochlorine pollution in the French Antilles (Cabidoche et al., 2006) the pesticides actually used Guadeloupe and Martinique containing active substances such as cadusaphos and glyphosate are biodegradable within several weeks.

Alternatives pesticide products have been reviewed by the Canadian Pest Management Regulatory Agency (PMRA) and the environmental and health risks associated with their pesticide uses have been considered acceptable (Annex F responses, Canada 2007).

Algeria compiled a number of principal measures to control impacts of pesticides without specifically addressing Chlordecone as pesticide. Measures should include preventive techniques like soil aeration, mechanical control techniques like raking, burning of weeds, use of antagonistic macro-organisms (insects, parasites, predator insects), use of bio-insecticides and pesticides, use of composed measures; Application of precautionary principle, permitting, information and education, research and development, environmental sound waste management to protect environment and human health.
Croplife, the international association for pesticides industry, did not provide any information but stated that a comparative evaluation of the risk of the alternatives to Chlordecone is meaningless as a risk evaluation was never performed for Chlordecone itself (Annex F responses, Croplife 2007).

**Technical feasibility**
Alternative pesticide products are currently being utilized in Canada and the USA in a technically feasible manner. Technical feasibility is a requirement of registration by Canada’s PMRA and for registration for use in the USA. (Annex F responses, 2007).

**Costs, including environmental and health costs**
Information on costs of alternatives has not been provided from parties. In Canada however, PMRA reviewed environmental and health risks from alternatives in use and considered them acceptable (Annex F responses, Canada 2007). Correspondingly at least a slight benefit for the environment and health could be expected. There are important general points to consider when evaluating the costs of alternatives for any product as specified in (Ackerman et al., 2006):

- Alternatives with a higher initial purchase cost may actually be more cost effective over the life of the product when durability and other factors are taken into account.
- Mass-production of alternatives can significantly lower their costs.
- The costs of initiatives to protect health and the environment are frequently overestimated in advance and later decline rapidly after the regulation is implemented.

**Efficacy**
Alternative pesticide products have been reviewed by the PMRA and have been determined to be efficacious for each registered pesticide use (Annex F responses, Canada 2007). This topic has not been commented on by the USA.

**Availability**
The alternative pesticide products listed in chapter 2.1.1. were readily available in the USA. In Canada availability of all the registered alternatives listed in 2.1.1. was reported to be market dependent. (Annex F responses, 2007)

**Accessibility**
The alternatives listed in chapter 2.1.1 are accessible in the USA and in Canada was reported to be market dependent. (Annex F responses, 2007)

**Efficacy and efficiency of possible control measures in meeting risk reduction goals**

**Technical feasibility**
In all parties responding to the questionnaire production, sale, and use of Chlordecone is prohibited. This essential phase-out of production and use of Chlordecone indicates that technically feasible alternatives have already been implemented. Any stocks that existed in Canada at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable, after which their sale or use became a violation of the Pest Control Products Act (PCPA). Therefore, there is no commercial reason to maintain stockpiles. Canada has established post-registration monitoring and compliance programs to ensure compliance with federal and provincial legislation. Although there is no Convention obligation to do so, federal, provincial and territorial hazardous waste programs address small quantities of retired material in the possession of consumers and have collected and safely disposed of pesticide products that are no longer registered. Also in the USA it was deemed technically feasible to cancel the registration of all uses. (Annex F responses, 2007)

**Costs, including environmental and health costs**
The phase-out of Chlordecone that has already occurred indicates that costs of alternatives have not inhibited their substitution. For the USA, there would be no additional costs to cancel the use of Chlordecone, as USEPA cancelled these uses in 1977. In Canada disposal of no longer registered pesticides has already been taken place (see 2.2.1). No specific comments have been provided by
other parties. (Annex F responses, 2007) Costs could arise from elimination of unknown production and potential disposal of remaining stocks. Costs, however, are not expected to be important, even though no information has been provided. Benefits to health and environment are expected from decreasing environmental levels when ban of chlordecone production and use is established at a global scale.

Summary of information on impacts on society of implementing possible control measures

**Health, including public, environmental and occupational health**
No discernible negative impacts on society have been reported from prohibition or phase-out of Chlordecone as it is apparently not currently in use. A listing in Annex A would prevent future production and integration into products. This would therefore prevent negative impacts on public, environmental and occupational health that would accrue from any future production or use of chlordecone.

As production, sale and use of Chlordecone as a pesticide are prohibited in Canada, negative human health effects due to an ongoing pesticide use of Chlordecone are eliminated.

As Chlordecone has not been and is not used, no impacts in the context of pesticide use are expected from a regulation of Chlordecone under the Stockholm Convention for Germany. On a global level, a positive impact on human health can be expected from a ban of Chlordecone from the German point of view. (Annex F responses, Germany 2007)

**Agriculture, including aquaculture and forestry**
No discernible negative impacts on agriculture have been reported from prohibition or phase-out of Chlordecone due to the existence of viable alternatives.

There are no negative impacts on this sector in Canada as viable alternative pesticide products are available. A corresponding situation can be expected for the USA and other regions although no specific comment has been provided on this topic. No impacts in the context of pesticide-use are expected from a regulation of Chlordecone under the Stockholm Convention in countries which never used this pesticide. (Annex F responses, 2007)

**Biota (biodiversity)**
As production, sale and use of Chlordecone as a pesticide are prohibited in Canada, negative effects on biota due to an ongoing pesticide use of Chlordecone are eliminated.

As Chlordecone has not been and is not used, no impacts in the context of pesticide use are expected from a regulation of Chlordecone under the Stockholm Convention for Germany. On a global level, a positive impact on biota can be expected from a ban of Chlordecone from the German point of view. (Annex F responses, Germany 2007)

**Economic aspects**
No negative economic impacts to Canada are apparent through the current prohibition of Chlordecone as a pesticide. As Germany does not use Chlordecone, no impacts in the context of pesticide-use are expected from a regulation of Chlordecone under the Stockholm Convention. Information for other countries is not available, however cost competitive alternatives that do not exhibit POPs characteristics have already been implemented for all uses of Chlordecone, so no negatives economic impacts from a global ban on Chlordecone are expected.

**Movement towards sustainable development**
The prohibition of Chlordecone contributes positively to sustainable development in that protection of crops through previous Chlordecone pesticide uses is still maintained by alternative methods and the risk to the environment and human health is less (Canada).

As the persistent, bioaccumulative and toxic properties of Chlordecone as well as its potential for a long-range transboundary transport were shown under the POPs-Protocol and by the POP RC of the Stockholm Convention which concluded that Chlordecone meets the screening criteria listed in Annex
D, a positive impact on a globally sustainable development from a ban/restriction of the substance is to be expected (Germany). As Germany does not use Chlordecone, no impacts in the context of pesticide use on the national level are expected from a regulation of Chlordecone under the Stockholm Convention.

Reduction and elimination of Chlordecone is consistent with sustainable development plans that seek to reduce emissions of toxic chemicals. A relevant global plan is the Strategic Approach to International Chemicals Management (SAICM) that emerged from the World Summit on Sustainable Development. The Global Plan of Action of SAICM contains specific measures to support risk reduction that include prioritizing safe and effective alternatives for persistent, bioaccumulative, and toxic substances.

**Social costs**

No negative social costs to Canada are apparent through the current prohibition of Chlordecone used as a pesticide. Since Chlordecone has already been replaced with other substances or technologies, the impact of an Annex A listing on consumers and farmers should be invisible and not incur any social costs.

**Other impacts (waste and disposal implications- technical feasibility)**

Technical feasibility of waste and disposal of Chlordecone is not applicable any more in Canada, as any stocks that existed at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable. Canada has established post-registration monitoring and compliance programs to ensure compliance with federal and provincial legislation and federal, provincial and territorial hazardous waste programs address and have collected and safely disposed of small quantities of retired pesticide products in the possession of consumers.

As the pesticide was not applied in Germany, no obsolete stocks of Chlordecone are expected to be found. But, the introduction of a critical threshold for Chlordecone in waste (regulation 1195/2006/EC8) will lead to measures taken in Germany as well. At the current moment no information on costs is available.

Further Regulations concerning the annexes of directive (EC) 850/2004 are expected to be elaborated for the European Union. These are related to thresholds and regulations of destruction measurements.

All in all, no data on existing Chlordecone stockpiles have been provided but it can be assumed that some countries may still possess obsolete stockpiles which would need to be managed as waste. At least two regions (Sub-Saharan Africa and South East Asia / South Pacific) have identified Chlordecone as a possible substance of concern in their Regionally Based Assessment of Persistent Toxic Substances but no further information on possible obsolete stockpiles is provided in those reports. (UNEP 2002a, UNEP 2002b)

**Other considerations**

**Access to information and public education**

The Pest Management Regulatory Agency of Health Canada (PMRA) provides a wide variety of information regarding pesticide regulation through its web site, www.pmra-arla.gc.ca, including information regarding regulatory decisions taken on pest control products. In taking regulatory decisions on registered products, the PMRA considers the availability of alternatives, and includes relevant information in its documentation. The PMRA website also provides access to a Public Registry that includes a collection of information on pesticides or the pesticide regulatory system, including all publicly available information on currently registered pesticides.

In the Czech Republic information on Chlordecone is part of the SC/UN ECE CRLTAP education and awareness raising campaign under the national implementation plan.

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7 [http://www.chem.unep.ch/saicm/](http://www.chem.unep.ch/saicm/)
8 Amending Regulation (EC) 850/2004
In Zambia access to environmental information is low, though it has improved in the recent past (ECZ 2001, State of the Environment, Lusaka, Zambia).

**Status of control and monitoring capacity**

Information on control and monitoring capacity has been provided by Canada, Czech Republic and Zambia only. In Canada control and monitoring capacity of pesticide uses is managed by the PMRA through compliance mechanisms in place at border crossings and entry points to prohibit import of Chlordecone or any other applicable chemicals into Canada. Compliance issues within Canada may be identified to the PMRA through the following avenues:

- PMRA compliance activities;
- reporting of suspected infractions; and/or
- results reported from other government agencies.

In Zambia general chemical control and monitoring capacity available through the Environmental Protection and Pollution Control Act which is enforced by the Environmental Council of Zambia (ECZ 2001, State of the Environment, Lusaka, Zambia).

In the Czech Republic specific control and monitoring capacity for Chlordecone has been reported as none. Other Parties and observers did not cover this topic in their answers.

In general, listing Chlordecone in Annex A will involve control measures that are straightforward to communicate and therefore should be effective and suitable, even in countries that have limited chemical regulatory infrastructure.

**Synthesis of information**

According to the Risk Profile on Chlordecone main production of Chlordecone in the USA ceased in 1975 and use of Chlordecone (or related formulations) may have largely ceased by the end of the eighties, but it is assumed that it can still be produced or used as an agricultural pesticide in some developing countries, although there are no reports of such production or use. In French overseas territories Chlordecone has been used until September 1993. If it is still used as pesticide, it will be directly released to the environment. Moreover, the high persistency of the substance has caused high contamination of soil and waters in areas where it has been used and these contaminated sites can serve as a source of pollution for long times.

Chlordecone is already listed in Annex I of the CLRTAP POP Protocol and in the European POP Regulation (EC) No 850/2004. In addition it is addressed under the OSPAR and HELCOM convention. At the national level a legal ban has been reported by Germany, Canada, the USA and Switzerland. In Japan Chlordecone is included in a list of substances where further information on “environmental risk” is sought.

Chlordecone is an intentionally produced pesticide and thus the most evident and efficient control measure would be the prohibition of all production and uses of Chlordecone and Chlordecone containing products. As no remaining production uses of Chlordecone have been identified, listing of Chlordecone in Annex A without any specific exemptions would be the primary control measure under the Convention. Listing of Chlordecone in Annex A would also mean that the provisions of Article 3 on export and import and of Article 6 on identification and sound disposal of stockpiles and waste would apply.

As production of Chlordecone has ceased some decades ago in the main producing countries, availability of alternatives, efficacy and cost implications do not constitute a problem. Based on the same background significant impacts on society are not expected if Chlordecone is listed in Annex A of the Convention. No needs for specific exemptions have been identified.

A beneficial effect could be expected as currently unknown production in parts of the world could cease. In addition, potential ongoing uses in developing countries or management and disposal of potentially remaining stocks would be further regulated. Finally reintroduction of Chlordecone which currently remains possible and which would directly lead to increased releases and levels in the environment would be prevented on a global scale.
To effectively avoid releases of Chlordecone into the environment however, also the issue of environmental degradation of related substances or derivates (such as Kelevan) into Chlordecone would have to be taken into consideration. Simple listing of Chlordecone in Annex A would not cover this type of release, unless a supplementary provision will be added in Annex A Part II.
Concluding statement

The Committee decides that it is important to prevent the re-introduction of Chlordecone, recognizing it is not known to be currently produced or used.

Therefore, in accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends the Conference of the Parties to the Stockholm Convention to consider listing and specifying the related control measures of Chlordecone in Annex A. As no remaining production or uses of Chlordecone have been identified, listing of Chlordecone in Annex A without any specific exemptions is feasible.
References


- (NPIRS, 2007) National Pesticide Information Retrieval System (NPIRS) is a collection of pesticide-related databases available by subscription. NPIRS is under the administration of the Center for Environmental and Regulatory Information Systems at Purdue University in West Lafayette, Indiana, USA, 2007, available at http://ppis.ceris.purdue.edu/npublic.htm


B.4. Endosulfan – SUMMARY

SUMMARY
4. Endosulfan

Proposal by the European Union August 2007
http://www.pops.int/documents/meetings/poprc/docs/chem_review.htm

Supporting information by German Federal Environment Agency

<table>
<thead>
<tr>
<th>Composition</th>
<th>There are four relevant forms of endosulfan: alpha endosulfan, beta endosulfan, endosulfan sulphate, and technical endosulfan which is a 2:1 to 7:3 mixture of the alpha and beta isomers.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Insecticide for control of aphids, thrips, beetles, foliar feeding larvae, mites, borers, cutworms, bollworms, whiteflies, and leafhoppers. Used on cotton, tobacco, cantaloupe, tomatoes, squash, eggplant, sweet potato, broccoli, pears, pumpkins, corn, cereals, oilseeds, potatoes, tea, coffee, cacao, soybean, and other vegetables. Historically used to control termites and tsetse fly. Used in some countries in the past as a wood preservative.</td>
</tr>
<tr>
<td>Releases</td>
<td>The vast majority of endosulfan is used as active ingredient of plant protection products. That means it is deliberately spread over large soil or plant areas. Worldwide production estimated at 10,000 metric tonnes, however, current global production is likely to be significantly higher as use remains widespread. Recently the GAPS study, a global monitoring project on POPs, revealed that endosulfan “showed highest values of all the organochlorine pesticides (OCPs) investigated, in the range of tens to hundreds of pg/m³, with a geometric mean of 58”. Endosulfan was also among those organochlorine chemicals which were present in highest concentrations worldwide in samples from tree bark lipids. Unlike for more volatile compounds no significant correlation with geographical latitude was found. The authors concluded that these compounds are not as effectively distilled and tend to remain near the original region of use.</td>
</tr>
<tr>
<td>Fate</td>
<td>In the environment, endosulfan is oxidized in plants and in soils to form primarily endosulfan sulfate and endosulfan-diol. Formation of endosulfan sulfate is mediated essentially by micro-organisms, while endosulfan-diol was found to be the major hydrolysis product. Endosulfan was measured repeatedly in Arctic seawater during the 1990s. Mean concentrations were similar to those of chlordane. Concentrations of endosulfan from Arctic air monitoring stations increased from early to mid-1993 and remained at that level through the end of 1997. Reported values for measured bio-concentration factors of endosulfan in various aqueous organisms cover a wide range from 100 in oysters to 11,000 in whole fish. Half-lives in acidic to neutral soils range from one to two months for α-endosulfan and from three to nine months for β-endosulfan under aerobic condition. The estimated half-lives for the combined toxic residues (endosulfan+ endosulfan sulfate) ranged from roughly 9 months to 6 years. Anaerobic conditions may considerably extend half-lives in soils.</td>
</tr>
<tr>
<td>Effects</td>
<td>The oxidised metabolite, endosulfan sulfate, shows an acute toxicity similar to</td>
</tr>
</tbody>
</table>
that of the parent compound. In contrast, endosulfan-diol, which is another metabolite of endosulfan, is found substantially less toxic to fish by about three orders of magnitude. Recent literature has indicated the potential for endosulfan to cause some endocrine disruption in both terrestrial and aquatic species. Effects observed were impaired development in amphibians, reduced cortisol secretion in fish, impaired development of the genital tract in birds and hormone levels, testicular atrophy and reduced sperm production in mammals. Excessive and improper application and handling of endosulfan have been linked to congenital physical disorders, mental retardations and deaths in farm workers and villagers in developing countries in Africa, southern Asia and Latin America. Endosulfan was found among the most frequently reported intoxication incidents, adding unintentionally further evidence to its high toxicity for humans. In laboratory animals, endosulfan produces neurotoxicity effects, which are believed to result from over-stimulation of the central nervous system. It can also cause haematological effects and nephrotoxicity. The α-isomer was generally found more toxic than the β-isomer.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Endosulfan was detected in adipose tissue and blood of polar bears from Svalbard. Endosulfan has also been detected in blubber of minke whale and in liver of northern fulmar. Endosulfan was detected in all lake trout examined from isolated Ontario (Canada) and New Brunswick lakes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Status</td>
<td>Endosulfan has been included in the OSPAR List of Chemicals for Priority Action (update 2002). Endosulfan is on the list of priority substances agreed by the Third North Sea Conference (Annex 1A to the Hague Declaration). The second meeting of the Chemical Review Committee agreed to recommend to the Conference of the Parties that endosulfan should be listed in Annex III of the Rotterdam Convention and developed a rationale setting out how the criteria in Annex II had been met.</td>
</tr>
<tr>
<td>Alternatives</td>
<td>Will be discussed in Annex F evaluation if Endosulfan advances.</td>
</tr>
</tbody>
</table>
B.4.a. Endosulfan – DETAILED PROFILE

Endosulfan
Introduction

Endosulfan, a synthetic organochlorine compound, is widely used as an agricultural insecticide. It was introduced into the market already back in the mid 1950s but plant production products containing endosulfan are still used in a number of countries worldwide. In scientific literature a huge number of information is available, dealing with (eco)toxicity, environmental fate, residues in food and feedstuff, environmental concentrations, etc. of Endosulfan. In addition a number of various reviews were published during the last decade.

This dossier focuses solely on the information required under paragraphs 1 and 2 of Annex D of the Stockholm Convention and it is mainly based on the following documents:
- US EPA’s re-registration eligibility decision (RED) 9.
- Toxicological profile for endosulfan published by the U.S. Department of Health and Human Services 10.
- Final review of endosulfan by the Australian National registration authority for agricultural and veterinary chemicals 11.
- Arctic Monitoring and Assessment Programme (AMAP) 14.
- US EPAs and Environment Canada’s common monitoring project IADN (Integrated Atmospheric Deposition Network) 15.
- OSPAR List of Potential Endocrine Disruptors - Part B 17.

These extensive review reports also serve as a source of further information referred to in paragraph 3 of Annex D of the Stockholm Convention on this candidate POP chemical.

1 Identification of the chemical

1.1 Names and registry numbers

<table>
<thead>
<tr>
<th>common name</th>
<th>endosulfan</th>
</tr>
</thead>
<tbody>
<tr>
<td>IUPAC</td>
<td>6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxide</td>
</tr>
<tr>
<td>Chem. Abstracts</td>
<td>6,9-methano-2,4,3-benzodioxathiepin-6,7,8,9,10,10-hexachloro-1,5,5°,6,9,9-hexahydro-3-oxide</td>
</tr>
</tbody>
</table>

| CAS registry numbers | • alpha (α) Endosulfan 959-98-8 |
|                      | • beta (β) Endosulfan 33213-65-9 |
|                      | • technical Endosulfan 115-29-7 |
|                      | • Endosulfan sulfate: 1031-07-8 |
|                      | • * stereochemically unspecified |

| trade name | Thiodan®, Thionex, Endosan, Farmoz, Nufarm, Endosulfan |

* technical endosulfan is a 2:1 to 7:3 mixture of the α- and the β-isomer.

1.2 Structures

| formula | C₉H₆Cl₆O₃S |

9 [http://www.epa.gov/oppsrrd1/REDs/endosulfan_red.pdf](http://www.epa.gov/oppsrrd1/REDs/endosulfan_red.pdf)
12 [to be published by the Spanish Authorities](http://www.epa.gov/glhpo/fund/projects/99projects/integrated.html)
13 [http://www.inchem.org/documents/hsg/hsg/hsg017.htm](http://www.inchem.org/documents/hsg/hsg/hsg017.htm)
14 [http://www.amap.no/](http://www.amap.no/)
16 [http://www.chem.unep.ch/pts/regrepts/North%20America%20full%20report.pdf](http://www.chem.unep.ch/pts/regrepts/North%20America%20full%20report.pdf)
molecular mass  | 406.95 g/mol
---|---
structural formulas | 
[![alpha-endosulfan](image1.png)](image1.png) | [![beta-endosulfan](image2.png)](image2.png)

2 Persistence

In the environment, endosulfan is oxidized in plants and in soils to form primarily endosulfan sulfate and endosulfan-diol\(^\text{18}\). Formation of endosulfan sulfate is mediated essentially by micro-organisms, while endosulfan-diol was found to be the major hydrolysis product. Microbial mineralisation is generally slow.

Given a comparable toxicity of the sulfate metabolite a number of authors make use of the term “endosulfan(sum)” which includes the combined residues of both isomers of the parent and endosulfan sulfate.

In five different soil types, under aerobic conditions, DT\(_{50}\) values of 12 to 39 d (mean: 27.5 d) and 108 - 264 d (mean of 157 d) were determined for the \(\alpha\)-isomer and \(\beta\)-isomer, respectively. Encompassing both isomers and the metabolite endosulfan sulfate (“total endosulfan”) values of 288 to 2,241 days resulted for DT\(_{50}\)\(^\text{19}\).

Half-lives in acidic to neutral soils range from one to two months for \(\alpha\)-endosulfan and from three to nine months for \(\beta\)-endosulfan under aerobic condition. The estimated half-lives for the combined toxic residues (endosulfan+ endosulfan sulfate) ranged from roughly 9 months to 6 years\(^\text{20}\). Anaerobic conditions may considerably extend half-lives in soils.\(^\text{21}\)

In two tropical soils from Brazil dissipation half-lives of endosulfan (total endosulfan) were determined to > 161 and 385 days\(^\text{22}\).

Hydrolytic breakdown of endosulfan is enhanced with increasing pH resulting in DT\(_{50}\) of 10-20 days at pH 7 and around 0.2 days at pH 9 (at 25 °C)\(^\text{23}\). In alkaline sea water hydrolysis is deemed to be the main degradation process.

Photochemical transformation does not contribute to environmental breakdown in water since endosulfan does not absorb solar radiation of the troposphere (wavelengths > 290 nm). No indication for potential photo-transformation in natural water bodies could be made available from literature.

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\(^{22}\) Laabs, V. \textit{et al}. Fate of 14C-labelled soybean and corn pesticides in tropical soils of Brazil under laboratory conditions. J. Agric. Food Cehm. 50, 4619-4627 (2002).

\(^{23}\) To be added [178]
3 Bioaccumulation

Reported values for measured BCF of endosulfan in various aqueous organisms cover a wide range. In some species like oysters and bivalves BCF values as low as < 100 are reported, while on the other end studies on freshwater as well as marine fish suggest bioconcentration factors from 2,400 up to 11,000 in whole fish.

4 Potential for long-range environmental transport

There is much information available from studies on volatile soil losses to basically support the presence of endosulfan at distant sites and as a global pollutant.

An atmospheric half-life of 27 d (± 11 days) was estimated at 75 C based on concentration of [OH] = 5 x 10^5 cm^-3 in an experiment using a direct measurement techniques. Taking into account much lower temperatures of the troposphere, environmental half life of endosulfan might even be longer. Half-lives of > 2.7 days were found for α-endosulfan and of > 15 days for β-endosulfan in an experiment using an indirect measurement technique.

---

Evidence for long range transport of endosulfan and endosulfan sulfate is provided from a number of literature sources reporting concentrations in various environmental media from Arctic regions. Concentrations of endosulfan from Arctic air monitoring stations increased from early to mid-1993 and remained at that level through the end of 1997 at 0.0042-0.0047 ng/m$^3$.  Endosulfan was measured repeatedly in Arctic seawater during the 1990s. Mean concentrations were similar to those of chlordane and ranged from 2-10 pg/L.

Endosulfan was detected in adipose tissue and blood of polar bears from Svalbard. Mean values found for $\alpha$-endosulfan were 3.8 ± 2.2 ng/g wet weight and 2.9 ± 0.8 ng/g for $\beta$-endosulfan. Endosulfan has also been detected in blubber of minke whale and in liver of northern fulmar.

Recent modelling data of EMEP Meteorological Synthesizing Centre East show that once released in Central Europe endosulfan, may spread out over the Northern Atlantic reaching areas of Greenland.

5 Adverse effects

Endosulfan is a very toxic chemical for nearly all kind of organisms. Metabolism occurs rapidly, but the oxidised metabolite endosulfan sulfate shows an acute toxicity similar to that of the parent compound. In contrast, endosulfan-diol, which is another metabolite of endosulfan is found substantially less toxic to fish by about three orders of magnitude.

Numerous test results on effects of endosulfan and endosulfan sulfate on fish and aqueous invertebrates are available. The pattern of study results clearly establishes a high toxicity of endosulfan and its formulated end-products to aqueous organisms, in particular to aqueous vertebrates.

Recent literature has indicated the potential for endosulfan to cause some endocrine disruption in both terrestrial and aquatic species. Effects observed were impaired development in amphibians, reduced cortisol secretion in fish, impaired development of the genital tract in birds and hormone levels, testicular atrophy and reduced sperm production in mammals resulting from endosulfan exposure. Excessive and improper application and handling of endosulfan have been linked to congenital physical disorders, mental retardations and deaths in farm workers and villagers in developing countries in Africa, southern Asia and Latin America. Endosulfan was found among the most frequently reported intoxication incidents, adding unintentionally further evidence to its high toxicity for humans.

30 Meakin, S. What’s New with POPs Research in the Arctic Northern Perspectives 26 (1), 6-7 (2000).
31 Indian and Northern Affairs Canada (INAC). The Canadian Arctic Contaminants Assessment Report II (CACAR II), (2002).
33 Hobbs, K.E. et al. Levels and patterns of persistent organochlorines in minke whale (Balaenoptera acutorostrata) stocks from the North Atlantic and European Arctic. Environmental Pollution 121 (2), 239-252, (2003).
In laboratory animals, endosulfan produces neurotoxicity effects, which are believed to result from over-stimulation of the central nervous system. It can also cause haematological effects and nephrotoxicity. The α-isomer was generally found more toxic than the β-isomer.\(^\text{38}\)

Investigations of chronic human toxicity exert endosulfan to be neither a carcinogen nor a reproductive toxin nor a teratogen in mammals. There are several results *in vitro* and *in vivo* showing no mutagenic effect.

### 6 Statement of the reasons for concern

According to the available data, endosulfan is very persistent in the environment and is frequently found in environmental compartments. It has a great potential for bioaccumulation. Due to its physical and chemical properties and atmospheric half-life, and based on modelling data and findings in environmental samples, it has been proved that endosulfan is transported long distances, far from its sources. Endosulfan is a very toxic chemical for nearly all kind of organisms. Endosulfan has the potential to cause some endocrine disruption in both terrestrial and aquatic species. Endosulfan causes neurotoxicity and haematological effects and nephrotoxicity.

Placing on the market and use of endosulfan has been prohibited in the European Union. However, it is still produced in some countries (Worldwide production estimated at 10,000 metric tonnes.) and it continues to be used in many countries. Given the inherent properties of endosulfan, together with demonstrated or potential environmental concentrations that exceed maximum permissible concentrations; and given the widespread occurrence of endosulfan, including in remote areas; it is concluded that endosulfan is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

## B.5. Hexabromobiphenyl – SUMMARY

### SUMMARY

5. Hexabromobiphenyl (HBB)

Draft Risk Management Evaluation May 2007


Risk Profile UNEP/POPS/POPRC.2/17/Add3


| Composition | Hexabromobiphenyl belongs to a wider group of polybrominated biphenyls (PBBs). The term “polybrominated biphenyls” or “polybromobiphenyls” refers to a group of brominated hydrocarbons formed by substituting hydrogen with bromine in biphenyl. The hexabromo congeners exist as 42 possible isomeric forms. Trade names include FireMaster BP-6 and FireMaster FF-1. The commercial production of PBBs began in 1970. Approximately 6 million kg of PBBs were produced in the United States from 1970 to 1976. Production and use of hexabromobiphenyl has ceased in most, if not all, countries. However, it is possible that hexabromobiphenyl is still being produced in some developing countries or in countries with economies in transition. |
| Uses | Hexabromobiphenyl has been used as a fire retardant in acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business, machine housings and in industrial and electrical products and in polyurethane foam for auto upholstery. A considerable part of the substance produced will probably reach the environment sooner or later because of the high stability of these compounds. |
| Releases | Data for loss into the environment during normal production are published only for the United States. In 1973, an accidental release of PBBs occurred in Michigan (referred to as the “Michigan disaster” in EHC 152), when two products manufactured by the Michigan Chemical Company were inadvertently confused and 250-500 kg (Di Carlo *et al.*, 1978) of FireMaster®, instead of NutriMaster®, a magnesium oxide-based cattle feed supplement, were added to animal feed and distributed to farms within the state. This accidental mix-up resulted in widespread contamination by PBBs. Approximately 5350 tonnes of hexabromobiphenyl were used in commercial and consumer products in the United States, most in the production of plastic products with an estimated use life of 5–10 years (Neufeld *et al.*, 1977). Since the cessation of production, all of these products, such as TV cabinet and business machine housings, are expected to have been disposed of by landfilling or incineration (Neufeld *et al.*, 1977) |
| Fate | According to available data, hexabromobiphenyl can be considered to be highly persistent in the environment. There is evidence of low or no degradation in water, soil and sediment, in the laboratory as well as in the field. Hexabromobiphenyl is less volatile than many of the currently listed POP substances. However, extensive data on monitoring shows that it is found throughout the Arctic wildlife, demonstrating that it does have a high potential for long range environmental transport. With measured weight-based BCF values in the range 4,700–18,100 and biomagnification factors in the aquatic food chain exceeding 100, hexabromobiphenyl is considered to be highly bioaccumulative and to have a high potential for biomagnification. These properties are demonstrated by several authors to be comparable to those of |
hexachlorobiphenyl (a PCB compound), for which the bioaccumulative properties are well documented.

| Effects | Hexabromobiphenyl is readily absorbed into the body and accumulates following prolonged exposure. Although the acute toxicity of hexabromobiphenyl is low, a number of chronic toxic effects including hepatotoxicity have been observed in experimental animals at doses around 1 mg/kg bw/day following long-term exposure, and effects are seen in the rat thyroid at doses as low as 0.05 mg/kg bw/day. The International Agency for Research on Cancer has classified hexabromobiphenyl as a possible human carcinogen (IARC group 2B). The PBBs are endocrine disrupting chemicals, and effects are seen on reproductive capacity in rats, mink and monkeys. There is epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer in exposed women. Data on toxicity to other species than laboratory mammals is scarce but suggests the environmental toxicity of hexabromobiphenyl is comparable to that of hexachlorobiphenyl. |
| Exposure | Recent monitoring data in soil, water and sediments for PBBs are limited. Historical monitoring data from the United States indicate that environmental PBB concentrations are confined to areas near former manufacturing facilities and regions of Michigan affected by the farm accident of the early 1970's (see Section 2.2.3) (US ATSDR, 2004). The only available data for environmental concentrations of PBBs in areas outside the vicinity of former production sites are those from sediment samples from Greenland (Vorkamp et al., 2004), where PBBs (including PBB 153) were not detected in any sample (the limits of detection/quantification are, however, not well defined in the paper). The US ATSDR (2004), considers the current human exposure to PBBs to be very low, because PBBs are no longer produced or used. Thus, the general population exposure to PBBs will only be from historical releases. For people residing in the lower peninsula of Michigan, especially in the immediate vicinity of the PBB contaminated areas of this region, exposure to PBBs may still be occurring today. However, environmental levels have decreased since the 1970s and current exposure, if any, will be at low levels. For other regions of the United States, the levels of exposure will either be very low or none (Quoted from US ATSDR, 2004). In Arctic and North Atlantic regions, where the traditional diet includes top predators (e.g. seal in Greenland and pilot whale in the Faroe Islands), exposure has not ceased. Especially the level of PBBs in pilot whale blubber of up to 17 µg/kg lipid indicate the presence of hexabromobiphenyl in food. Pilot whale blubber is consumed as a delicacy in the Faroe Islands. |
| Status | Hexabromobiphenyl is listed in Annex A of the Protocol to the Convention on Long-range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants. The provisions of the Protocol oblige Parties (currently 25) to phase out all production and uses of hexabromobiphenyl. Hexabromobiphenyl, together with other PBBs, is also included in the UNEP/FAO Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade. Under the Helsinki Convention on the Protection of the Marine Environment of the Baltic Sea Area (HELCOM) hexabromobiphenyl is listed as a selected substance for immediate priority action (Recommendation 19/5, Attachment, Appendix 3) and is scheduled for elimination (Annex I, part 2). HELCOM aims to move towards the target of the cessation of discharges, emissions and losses of hazardous substances by the year 2020. Under the Basel Convention, PBBs are classified as hazardous in Annex VIII without further specification. SAICM does not specifically address Hexabromobiphenyl but includes POPs as a class of chemicals that might be prioritized for assessment and related studies. |
Alternatives

The hexabromobiphenyl risk profile describes three principal commercial products that contained hexabromobiphenyl in the USA and Canada: 1) acrylonitrile-butadiene-styrene (ABS) thermoplastics used for business machine housings and electrical products such as radio and TV; 2) fire retardant in cable coatings and lacquers, and 3) fire retardant in polyurethane foam for auto upholstery. A number of reports on risk assessment of alternative substances and processes are available. The OSPAR priority substances Series (OSPAR, 2001) provides summary information on alternatives for brominated flame retardants. The Danish Environmental Protection Agency has described alternative halogen-free flame retardants for a variety of uses including epoxy, phenolic resins, rigid and soft polyurethane foam, textiles, and a variety of plastics including ABS (Danish EPA, 1999). Both drop-in chemical substitutes and alternative materials are listed. US EPA has described process alternatives and chemical substitutes for polyurethane foam (USEPA, 2005). The German Federal Ministry of Environment has reported on alternatives for flame retardants used in electronics, upholstery, and other sectors (BMU, 2000). As brominated flame retardants only account for about 15% of the global flame retardant consumption, principally a large number of compounds may be considered as alternatives (OSPAR, 2001). Substitution can take place at three levels: 1) brominated flame retardants can in some applications be replaced by another flame retardant without changing the base polymer; (major group of substitutes); 2) the plastic material, i.e. the base polymer containing flame retardants and other additives, can be replaced by another plastic material; (e.g. polysulfone, polyaryletherketone and polyethersulfone) 3) a different product can replace the product, e.g. the plastic material is replaced by another material (e.g. wool), or the function can be fulfilled by the use of a totally different solution.
B.5.a. Hexabromobiphenyl – DETAILED PROFILE

Candidate for POPs List

Hexabromobiphenyl
Background

The European Community and its Member States being Parties to the Stockholm Convention have proposed hexabromobiphenyl to be listed in Annex A to the Convention.

Introduction

Hexabromobiphenyl belongs to a wider group of polybrominated biphenyls. The term polybrominated biphenyls or polybromobiphenyls (PBBs) refers to a group of halogenated hydrocarbons, formed by substituting hydrogen by bromine in biphenyl. These intentionally produced chemicals have mainly been used as flame retardants in synthetic fibres and plastics. Technical PBBs contain several PBB compounds, isomers and congeners, hexabromobiphenyl being one of the main components.

Hexabromobiphenyl has been identified as a Persistent Organic Pollutant (POP) chemical under the Protocol to the Convention on Long-range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants and the provisions of the Protocol oblige Parties to phase out all production and uses of hexabromobiphenyl.

1. Identification of the chemical

1.1. Names and registry numbers

Hexabromobiphenyl belongs to a wider group of polybrominated biphenyls (PBBs). The term “polybrominated biphenyls” or “polybromobiphenyls” refers to a group of brominated hydrocarbons formed by substituting hydrogen with bromine in biphenyl. The hexabromo congeners exist as 42 possible isomeric forms.

**CAS chemical name:**
Hexabromo-1,1’-biphenyl

**Synonyms:**
Hexabromobiphenyl
Biphenyl, hexabromo
1,1’-biphenyl, hexabromo-HBB

**Trade names:**
FireMaster(R) BP-6
FireMaster(R) FF-1

Technical grade PBBs (FireMaster(R)) contain several PBB compounds, isomers and congeners, hexabromobiphenyl being one of the main components. The composition of FireMaster(R) BP-6 changes from batch to batch, but its main constituents are 2,2’,4,4’,5,5’-hexabromobiphenyl (60-80%), and 2,2’,3,4,4’,5,5’-heptabromobiphenyl (12-25%) together with lower brominated compounds. Mixed bromochlorobiphenyls and polybrominated naphthalenes have also been observed as minor components of FireMaster(R) (EHC 152 (IPCS, 1994)). FireMaster FF-1 (white powder) is FireMaster BP-6 (brown flakes) to which 2% calcium silicate has been added as an anti-caking agent (EHC 152 (IPCS, 1994)).

Additional data on the composition of identified PBB congeners in FireMaster(R) BP-6 and FireMaster(R) FF-1 is given in US ATSDR (2004).
**CAS registry number:**
- 6355-01-8\(^{39}\) (Common CAS number for hexabromobiphenyl isomers)
- 59536-65-1 (EHC 192 (IPCS, 1997))\(^{40}\)
- 67774-32-7 (EHC 192 (IPCS, 1997))\(^{41}\)

US ATSDR (2004) provides CAS numbers for a wider number of individual hexabromobiphenyl isomers, as shown in Annex B.

### 1.2. Chemical Structure

![Structure of 2,2',4,4',5,5' hexabromobiphenyl (CAS No. 59080-40-9, PBB congener No. 153).](image)

Chemical formula:
\[ \text{C}_{12}\text{H}_{4}\text{Br}_6 \]

Molecular weight:
627.58

### 1.3. Physical chemical properties

The physical and chemical properties of hexabromobiphenyl are listed in Table 1.1.

---

\(^{39}\) The CAS registry number 36355-01-8 is given as a generic CAS number for PBBs in the 1988 EU Export-Import Regulation and the UNEP Rotterdam Convention.

\(^{40}\) US ATSDR refers to Firemaster\textsuperscript{(R)} BP-6 as CAS No. 59536-65-1.

\(^{41}\) US ATSDR refers to FireMaster\textsuperscript{(R)} FF-1as CAS No. 67774-32-7.
Table 1.1  Physical and chemical properties of hexabromobiphenyl.

<table>
<thead>
<tr>
<th>Property</th>
<th>Unit</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula:</td>
<td></td>
<td>C_{12}H_{4}Br_{6}</td>
<td></td>
</tr>
<tr>
<td>Molecular weight:</td>
<td>g/mol</td>
<td>627.58</td>
<td></td>
</tr>
<tr>
<td>Appearance at normal temperature and pressure</td>
<td></td>
<td>White solid</td>
<td>Jacobs et al. (1976)(^{a})</td>
</tr>
<tr>
<td>Vapour Pressure</td>
<td>Pa</td>
<td>6.9*10^-6 (25°C)</td>
<td>Tittlemier et al. (2002)(^{a})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7.5*10^-4 (liquid, sub-cooled)</td>
<td></td>
</tr>
<tr>
<td>Water solubility</td>
<td>µg/L</td>
<td>11</td>
<td>Tittlemier et al. (2002)(^{a})</td>
</tr>
<tr>
<td>Melting point</td>
<td>ºC</td>
<td>72</td>
<td>A)</td>
</tr>
<tr>
<td>Boiling point</td>
<td></td>
<td>No data</td>
<td></td>
</tr>
<tr>
<td>Log K(_{OW})</td>
<td></td>
<td>6.39</td>
<td>Doucette &amp; Andren (1988)(^{a})</td>
</tr>
<tr>
<td>Log K(_{oc})</td>
<td></td>
<td>3.33-3.87</td>
<td>Calculated(^{a})</td>
</tr>
<tr>
<td>Henry’s Law Constant</td>
<td>Pa m(^3)/mol</td>
<td>3.95*10^-1</td>
<td>Waritz et al. 1977(^{a})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.40*10^-1</td>
<td>Calculated(^{a})</td>
</tr>
</tbody>
</table>

a): Quoted from US ATSDR, 2004

Some of the data for the properties listed in Table 1.1 may not be reliable because products of questionable purity were used by earlier investigators to derive them. Therefore, recent physical and chemical property data that have been reported for hexabromobiphenyl in Tittlemier et al. (2002) (Quoted from US ATSDR, 2004) are included in Table 1.1.

2. Persistence

The EHC review concludes that polybrominated biphenyls (PBBs) are stable and persistent in the environment. The degradation of PBBs by purely abiotic chemical reactions (excluding photochemical reactions) is considered unlikely.

PBBs have been reported to be persistent under field conditions. Soil samples from a former PBB manufacturing site, analysed several years after accidental release, still contained PBBs. However, the congener composition was different, indicating partial degradation of the PBB residue in the soil samples. According to the EHC Review, follow-up surveys over a three-year period following the termination of PBB production showed no significant decline in PBB levels in sediments from a river. In laboratory investigations, mixtures of PBBs appear to be fairly resistant to microbial degradation.

3. Bioaccumulation

The EHC review states that PBBs are lipophilic and able to bioconcentrate in the food chain. This is also supported by monitoring results from wildlife studies. For example, fathead minnows (Pimephales promelas) caged in a river, where water levels of PBB remained consistently at less than 0.1 µg/litre, concentrated these contaminants in their bodies more than 10 000 fold in two weeks of exposure.

Log Kow: 6.39 - 7
BCF: > 10 000 (fish)

4. Potential for long-range environmental transport
Vapour pressure of hexabromobiphenyl is 6.9 x 10^-9 kPa. There is no information available about measured half-life of hexabromobiphenyl in the air. According to the EHC review, the photoreactivity of 2,2',4,4',5,5'-hexabromobiphenyl was found to be relatively high but on the other hand, the rates and extent of photolytic reactions of PBBs in the environment have not been determined in detail. The few field observations available indicate a high persistence of the original PBBs or a partial degradation to less brominated (and often more toxic) photoproducts. The EHC review concludes that long-range transport of PBBs in the atmosphere has not been proven, but that the presence of these compounds in Arctic seal samples indicates a wide geographical distribution.

5. Adverse effects

Only few data are available on the effects of PBBs on organisms in the environment. No information is available on the effects of PBBs on the ecosystems.

The EHC review concludes that polybrominated biphenyls are extremely persistent in living organisms and have been shown to produce chronic toxic effects and cancer in animals. Though the acute toxicity was low, cancer was induced at a dose of 0.5 mg/kg body weight per day and the no-observed-effect level was 0.15 mg/kg body weight per day. A number of chronic toxic effects have been observed in experimental animals at doses around 1 mg/kg body weight per day following long-term exposure. International Agency for Research on Cancer (IARC) has classified hexabromobiphenyl as a possible human carcinogen (IARC group 2B).

Reasons for concern

Hexabromobiphenyl is very persistent in the environment. It has a great potential for bioaccumulation and in addition it is assumed to have potential for bio-magnification. Due to its physical and chemical properties and based on findings in environmental samples, it can be assumed that hexabromobiphenyl can be transported long distances in air, far from its sources.

Hexabromobiphenyl is a possible human carcinogen and can also be regarded as a substance of endocrine disrupting activity.

Production and use of polybrominated biphenyls has been ceased over the last decades in developed countries but it cannot be excluded that these substances are still produced and used in some developing countries. In addition to emissions during manufacture or use, these substances enter the environment from the widespread use of flame-retarded products. A considerable part of the PBBs produced will probably reach the environment sooner or later because of the high stability of these compounds. Furthermore, some these chemicals may form toxic polybrominated dibenzofurans during combustion processes.

Neither single country nor groups of countries alone can abate the pollution caused by hexabromobiphenyl. Regional action has already been considered necessary and hexabromobiphenyl is totally banned under the CLRTAP Protocol on POPs. Due to the harmful POP properties and risks related to its possible continuing production and use, global action is warranted to eliminate this pollution.
RISK PROFILE

EXECUTIVE SUMMARY

The European Community and its Member States being Parties to the Stockholm Convention proposed hexabromobiphenyl to be listed in the Convention. The Persistent Organic Pollutants Review Committee concluded in its meeting in November 2005 that the substance comply with the screening criteria set out in Annex D of the Convention and that a draft risk profile should be prepared to review the proposal further.

Hexabromobiphenyl belongs to a wider group of polybrominated biphenyls (PBBs). The term “polybrominated biphenyls” or “polybromobiphenyls” refers to a group of brominated hydrocarbons formed by substituting hydrogen with bromine in biphenyl. The hexabromo congeners exist as 42 possible isomeric forms. According to the available data, production and use of hexabromobiphenyl has ceased in most, if not all, countries. However, it is possible that hexabromobiphenyl is still being produced in some countries.

Hexabromobiphenyl has been used as a fire retardant in acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business, machine housings and in industrial and electrical products and in polyurethane foam for auto upholstery. A considerable part of the substance produced will probably reach the environment sooner or later because of the high stability of these compounds. According to available data, hexabromobiphenyl can be considered to be highly persistent in the environment. There is evidence of low or no degradation in water, soil and sediment, in the laboratory as well as in the field.

Hexabromobiphenyl is less volatile than many of the currently listed POP substances. However, extensive data on monitoring shows that it is found throughout the Arctic wildlife, demonstrating that it does have a high potential for long range environmental transport.

With measured weight-based BCF values in the range 4,700 - 18,100 and biomagnification factors in the aquatic food chain exceeding 100, hexabromobiphenyl is considered to be highly bioaccumulative and to have a high potential for biomagnification. These properties are demonstrated by several authors to be comparable to those of hexachlorobiphenyl (a PCB compound), for which the bioaccumulative properties are well documented.

Hexabromobiphenyl is readily absorbed into the body and accumulates following prolonged exposure. Although the acute toxicity of hexabromobiphenyl is low, a number of chronic toxic effects including hepatotoxicity have been observed in experimental animals at doses around 1 mg/kg bw/day following long-term exposure, and effects are seen in the rat thyroid at doses as low as 0.05 mg/kg bw/day. The International Agency for Research on Cancer has classified hexabromobiphenyl as a possible human carcinogen (IARC group 2B). The PBBs are endocrine disrupting chemicals, and effects are seen on reproductive capacity in rats, mink and monkeys. There is epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer in exposed women. Data on toxicity to other species than laboratory mammals is scarce but suggests the environmental toxicity of hexabromobiphenyl is comparable to that of hexachlorobiphenyl.

Based on the available data, it is likely that hexabromobiphenyl can, as result of long-range environmental transport, cause significant adverse effects on human health and/or the environment, such that global action is warranted.

Data sources

This Draft Risk Profile is mainly based on information from the following review reports:

1. STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS


2. SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

2.1. SOURCES

Production

The commercial production of polybrominated biphenyls (PBBs) generally involves bromination of biphenyl, a process involving a much more specific reaction and producing a smaller number of product mixtures than chlorination (Sundstrom et al. 1976a). (Quoted from US ATSDR, 2004)

The process of manufacturing PBBs consists of a Friedel-Crafts type reaction in which biphenyl is reacted with bromine in the presence of chloride in an organic solvent, using aluminium chloride, aluminium bromide, or iron as catalyst (Brinkman & de Kok, 1980). (Quoted from EHC 152 (IPCS, 1994))

Trade and stockpiles

The commercial production of PBBs began in 1970. Approximately 6 million kg of PBBs were produced in the United States from 1970 to 1976. Only three commercial PBB products were manufactured (i.e. hexabromobiphenyl, octabromobiphenyl, and decabromobiphenyl) and these three products were based on a limited number of congeners (Hardy 2002b). Hexabromobiphenyl constituted about 5.4 million kg (ca 88%) and octa- and decabromobiphenyl constituted = 0.68 million kg together of this total (Neufeld et al. 1977). Michigan Chemical Corporation, St. Louis, Michigan, the sole producer of hexabromobiphenyl in the United States, stopped producing this PBB in 1975. (Quoted from US ATSDR, 2004.) Subsequent production of PBBs appears to have been limited to the octa- and decabromobiphenyls.

Production of octa- and decabromobiphenyl continued in the United States until 1979 (IARC 1986; Neufeld et al. 1977). Shortly after the 1973–1974 agriculture contamination accident in Michigan, PBB production in the United States was voluntarily discontinued (Hardy 2000); PBBs are no longer produced in the United States (SRI 2001). Re-initiation of manufacture of PBBs would require approval from the EPA. (Quoted from US ATSDR, 2004)

Two UK companies are reported to have marketed or produced technical-grade decabromobiphenyl in the United Kingdom. In 1977, the production of PBBs in the UK was discontinued. Highly brominated PBBs (Bromkal 80-9D) were produced in Germany until

See http://www.amap.no/
mid-1985, when the activities concerning bromine-based fire retardants were shifted to the USA. No domestic producer has been identified in the Netherlands. In the early nineties, an Israeli company with two bromine plants in the Netherlands denied the production of PBBs. (Modified from EHC 152 (IPCS, 1994)). There is no information available regarding possible use and production of hexabromobiphenyl in Russia.

Until the year 2000, the only PBB in commercial production was decabromobiphenyl, which was manufactured by one company (Atochem) in France (Hardy 2000). (Modified from US ATSDR, 2004) An author (Darnerud, 2003) has stated that with the closure of the decabBB production in France, the PBB production in the world has ceased.

In the United States, PBBs are not known to be imported or exported anymore except possibly in small quantities for laboratory uses. PBBs have not been imported from other countries into the United States, except in finished products (Neufeld et al. 1977). The two companies that manufactured octa- and decabromobiphenyl in the United States between 1976 (0.805 million pounds) and 1978 exported all of their products to Europe (Neufeld et al. 1977). (Quoted from US ATSDR, 2004)

EXIDIM, the European Database on the Export Import of Dangerous Chemicals under the Rotterdam Convention has registered a total of 6 export applications for PBBs (which do not however include hexabromobiphenyl) in the years 2003 – 2006 (1 in 2003 and 2004, 2 each in 2005 and 2006). No imports of PBBs to the European Unions are registered in this period.

Information received by 27 January 2006 as a result of the request for information from Stockholm Convention Parties and observers, included response from Brazil, Australia, Japan, Republic of Lebanon and the USA, all stating that there is no production or use of hexabromobiphenyl in these countries.

In summary, according to the information available, production and use of hexabromobiphenyl has ceased in most, if not all, countries. However, it is possible that hexabromobiphenyl is still being produced in some developing countries or in countries with economies in transition.

Uses
In the United States and Canada, hexabromobiphenyl (FireMaster(R)) was the principal PBB product. It was used as a fire retardant in three main commercial products: acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business machine housings and in industrial (e.g. motor housing), and electrical (e.g. radio and TV parts) products: as a fire retardant in coatings and lacquers, and in polyurethane foam for auto upholstery (Neufeld et al. 1977) (Modified from EHC 152 (IPCS, 1994) and US ATSDR, 2004).

Of the estimated 2,200 tonnes hexabromobiphenyl produced in 1974 (IARC, 1978), about 900 tonnes (Mumma & Wallace, 1975; Neufeld et al., 1977; IARC, 1978) were used in ABS plastic products and about 34,000 tonnes (Mumma & Wallace, 1975; Neufeld et al., 1977; IARC, 1978) in cable coatings. The exact quantity of FireMaster(R) used in polyurethane foam for automobile upholstery was not published. The two larger consumers ceased using hexabromobiphenyl (one of these in 1972) because PBBs did not decompose in the ultimate incineration of scrapped automobiles (Neufeld et al., 1977). (Quoted from EHC 152 (IPCS, 1994))

In the EHC 152 (IPCS, 1994), it is stated that at the time, no users of hexabromobiphenyl had been identified (Neufeld et al., 1977; Di Carlo et al., 1978; Brinkman & de Kok, 1980). (Quoted from EHC 152 (IPCS, 1994))

Releases to the environment
Data for loss into the environment during normal production are published only for the United States. The following information refers to reviews by Neufeld et al. (1977) and Di Carlo et al. (1978). Losses of PBBs to the environment at sites of its manufacture can total 51 kg/1000 kg of product. These losses occur through:
1) **Emission into the air:** In 1977, the maximum air losses as particulate matter at production sites were estimated to total 1.1 kg of PBBs/1000 kg manufactured.

2) **Losses in waste waters** resulting from the quenching and washing of the PBBs as they were recovered from the reaction mass. The losses of PBBs to sewers at manufacturing sites were estimated, in 1977, to be 4.6 µg/kg of product.

3) **Solid losses to landfills** resulting from drying, handling, shipping and transportation. An estimate of PBB losses as solid waste to landfills was 50 g/kg of product.

4) **Losses to the soil**
   Soil samples from the bagging and loading areas of the Michigan Chemical Corp. contained PBBs at concentrations of 3500 and 2500 mg/kg, respectively. (Abbreviated from EHC 152 (IPCS, 1994))

In 1973, an accidental release of PBBs occurred in Michigan (referred to as the "Michigan disaster" in EHC 152), when two products manufactured by the Michigan Chemical Company were inadvertently confused and 250-500 kg (Di Carlo et al., 1978) of FireMaster(R), instead of NutriMaster(R), a magnesium oxide-based cattle feed supplement, were added to animal feed and distributed to farms within the state. The compound is believed to have been FireMaster(R) FF-1 (e.g., Fries, 1985b), even if in some publications the name FireMaster(R) BP-6 is used (e.g., Neufeld et al., 1977; Di Carlo et al., 1978). This accidental mix up resulted in widespread contamination by PBBs. Chronological reports or reviews of the PBB disaster are given by Carter (1976), Getty et al. (1977), Kay (1977), Di Carlo et al. (1978), Damstra et al. (1982), Zabik (1982), and Fries (1985b). (Quoted from EHC 152 (IPCS, 1994))

Approximately 5350 tonnes of hexabromobiphenyl were used in commercial and consumer products in the United States, most in the production of plastic products with an estimated use life of 5–10 years (Neufeld et al. 1977). Since the cessation of production, all of these products, such as TV cabinet and business machine housings, are expected to have been disposed of by land filling or incineration (Neufeld et al. 1977). (Quoted from US ATSDR, 2004)

In conclusion, hexabromobiphenyl can enter the environment from the widespread use of flame-retarded products. A considerable part of the substance produced will probably reach the environment sooner or later because of the high stability of these compounds. Furthermore, some of these chemicals may form toxic polybrominated dibenzofurans during combustion processes.

### 2.2. ENVIRONMENTAL FATE

**PERSISTENCE**

The EHC review (1994) concludes that polybrominated biphenyls are stable and persistent in the environment. The degradation of PBBs by purely abiotic chemical reactions (excluding photochemical reactions) is considered unlikely.

In air, the two processes that may result in significant degradation or transformation of PBBs are photo-oxidation by hydroxyl (OH) radicals and direct photolysis. Based on a structure-activity relationship for the estimation of half-lives for the gas phase reactions of hydroxyl radicals with organic compounds (Atkinson 1987b), the estimated half-life of hexabromobiphenyl due to reaction with OH radicals is 182 days. The importance of the photochemical reaction under sunlight illumination conditions for the degradation/transformation of PBBs in air cannot be evaluated due to the lack of information. (Abbreviated from US ATSDR, 2004)

The EHC 152 (IPCS, 1994) refers to laboratory experiments in methanol, showing rapid photodegradation of 2,2',4,4',5,5'-hexabromobiphenyl (90% degradation after 9 minutes) and resulting in mainly lower brominated PBBs. However, in the US ATSDR
(2004), it is questioned whether this photolysis could take place in water due to the lack of active groups. Therefore it is questionable whether hexabromobiphenyl can be degraded rapidly in air.

Biodegradation in water under aerobic conditions is low, although the lower substituted biphenyls might biodegrade in aerobic water and sediment (Kong and Sayler 1983; Sugiura 1992; Yagi and Sudo 1980), the higher substituted biphenyls are resistant to aerobic biodegradation (Kawasaki 1980; Sasaki 1978; Shelton and Tiedje 1981) (quoted from US ATSDR, 2004). This is further supported by the measurement (by GC) of negligible biodegradation of hexabromobiphenyl in a four week ready biodegradability test (OECD TG 301C), resulting in 4% reduction in total concentration as measured by GC (Governmental Japanese database NITE, 2006). The latter result implies that the degradation half-life in water is > 2 months.

Under anaerobic conditions, it has been shown that microorganisms in river sediments obtained from populated areas can biodegrade higher substituted PBBs, including FireMaster mixtures (Morris et al. 1992) to form lower brominated products (quoted from US ATSDR, 2004). However, the potential of sediment microflora from remote areas has not been investigated, so it cannot be evaluated whether anaerobic debromination may be a considerable cause for degradation under anaerobic conditions.

PBBs have been reported to be persistent under field conditions. The information on the fate of PBBs in soil is limited. Soil samples from a former PBB manufacturing site, analysed several years after accidental release, still contained PBBs. However, the congener composition differed from the original PBB mixture, indicating partial degradation of the PBB residue in the soil samples. According to the 1994 EHC Review, follow-up surveys over a three-year period following the termination of PBB production showed no significant decline in PBB levels in sediments from a river. In laboratory investigations, mixtures of PBBs appear to be fairly resistant to microbial degradation. (Quoted from EHC 152 (IPCS, 1994)) This implies that the degradation half-life in soil and sediment is > 6 months.

The US ATSDR (2004) refers to studies in soils with high levels of FireMaster, in which degradation of hexabromobiphenyl was “significant” during a period of several years but it was not complete. However in other soils, in which the concentrations were lower, or to which manure was added, degradation was even slower. The degradation was attributed to photodegradation even if this process will only take place at the soil surface (US ATSDR, 2004).

Conclusion
In spite of photodegradation in methanol, it is questionable whether hexabromobiphenyl can be degraded rapidly in air. There is evidence of low or no degradation in water (DT$_{50} > 2$ months), soil and sediment (DT$_{50} > 6$ months) in the laboratory as well as in the field. Therefore, hexabromobiphenyl is considered to be highly persistent.

BIOACCUCLMULATION
The EHC review states that PBBs are lipophilic and able to bioconcentrate in the food chain. This is also supported by monitoring results from wildlife studies. For example, fathead minnows (Pimephales promelas) caged in a river where water levels of PBB remained consistently at less than 0.1 µg/l concentrated these contaminants in their bodies more than 10,000 fold in two weeks of exposure (EHC 152 (IPCS, 1994)).

As expected from their high lipophilicity, PBBs show a marked tendency to accumulate in animals. However, data are available only on single links of food chains. It has been reported that similar compounds, e.g. PCBs, which are more widely spread in the environment, may have bioconcentration factors of 3-4 orders of magnitude between water and fish, with a further 1-2 orders of magnitude between
whole fish and the fat storage tissues of fish predators, such as cormorant, heron, and seal (Pearson, 1982). (Quoted from EHC 152 (IPCS, 1994))

This has been further supported in recent literature. Thus, US ATSDR, (2004) states that PBBs may also be transported from water to aquatic organisms in which bioconcentration may take place. Data from different laboratories on the bioconcentration of PBBs in fish show wide variation. The experimentally determined bioconcentration factor (BCF) for hexabromobiphenyl (mixtures of unspecified congeners) in the whole body of fathead minnows (*Pimephales promelas*) was 18,100 in a 32-day exposure (Veith *et al.* 1979). In fillet of fathead minnow, the estimated BCF was >10,000 (Hesse and Powers 1978). The lipid weight-based BCF value of 2,2',4,4',6,6'-hexabromobiphenyl in guppies (*Poecilia reticulata*) was 708,000 (Gobas *et al.* 1989) (abbreviated from US ATSDR, 2004). Weight-based BCF values in the range 4,700-16,000 were recorded in a 60 days test with the carp *Cyprinus carpio* and concentrations of hexabromobiphenyl of 0.1 - 1 µg/L (Governmental Japanese database NITE, 2006).

Furthermore, a potential for biomagnification has been demonstrated by Jansson *et al.* (1993), who found a biomagnification factor (BMF) for 2,2',4,4',5,5'-hexabromobiphenyl (PBB congener 153) of about 175 comparing lipid-based concentrations in prey (herring) and predator (Baltic seal). This BMF was at the same level as that of the PCB congener 153. These findings were supported by Vorkamp *et al.* (2004), who found lipid-based concentrations of hexabromobiphenyl (PBB 153) in polar bear to be a factor of about 100 higher than in ringed seal from East Greenland. They conclude further, that the PBBs (and PBDEs) seem to biomagnify along the marine food chain in a manner similar to PCBs and that PBBs show indications of a higher biomagnification potential than PBDEs (Vorkamp *et al.*, 2004).

**Conclusion**

With measured weight-based BCF values in the range 4,700 - 18,100 (most of which exceed 5,000) and biomagnification factors in the aquatic food chain exceeding 100, hexabromobiphenyl is considered to be highly bioaccumulative and to have a high potential for biomagnification. These properties are demonstrated by several authors to be comparable to those of hexachlorobiphenyl, for which the bioaccumulative properties are well documented. Evidence appears to be satisfactory to conclude high bioconcentration and biomagnification.

**POTENTIAL FOR LONG RANGE ENVIRONMENTAL TRANSPORT**

The partitioning of hexabromobiphenyl in the environment will be governed by its high log *K*\textsubscript{ow} (6.39) and low water solubility (3 µg/L) resulting in sorption to particulate matter (dust, soil and sediment) and organic material (including living organisms). Furthermore, the combination of these properties and the relatively low vapour pressure (6.9*10\textsuperscript{-6} to 7.5*10\textsuperscript{-4} Pa) of hexabromobiphenyl, results in a low potential for volatilisation. The latter is specified in US ATSDR (2004) as follows: Based on an estimated Henry’s law constant of 3.95*10\textsuperscript{-1} Pa m\textsuperscript{3}/mol (where Henry’s law constant = vapor pressure/water solubility) and an estimation method (Thomas 1990), the estimated volatilization half-life of hexabromobiphenyl is 23 days. Therefore, the transport of PBBs from water to the atmosphere by volatilization is not expected to be important.

The assessment of the potential for long-range transport of hexabromobiphenyl could be done by comparing the properties of hexabromobiphenyl to those of the currently listed POPs. As a starting point for the assessment of hexabromobiphenyl, the highest and lowest of the values in Table 1.1 were used (for vapour pressure, only the value at 25 °C) and, for comparison, the information on the UNEP-POPs homepage. Among the currently listed POPs, most of the relevant properties were available for aldrin, chlordane, dieldrin, DDT, hexachlorobenzene, mirex, toxaphene, endrin and heptachlor. Missing information (water solubility of mirex) was sought in

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43 These investigations are part of the Arctic Monitoring and Assessment Programme (AMAP).
US ATSDR (1995), so as not to introduce what seems to be an outlier in the comparison by using the value of \(6.5 \times 10^{-5}\) mg/L from AMAP (2004).

The water solubility and vapour pressure as well as Henry’s Law Constants calculated from these values of the currently listed POPs are summarised in Table 2.1 together with information on hexabromobiphenyl from Table 2.1.

Table 2.1 Water solubility (WS), vapour pressure (VP) and (calculated) Henry’s Law Constant (HLC) (at 25 °C) for hexabromobiphenyl and currently listed POPs.

<table>
<thead>
<tr>
<th>Substance</th>
<th>WS mg/L</th>
<th>VP Pa</th>
<th>HLC Pa m^3/mol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hexabromobiphenyl-min</td>
<td>0.011</td>
<td>(6.9 \times 10^{-6})</td>
<td>0.39</td>
</tr>
<tr>
<td>Hexabromobiphenyl-max</td>
<td>0.003</td>
<td>(6.9 \times 10^{-6})</td>
<td>1.44</td>
</tr>
<tr>
<td>POP-min (DDT)</td>
<td>0.0012</td>
<td>(2.5 \times 10^{-5}) (DDT)</td>
<td>0.04 (endrin)</td>
</tr>
<tr>
<td>POP-max (toxaphene)</td>
<td>3.0</td>
<td>27 (toxaphene)</td>
<td>3726 (toxaphene)</td>
</tr>
<tr>
<td>POP-2^nd max (dieldrin)</td>
<td>0.5</td>
<td>0.04 (heptachlor)</td>
<td>267 (heptachlor)</td>
</tr>
</tbody>
</table>

Table 2.1 shows that the water solubility of hexabromobiphenyl is at the level of the least water soluble among the currently listed POPs (DDT), while the vapour pressure of HBB is one order of magnitude lower than that of DDT. The two Henry’s Law Constants calculated for hexabromobiphenyl are well inside the range marked by the currently listed POPs, being at least one order of magnitude higher than the lowest (endrin).

Based on the vapour pressure alone, the potential for long-range airborne transport of hexabromobiphenyl is low compared to most of the currently listed POPs, while a comparison of the Henry’s Law Constants places hexabromobiphenyl in a position close to endrin.

The EHC 152 (1994) argues that the vapour pressure of hexabromobiphenyl is \(6.9 \times 10^{-6}\) Pa and, thereby the potential for volatilisation is low. There is no information available about measured half-life of hexabromobiphenyl in the atmosphere. In the laboratory photodegradation of 2,2’,4,4’,5,5’-hexabromobiphenyl was rapid (90% degradation after 9 minutes) mainly resulting in lower brominated PBBs (EHC 152 (IPCS, 1994)). On the other hand, the rates and extent of photolytic reactions of PBBs in the environment have not been determined in detail. The few field observations available indicate a high persistence of the original PBBs or a partial degradation to less brominated, and often more toxic, photoproducts.

In support of the assessment of the potential for long-range environmental transport, monitoring data demonstrate that this substance has managed to reach remote areas like the Barents Sea and Greenland. In the Arctic, hexabromobiphenyl has been measured in samples of animals in several investigations. The results are summarised in Annex A, Table A.1.

In whitefish from Lapland (North Scandinavia) and ringed seal from Svalbard, concentrations of 0.29 and 0.42 µg/kg lipid, respectively, were reported by Jansson et al. (1993). In another paper, Jansson et al. (1987) reported concentrations of hexabromobiphenyl (Firemaster BP-6) in ringed seal from Svalbard to be 4 µg/kg lipid and concentrations in guillemot muscle of 50 µg/kg lipid. It is not clear whether these results are from different investigations. For comparison, Krüger (1988) measured 0.8 µg/kg of PBB 153 in unspecified seal samples from the same area (Quoted from US ATSDR, 2004).

In samples of large char collected in 1999-2001 from one of two lakes in Bear Island in the Barents Sea, Evenset et al. (2005) measured concentrations of 4.11-51.5 µg/kg lipid of hexabromobiphenyl (PBB 153). These levels are the same as or higher than levels of PBB 153 (0.2-9.4 µg/kg lipid) in lake trout sampled in 1997 from Lakes Ontario, Erie, Huron and Superior, which were measured by Luross et al. (2002) (Table 2.2).
Vorkamp et al. (2004) measured concentrations of PBDEs in samples from Greenland and the Faroe Islands of sediment and seven species of animals representing different trophic levels of the food chain. As a pilot investigation, analyses for five PBBs including PBB 153 were made in selected samples of blubber or fat from ringed seal, minke whale and polar bear from Greenland as well as pilot whale and fulmar from the Faroe Islands. PBBs were detected in all samples, except sediment samples, shorthorn sculpin samples and samples of ringed seal from West Greenland. In all other samples, PBB 153 was generally the dominant congener. The concentrations measured in samples from (East) Greenland were in the range 0.34 – 44.26 µg/kg lipid with the lowest values found in the seal and the highest in polar bear. In the Faroese samples, the range of concentrations of PBB 153 was 8.71 – 25.54 µg/kg lipid weight with the highest values found in fulmar, a fish predator (Vorkamp et al., 2004).

For comparison, concentrations of PBB 153 in grey seal and osprey from the Baltic Sea were 26 and 22 µg/kg lipid weight; respectively (Jansson et al., 1993). Thus, concentrations of PBB 153 as µg/kg lipid weight in seals from the Arctic (0.34-0.74) are considerably lower than in seals from the Baltic Sea (26 µg/kg lipid weight), while concentrations in predatory birds from the two areas (fulmar and osprey) are of the same order of magnitude, being 25 and 22 µg/kg lipid weight; respectively.

Vorkamp et al. (2004) conclude that PBBs and PBDEs seem to biomagnify along the marine food chain in a similar manner to PCBs. PBBs show indications of a higher biomagnification potential than PBDEs. Even though their absolute concentrations are lower than those of PBDEs, the PBDE/PBB ratio increases in the order ringed seal<pilot whale<minke whale<fulmar<polar bear, leading to almost equal concentrations of PBDEs and PBBs in polar bear. Apparently, the compounds follow the same spatial trend as previously observed for organochlorine compounds, with higher concentrations in East Greenland than in West Greenland (Vorkamp et al., 2004). This indicates that the long-range transport of hexabromobiphenyl may be slow.

Monitoring information on PBBs from areas outside the Arctic, Northern Europe and America is scarce, as only one reference has been found. Hexabromobiphenyl (PBB 153) was not detected (LOD between 0.02 and 0.1 µg/kg wet weight) in samples of muscle and liver from several species of fish from the eastern Mediterranean region of Turkey (Erdogrul et al., 2005).

In summary, the 1994 EHC review concludes that long-range transport of PBBs in the atmosphere has not been proven, but that the presence of these compounds in Arctic seal samples indicates a wide geographical distribution (EHC 152 (IPCS, 1994)). Several authors report levels of hexabromobiphenyl (and other brominated biphenyls) in arctic animals, especially in fish eating predators and predators at higher trophic levels.

In a recent modelling study, Scheringer et al. (2006) investigated the persistence and long range transport potential of four potential POPs, including chlordecone and hexabromobiphenyl. They concluded that these POP candidates have persistence and long range transport potential properties similar to those of several known POPs. Furthermore, they included the uncertainty regarding the data quality in a Monte Carlo analysis, which indicated that the result is valid although there are considerable uncertainties in the chemical properties of the four POP candidates.

**Conclusion**

Although hexabromobiphenyl is less volatile than any of the currently listed POPs, it is found throughout the Arctic wildlife, demonstrating that it does have a high potential for long range environmental transport. The potential for long range environmental transport of hexabromobiphenyl is further supported by the modelling study of Scheringer et al. 2006.
2.3. EXPOSURE

Because production of hexabromobiphenyl is assumed to have ceased (section 2.1.2) the assessment of the exposure will focus on general exposure instead of local production sites.

Concentrations in abiotic environmental media

Recent monitoring data in soil, water and sediments for PBBs are limited. Historical monitoring data from the United States indicate that environmental PBB concentrations are confined to areas near former manufacturing facilities and regions of Michigan affected by the farm accident of the early 1970's (see Section 2.2.3). (US ATSDR, 2004)

The only available data for environmental concentrations of PBBs in areas outside the vicinity of former production sites are those from sediment samples from Greenland (Vorkamp et al., 2004), where PBBs (including PBB 153) were not detected in any sample (the limits of detection/quantification are, however, not well defined in the paper).

Concentrations in biota

In the vicinity of Michigan

Concentrations in biota in the vicinity of the Michigan production and contamination accident sites were measured in a multitude of samples during the decade following the cessation of production. The US ATSDR (2004) includes the following: In the late 1980's, PBBs were detected in the concentration range of 15–15,000 µg/kg (lipid basis) in fish from embayments and tributaries of Lake Huron, but not from Lake Superior. Recently, Luross et al. (2002) determined the concentrations of several PBB congeners in lake trout from Lakes Huron, Superior, Erie, and Ontario. 2,2',4,4',5,5'-Hexabromobiphenyl (PBB-153) and 2,2',4,5,5'-pentabromobiphenyl (PBB-101) were found at the highest levels at concentrations ranging from 0.189 to 2.083 µg/kg wet weight and from 0.042 to 0.633 µg/kg wet weight, respectively. Several other congeners were also detected in these lake trout samples (Quoted from US ATSDR, 2004). The concentrations of PBBs in eggs of fish-eating birds (common tern, little gull, herring gull, and red-breasted mergansers) collected during 1975–1980 from nesting islands in northwestern Lake Michigan and Green Bay contained PBBs in the concentration range of 0.02–0.25 mg/kg (µg/g) wet weight (Heinz et al. 1983, 1985) (quoted from the US ATSDR, 2004).

Other areas

Monitoring data from areas outside the Arctic (see chapter 2.2.3) and the most exposed region of the US are summarised in Table A.2. in Annex A.

EHC 152 (1994) includes the following investigations on residues of (hexa)bromobiphenyl in biota:

- In Europe, 2,2',4,4',5,5'-hexabromobiphenyl (PBB 153) was found in fish from German and Swedish rivers at concentrations ranging from 0.3 to 0.6 µg/kg lipid (Krüger, 1988; Jansson et al., 1992). A trout sample from a breeding farm contained much lower levels of PBBs than the fish samples from the rivers (Krüger, 1988).
- Swedish reindeers (pooled samples) showed PBB 153 levels as low as 0.04 µg/kg lipid (Jansson et al., 1992).
- PBBs (as a group) were not found in otters (Lutra canadensis) from a region relatively remote from industrial sites in north eastern Alberta (Canada) (Somers et al., 1987).
- Fish samples (freshwater and marine species) collected in 1983 from an industrial area of Japan (Osaka) did not contain "PBBs" (not specified) (Watanabe & Tatsukawa, 1990).
- In Europe, PBBs have been detected in seals (Phoca vitulina; Pusa hispida), guillemots (Uria aalge; U. lomvi), and white-tailed sea eagles (Haliaeetus albicilla). The concentrations (estimated by comparison with the technical product Firemaster...
BP-6) ranged from 3 to 280 µg/kg lipid (Jansson et al., 1987). The concentrations of PBBs in comparable samples from the Baltic Ocean were all higher than concentrations in samples from the Arctic Ocean. The same was true for polybrominated biphenyl ethers and PCBs (Jansson et al., 1987).

- Concentrations of PBB 153 determined in marine fish ranged from 0.2 to 2.4 µg/kg lipid (Krüger, 1988; Jansson et al., 1992). PBB 153 levels of 0.4-26 µg/kg lipid were found in seals (Krüger, 1988; Jansson et al., 1992).

- Detailed isomer-specific PBB analyses were carried out by Krüger (1988) in fish (several species) from the Baltic and North Seas and from sections of the Lippe and Rur rivers in North Rhine-Westphalia, Germany. Seal samples from Spitsbergen (Norway) were also included in this investigation. All samples contained PBBs. The smallest number of PBB congeners was found in seals (n = 5) from an area remote from industrial sites. The main components were different hexabrominated isomers with 2,2',4,4',5,5'-hexabromobiphenyl reaching a mean concentration of 0.8 µg/kg fat. The mean concentrations of several PBB congeners and isomers (penta- to nonabrominated biphenyls) measured in fish (n = 35) ranged, mostly, between 0.01 and 2 µg/kg fat. The pattern of PBB congeners found in fish differed in a characteristic manner, depending on the different capture sites. While relatively high amounts of nona- and octabromobiphenyls (besides polybrominated biphenyl ethers) were present in fish from German rivers (n = 17; several species), hexabrominated biphenyls were predominant in fish from the North Sea and the Baltic Sea (n = 17; several species). In all samples from the Baltic Sea (n = 6), 3,3',4,4',5,5'-hexabromobiphenyl was found in relatively high concentrations (maximum concentration: 36 µg/kg fat), but it was not detected in samples from the North Sea and from rivers. The concentrations of the other hexabrominated biphenyls were mostly higher in fish from the Baltic Sea than in fish from the North Sea. (Quoted from EHC 152 (IPCS, 1994))

US ATSDR (2004) supplements with:

- Three bottlenose dolphins (Tursiops truncatus) collected during 1987–1988 from the U.S. mid-Atlantic contained PBBs at concentrations of 14–20 µg/kg lipid basis (Kuehl et al. 1991). The source of the PBBs in the dolphins was not given.

- The median concentrations of PBBs in carcass and brain of 10 specimens of bald eagles (Haliaeetus leucocephalus) collected from 29 states in 1977 were 0.07 and 0.05 mg/kg (µg/g), respectively (Kaiser et al. 1980). Twenty-two other specimens did not contain detectable levels (<0.03 mg/kg [µg/g]) of PBBs.

- In whitebeaked dolphins from the North Sea, the concentration of hexa-, penta-, and deca-BBs were 13, 8.3, and <0.9 µg/kg (µg/kg) wet weight, respectively. Tetra-, penta-, and deca-BBs concentration ranges were 1.1–1.9, 0.4–0.9, and <0.5 µg/kg wet weight, respectively, in sperm whales from the Atlantic Ocean (de Boer et al. 1999).

The German Baltic fish samples (as the only samples in that investigation) also contained PBB 169 at a concentration of 15.16 µg/kg lipid (EHC 152 (IPCS, 1994)).

In the Belgian samples from corpses of birds of prey, the variation in concentrations of hexabromobiphenyl was high. Thus, the maximum concentrations measured in muscle and liver were 150 and 180 µg/kg lipid; respectively (Jaspers et al. 2006).

Jansson et al. (1993) measured hexabromobiphenyl (PBB 153) in samples of reindeer (a herbivore) from northern Sweden at a level of 0.037 µg/kg lipid. In two other herbivores (rabbit and moose) from Southern Sweden, PBBs were not detectable (level of detection not well defined).

Concentrations in human tissue and breast milk

**Michigan**

The human exposure to hexabromobiphenyl subsequent to the Michigan accident is discussed in EHC 152 (1994) as well as in US ATSDR (2004). The general trends of the findings are described as follows in EHC 152 (1994):
Nearly 100% of the adipose samples randomly selected throughout the state had detectable PBB concentrations. Thus, statewide exposure of Michigan residents to PBBs can be demonstrated.

Levels of PBBs in serum (Landrigan, 1980; Wolff et al., 1982), breast-milk (Brilliant et al., 1978; Miller et al., 1984), and adipose tissue (Wolff et al., 1982) were highest in the area of the accident (lower peninsula), and lowest in the upper peninsula, farthest from the source.

Compared with residents of quarantined farms, direct consumers of products from quarantined farms, and PBB production workers, the tissue burdens among the general population of Michigan were 1-3 orders of magnitude lower. Moreover, for example, only 36% of the general population had serum PBB concentrations greater than 1 µg/L, compared with 78% among farmers (Anderson et al., 1979; Wolff et al., 1982).

PBB levels appear to be higher in males than females (Meester & McCoy, 1976; Landrigan et al., 1979; Landrigan, 1980; Wolff et al., 1978; 1980; Kreiss et al., 1982; Eyster et al., 1983) and higher in children (below the age of 10 years) than in adults (Humphrey & Hayner, 1975; Landrigan et al., 1979; Landrigan, 1980; Barr, 1980; Wolff et al., 1982). (Quoted from EHS 152 (IPCS, 1994))

The subsequent development is described in EHC 152 (1994):

In most cases, PBB concentrations did not appear to be decreasing significantly over time. Wolff et al. (1979b) did not find any significant variation in the serum PBB levels of nine dairy farm residents during 18 month of observation.

Paired serum samples, one collected in 1974 and the other in 1977, were also available for 148 members of the Michigan PBB cohort. The data indicate that levels were generally stable over the 3-year period with a mean change of 16 µg/litre (Landrigan et al., 1979). In another study of the Michigan PBB-cohort, the decrements in median serum levels of PBBs between matched pairs over one (1977-78) and two (1977-79) year intervals were both only 1 µg/litre (Kreiss et al., 1982). No significant change in blood plasma PBB levels was observed over a 5-month period in 41 residents of quarantined farms (Humphrey & Hayner, 1975). In contrast, Meester & McCoy (1976) reported a marked decline over 3 years (1974-76) in serum levels of PBBs. These authors also found that the average decrease in PBB concentrations in the fat of 16 individuals was about 40% in a period of 6 months. No changes in PBB levels were seen over an 11-year period (1976-87) in fat samples from a patient with long-term exposure to PBBs from the early 1970s as a result of the Michigan PBBs accident. The average fat level of PBBs was 0.8 mg/kg (Sherman, 1991).

In 1981, PBBs were found in 13-21% of serum samples from 4-year-old Michigan children. Their mothers belonged to a group that was surveyed either with regard to the consumption of Lake Michigan sport fish (mean PBB level detected in children: 2.4 ng/ml) or with regard to former exposure to quarantined farm products (mean PBB level detected in children: 3.0 ng/ml) (Jacobson et al., 1989). (Quoted from EHC 152 (IPCS, 1994))

Other areas
The EHC 152 (1994) stresses the lack of available monitoring studies from areas outside Michigan, as few human monitoring data are available for the US population outside of Michigan. One study deals with the population in the vicinity of industrial areas involved in PBB production or use (Stratton & Whitlock, 1979), the other with farmers of the state of Wisconsin who were examined as control group in connection with the Michigan PBB studies (Wolff et al., 1978).

PBBs were found in all studies, but, because of the limited data, the significance is unclear. The highest PBB levels were found in the hair of humans living near PBB industry. Of the nine samples analysed, five had detectable PBB levels. Both male and female hair samples contained PBBs (Stratton & Whitlock, 1979).
There is very little human monitoring data on PBBs in the populations of countries other than the United States. Krüger et al. (1988) reported PBB contamination of breast-milk from women in Europe in a survey from North Rhine-Westphalia, Germany. The milk samples (n = 25) contained a typical pattern of certain PBB congeners. It included penta- to octabromobiphenyls in concentrations ranging from 0.002 to 28 µg/kg, based on milk fat. The most abundant component was 2,2',4,4',5,5'-hexabromobiphenyl (PBB 153) followed by a peak consisting of two heptabromobiphenyl isomers (2,2',3,4,4',5,5',6- and 2,2',3,4,4',5,6'-heptabromobiphenyl, PBB 187 and 182 respectively). Differences in the pattern were only found in the milk given by a Chinese woman and in that given by a woman having been exposed to several fires in industry.

Concentrations of PBB 153 in human and cow's milk, both collected from the same region (North Rhine-Westphalia), were 1 µg/kg and 0.03 µg/kg, respectively, measured on a lipid basis (Krüger, 1988). (Quoted from EHC 152 (IPCS, 1994))

**Human exposure**

The US ATSDR (2004) considers the current human exposure to PBBs to be very low, because PBBs are no longer produced or used. Thus, the general population exposure to PBBs will only be from historical releases. For people residing in the lower peninsula of Michigan, especially in the immediate vicinity of the PBB contaminated areas of this region, exposure to PBBs may still be occurring today. However, environmental levels have decreased since the 1970s and current exposure, if any, will be at low levels. For other regions of the United States, the levels of exposure will either be very low or none. (Quoted from US ATSDR, 2004)

In Arctic and North Atlantic regions, where the traditional diet includes top predators (e.g. seal in Greenland and pilot whale in the Faroe Islands), exposure has not ceased. Especially the level of PBBs in pilot whale blubber of up to 17 µg/kg lipid indicate the presence of hexabromobiphenyl in food. Pilot whale blubber is consumed as a delicacy in the Faroe Islands.

**Hazard assessment for endpoints of concern**

**Toxicity**

As described in Section 1.1.1, the descriptor “hexabromobiphenyl” covers 42 different hexabrominated biphenyls or congeners, as individually listed in Annex B. The EHC review (IPCS, 1994) indicates that the hexabrominated biphenyls are the most toxic of the chemical class of polybrominated biphenyls (PBBs) and that the higher homologues (hepta-, octa-, nona- and decabrominated biphenyls) are of progressively lower toxicity. Toxicological studies on hexabromobiphenyl have been carried out mainly on the congener 2,2',4,4',5,5'-hexabromobiphenyl (PBB 153), which is the major component of the PBB mixture FireMaster® and on FireMaster® itself. The toxicity of FireMaster® appears to be primarily associated with the minor components 2,3,3',4,4',5-hexabromobiphenyl, 2,3',4,4',5,5'-hexabromobiphenyl, 3,3',4,4',5,5'-hexabromobiphenyl (PBB 169) and 2,3',4,4',5-pentabromobiphenyl (IPCS, 1994). The predominant congeners in FireMaster® (2,2',4,4',5,5'-hexabromobiphenyl and 2,2',3,4,4',5,5'-heptabromobiphenyl), are less toxic (IPCS, 1994). Other toxic contaminants in technical PBB mixtures include the polybrominated naphthalenes (HBNs). Hexabromonaphthalene has been identified as a toxic contaminant of Firemaster BP-6 or FF-1 at levels of approximately 150 ppm (Birnbaum et al. 1983, as reported in US ATSDR, 2004 ). The toxicological effects of the PBBs in humans and in animal studies, as described in the scientific literature, are considered to be attributable mainly to exposure to hexabromobiphenyl congeners (EHC 152 (IPCS, 1994) and US ATSDR, 2004), although a possible contribution of the HBNs to toxicity cannot be ignored.

**Mechanism of action**
Hexabromobiphenyl, in common with all PBBs, is a potent inducer of hepatic cytochrome P-450 metabolising enzymes in the liver. The mechanism of action underlying a number of the toxicological effects of some of these compounds, including induction of metabolising enzymes, immunotoxicity, hepatotoxicity and reproductive toxicity, is considered to be due to interaction with the cellular Ah receptor (also the target of the polychlorinated dioxins, furans and dioxin-like PCBs), causing altered gene expression (Poland & Glover, 1977, 1980; Poland et al., 1979; Goldstein, 1980; Moore et al., 1980; McKinney & Singh, 1981; Parkinson & Safe, 1981; Bandiera et al., 1982, 1983; McKinney & McConnell, 1982; Nebert et al., 1982; Poland & Knutson, 1982; Robertson et al., 1982b, 1984c,d; Safe et al., 1982, 1985; Aust et al., 1983; Dannan et al., 1983; Lai, 1984; Safe, 1984, as quoted in IPCS, 1994).

Toxicokinetics
Hexabromobiphenyl is readily absorbed into the body, the primary route of human exposure being via food, due to accumulation and biomagnification in the food chain (IPCS, 1994; US ATSDR, 2004). The majority of animal toxicology studies have used the oral route of exposure and little information is available on exposure via the inhalation and dermal routes, although worker exposure is likely to occur mainly via these routes (Wolff et al., 1979a, as quoted in IPCS, 1994). Following absorption, hexabromobiphenyl is widely distributed in the body and accumulates, with the highest concentrations found in adipose tissue and to a lesser extent the liver (IPCS, 1994).

Exposure in utero occurs via transfer of PBBs to offspring by placental transfer and infants are also exposed via milk. Human milk has been found to contain levels of 2,2',4,4',5,5'-hexabromobiphenyl 100 times higher than those found in maternal blood (Brilliant et al., 1978; Landrigan et al. 1979; Eyster, 1983, as reported in IPCS, 1994).

Metabolism and excretion of the hexabromobiphenyls is low (IPCS, 1994; US ATSDR, 2004), and the compounds therefore show marked bioaccumulation and persistence in all species. Average half-lives for 2,2',4,4',5,5'-hexabromobiphenyl in humans have been estimated to be between 8 and 12 years (IPCS, 1994), while shorter half-lives have been reported in rats, monkeys, and other species (see Table 68 in IPCS, 1994). It has been suggested that humans may retain certain congeners to a greater degree than experimental animals (e.g. Fries (1985b, as quoted in IPCS, 1994), a phenomenon that is also found with the polychlorinated dioxins and furans.

Toxicity of hexabromobiphenyl in animal studies
In experimental animal studies, hexabromobiphenyl shows relatively low acute toxicity (LD_{50} > 1 g/kg body weight) (see Table 70, IPCS, 1994). Toxicity is higher following repeated exposure (IPCS, 1994), due to progressive accumulation of the compounds and a characteristic delay in lethality after exposure is seen (Di Carlo et al., 1978; Gupta & Moore (1979, as quoted in IPCS, 1994). At lethal doses, death is reported to be due to a "wasting syndrome" with marked loss in body weight rather than to specific organ pathology (Hutzinger et al., 1985a; McConnell, 1985, as quoted in IPCS, 1994). However, prolonged exposure of laboratory animals to doses in the range of < 1 mg/kg bw/day to 100 mg/kg bw/day results in liver, kidney and thyroid changes, accompanied by effects in the nervous and immune systems, porphyria and skin disorders (IPCS, 1994).

A summary of outcomes of a number of the key toxicological studies on hexabromobiphenyl, including the NOAEL/LOAEL derived in each study is provided in Annex A, Table A.3 to this document. The studies included in Annex A, Table A.3 have been selected from the very large database on toxicological studies on hexabromobiphenyl, on the basis of the importance of the endpoint investigated (e.g. reproductive toxicity, carcinogenicity, other key target organ toxicity), robustness of the reported studies and the dose level (NOAEL/LOAEL) at which effects were reported. Table 2.2 below provides information on pivotal toxicological studies (also included in Annex A Table A.3) that provide information on the toxicity of...
hexabromobiphenyl at low levels of exposure, considered to be particularly relevant for characterisation of the toxicological risks of these compounds. Some of these studies have been used by US ATSDR to define Minimal Risk Levels (MRLs) for hexabromobiphenyl (US ATSDR, 2004).

Effects in toxicological studies included decreased circulating thyroid hormones in a 10-day gavage study in rats with a NOAEL of 1 mg/kg bw/day (Allen-Rowlands et al. 1981, as quoted in US ATSDR, 2004), decreased lymphoproliferative responses in rats at a dose level of 3 mg/kg/day (LOAEL) (Luster et al. 1980, as quoted in US ATSDR, 2004), and generalised toxicity in male Rhesus monkeys at 0.73 mg/kg bw/day (LOAEL) (Allen et al. 1978; Lambrecht et al. 1978 (as quoted in US ATSDR, 2004). PBBs produced porphyria in rats and male mice at doses as low as 0.3 mg/kg bw/day. The no-effect level was 0.1 mg/kg bw/day.

These results show that hexabromobiphenyl produced long-term toxicity in experimental animals at very low doses, a critical effect for the purposes of risk characterization being the effects seen in the thyroid in rats at doses as low as 0.05 mg/kg bw/day, comprising increased number and decreased size of follicles, accompanied by changes in levels of circulating T3 and T4 hormone (Akoso et al. 1982, as quoted in US ATSDR, 2004).

Hepatocarcinogenicity of hexachlorobiphenyl has been demonstrated in a number of studies including repeated dose studies in Fischer-344/N rats and B6C3F1 mice (males and females) administered FireMaster™ FF-1 at dosages of 0, 0.1, 0.3, 1, 3, or 10 mg/kg bw/day (NTP 1983, NTP, 1992, as quoted in US ATSDR, 2004). Tumors included hepatic cellular adenoma and carcinoma and, in female rats, cholangiocarcinoma. The lowest dose of FireMaster™ that produced tumors (primarily adenomas rather than carcinomas) in rats was 3.0 mg/kg bw/day for 2 years, and in mice the dose was 10 mg/kg bw/day (NTP 1983, as quoted in US ATSDR, 2004). Mice receiving 0.15 mg/kg bw/day in a study involving pre- and perinatal exposure in addition to lifetime exposure did not suffer any adverse effects (NTP, 1992, as quoted in US ATSDR, 2004). The International Agency for Research on Cancer (IARC) in 1987 concluded that there was sufficient evidence that hexabromobiphenyl is carcinogenic in mice and rats and possibly carcinogenic to humans (Group 2B). Hexabromobiphenyl is not genotoxic in in vitro microbial and mammalian cell gene mutation assays (see Table 88 in IPCS, 1994), although it has been reported to interfere with cell-to-cell communication (Sleight, 1985 as quoted in IPCS, 1994). These results, coupled with the results of tumor promotion studies (e.g. Schwartz et al., 1980; Jensen et al., 1982, 1983, 1984; Jensen & Sleight, 1986; Rezabek et al., 1987; Dixon et al., 1988, as quoted in IPCS, 1994) indicate that these chemicals cause cancer by epigenetic mechanisms, involving both hepatic toxicity and hypertrophy, including cytochrome P-450 induction (IPCS, 1994).

Oral administration of hexabromobiphenyl was associated with adverse effects on reproductive parameters in a range of experimental animals (see Table 86 and 87 in IPCS, 1994). The most common adverse effects on reproduction were failure in implantation and decreases in pup viability of offspring. These effects were seen at a dose level of 28.6 mg/kg bw/day in a 15-day reproductive toxicity study in rats, with dosing between gestational day 0-14 (Beaudoin 1979, as quoted in US ATSDR, 2004) and in mink at concentrations of 1 mg/kg diet (Aulerich and Ringer, 1979 as quoted in IPCS, 1994). Increased menstrual cycle duration and prolonged implantation bleeding were observed in female monkeys fed approximate daily dose levels of 0.012 mg/kg bw/day for 7 months before breeding and during pregnancy. Fetal deaths were also observed after approximately 1 year of exposure. Effects were attributed to decreases in serum progesterone (Lambrecht et al. 1978; Allen et al. 1978; Lambrecht et al. 1978; 1979, (as quoted in US ATSDR, 2004).
### Table 2.2  Pivotal toxicological studies on the toxicity of hexabromobiphenyl.

<table>
<thead>
<tr>
<th>Species</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat</td>
<td>Short-term/acute toxicity</td>
<td>decreased thyroid serum T4 hormones</td>
<td>3 mg/kg bw/day (LOAEL) 1 mg/kg bw/day (NOAEL)</td>
</tr>
<tr>
<td></td>
<td>10 day repeat dose gavage study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rat, Sprague Dawley</td>
<td>30-day dietary feeding study</td>
<td>increased number and decreased size of thyroid follicles</td>
<td>0.05 mg/kg bw/day (LOAEL)</td>
</tr>
<tr>
<td>Mice B6C3F1</td>
<td>In utero and post partum exposure from Gd 0-ppd 56</td>
<td>hepatocellular adenoma and carcinoma in offspring</td>
<td>1.5 mg/kg bw/day (LOAEL) 0.15 mg/kg bw/day (NOAEL)</td>
</tr>
<tr>
<td>Rhesus Monkey</td>
<td>25-50 wk dietary feeding study</td>
<td>34% weight loss in adult male, 0% weight gain in juvenile, proliferation of mucosal cells, chronic inflammation, severe ulcerative colitis, alopecia, keratinization of hair follicles and sebaceous glands, clinical chemical and hepatic changes</td>
<td>0.73 mg/kg bw/day (LOAEL, males)</td>
</tr>
<tr>
<td>Rat, Sprague Dawley</td>
<td>7 month dietary feeding study</td>
<td>decreased thyroid serum T3 and T4 hormones</td>
<td>0.45 mg/kg bw/day (LOAEL)</td>
</tr>
<tr>
<td>Monkey, Rhesus</td>
<td>增加了月经周期时间，其在4/7;implantation bleeding in 2/7, 1/7 fetuses were aborted, 1/7 fetuses stillborn, 12% decreased birth weight and 22% decreased postnatal weight gain in 4/7 survivors</td>
<td></td>
<td>0.012 mg/kg bw/day (LOAEL)</td>
</tr>
</tbody>
</table>

### Toxicity of hexabromobiphenyl in humans

Information on toxicological effects of PBBs (and by inference, hexabromobiphenyl) in humans has mainly been derived from the Michigan accident described in Section 2.1.4 of this draft Risk Profile (Carter (1976), Getty *et al.* (1977), Kay (1977), Di Carlo *et al.* (1978), Damstra *et al.* (1982), Zabik (1982), and Fries (1985b), as quoted in EHC 152 (IPCS, 1994). This accident resulted in widespread exposure of consumers for periods approaching 1 year, before the contamination of food by PBBs was identified and affected foodstuffs were removed from the food chain.

Adverse health effects reported included changes in liver enzymes, nausea, abdominal pain, loss of appetite, joint pain and fatigue (Anderson *et al.*, 1978b, 1979, as reported in IPCS, 1994), together with reports of skin disorders, including acne and hair loss, in the period following the contamination. (IPCS, 1994). Similar skin disorders have also been reported in workers with occupational exposure to PBBs (Anderson *et al.*, 1978a, as reported in IPCS, 1994), and also following exposure to the polychlorinated dioxins and furans.

Detailed epidemiological studies have been carried out on the health status of exposed individuals including immunological status, cancer incidence, reproductive effects and effects on development of young children. These studies have in the main failed to establish a definite link between any of these effects and exposure to PCBs, although some studies have reported decreased immune function in Michigan farm residents (Bekesi *et al.*, 1979, 1987) and effects have also been reported on pubertal development in young females (see endocrine-disrupting effects below). There are no reports of acute hexabromobiphenyl intoxication in humans, and there is also no consistent epidemiological evidence for hepatocarcinogenicity in exposed
humans. A relationship between increasing serum levels (> 2 ppb) of PBBs and increasing risk of breast cancer was indicated in case-control studies of women exposed during the Michigan contamination episode (Henderson et al. 1995; Hoque et al. 1998), but according to US ATSDR, 2004 (and quoted from this source) the results are only suggestive due to factors such as the small number of cases, insufficient information on known breast cancer risk factors, and confounding exposures to other organochlorine chemicals.

Effects on endocrine systems
The PBBs (and by inference, hexabromobiphenyl) are considered to have effects on endocrine systems. They have been evaluated under the EU-Strategy for Endocrine Disrupters and have been placed in category 1 (evidence of endocrine-disrupting activity in at least one species using intact animals) in the priority list of chemicals established under the EU-Strategy. This categorisation is based on evidence of delayed vaginal opening in newborn rats, epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer among women exposed to polybrominated biphenyls (as reported in BKH report, 2000). In an assessment (Blanck et al., 2000) of pubertal development in girls and young women exposed in utero and via breast milk to high levels of PBBs (> 7ppb), it was found that this population had an earlier age to menarche than a similar breastfed population exposed to lower levels of PBBs, or than a highly-exposed population who were not breastfed. Earlier pubic hair development was also seen in the more highly exposed population, suggesting an effect of PBBs on pubertal events (Blanck et al., 2000).

Conclusion on effects assessment and toxicity of hexabromobiphenyl
Hexabromobiphenyl is readily absorbed into the body and accumulates following prolonged exposure. Although the acute toxicity of hexabromobiphenyl is low, a number of chronic toxic effects including hepatoxicity have been observed in experimental animals at doses around 1 mg/kg bw/day following long-term exposure, and effects are seen in the rat thyroid at doses as low as 0.05 mg/kg bw/day. Cancer was induced in animal studies at a dose of 0.5 mg/kg bw/day and the no-observed-effect level was 0.15 mg/kg bw/day. The International Agency for Research on Cancer has classified hexabromobiphenyl as a possible human carcinogen (IARC group 2B). The PBBs (and by inference, hexabromobiphenyl) are endocrine disrupting (ED) chemicals, and effects are seen on reproductive capacity in rats, mink and monkeys. Effects were seen in monkeys fed 0.012 mg/kg bw/day for 7 months before breeding and during pregnancy, the lowest effect level reported for hexabromobiphenyl in toxicology studies. There is epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer in exposed women. It can be concluded that hexabromobiphenyl is a bioaccumulative chemical with a range of potentially adverse effects on health, including carcinogenicity, reproductive toxicity, endocrine and other hormone-disrupting effects, at very low levels of exposure.

Ecotoxicity
Only few data are available on effects of PBBs on other organisms than mammals. Toxicity tests with technical decabromobiphenyl (Adine 0102) and bacteria (Pseudomonas putida) and the water flea Daphnia magna are quoted in EHS 152 (1994). The results were an EC10 of 53 mg/L for Pseudomonas putida (cell multiplication) and an EC50 > 66 mg/liter for Daphnia magna (immobilization, 24 hours). Because the water solubility of hexabromobiphenyl is measured in µg/L, these data do not seem valid. However, the fact that the NOEC is reported to be < 2 mg/L indicates that the water fleas were affected at the lowest concentration tested – and thereby, that hexabromobiphenyl is toxic to these organisms at low concentrations.

MacPhee & Ruelle (1969) and Applegate et al. (1957) report results from short term tests with hexabromobiphenyl (CAS No. 36355-01-8) and several species of fish in the range 5-10 mg/L, which is far above the water solubility (Quoted from the Ecotox data base (US EPA, 2006)).
In a field study on water birds, correlations between behavioural effects and reproductive success were not unambiguously correlated to body burdens of PBBs. (EHS 152 (IPCS, 1994)).

Darnerud (2003) explains the validity of reading across, arguing that the pattern of toxicity of PBBs should be similar to that of PCBs apart from the change in effects that the chlorine-bromine substitution brings about. Consequently, the planar PBBs are expected to be most toxic (as they bind to the Ah receptor) and toxicity to decrease through mono-ortho congeners to di-ortho congeners. This should be supported by experimental evidence, as 3,3',4,4',5,5'-hexabromobiphenyl was found to be the most toxic PBB congener in several systems (Darnerud, 2003).

In an untraditional fish early life stage test, Hornung et al. (1996) injected halogenated organic contaminants into rainbow trout eggs. For 3,3',4,4',5,5'- hexabromobiphenyl they found an LD$_{50}$ of 3,910 µg/kg. This result is not comparable to those of traditional fish tests, where exposure is via the water but it is comparable to results of other test with similar exposure. Hornung et al. (1996) made such experiments to compare the toxicity of PBBs and PCBs and found that both 3,3',4,4'-tetrabromobiphenyl and 3,3',4,4',5,5'-hexabromobiphenyl were 10-fold more potent than identically substituted polychlorinated biphenyls.

Based on this, it seems to be relevant to expect the environmental toxicity of hexabromobiphenyl to be comparable to that of hexachlorobiphenyl.

**SYNTHESIS OF THE INFORMATION**

Hexabromobiphenyl belongs to a wider group of polybrominated biphenyls (PBBs). It has mainly been used as a fire retardant. Hexabromobiphenyl is already listed in Annex I of the UNECE Protocol on POPs.

According to available data, hexabromobiphenyl can be considered to be highly persistent in the environment. There is evidence of low or no degradation in water, soil and sediment, in the laboratory as well as in the field. Therefore, hexabromobiphenyl is considered to be highly persistent.

Hexabromobiphenyl is less volatile than many POP substances. However, extensive data on monitoring shows that it is found throughout the Arctic wildlife, demonstrating that it does have a high potential for long range environmental transport.

With measured weight-based BCF values in the range 4,700 - 18,100 and biomagnification factors in the aquatic food chain exceeding 100, hexabromobiphenyl is considered to be highly bioaccumulative and to have a high potential for biomagnification. These properties are demonstrated by several authors to be comparable to those of hexachlorobiphenyl (a PCB compound), for which the bioaccumulative properties are well documented.

Hexabromobiphenyl is readily absorbed into the body and accumulates following prolonged exposure. Although the acute toxicity of hexabromobiphenyl is low, a number of chronic toxic effects including hepatotoxicity have been observed in experimental animals at doses around 1 mg/kg bw/day following long-term exposure, and effects are seen in the rat thyroid at doses as low as 0.05 mg/kg bw/day. The International Agency for Research on Cancer has classified hexabromobiphenyl as a possible human carcinogen (IARC group 2B). The PBBs are endocrine disrupting chemicals, and effects are seen on reproductive capacity in rats, mink and monkeys. There is epidemiological evidence of hypothyroidism in workers exposed to polybrominated biphenyls and of increased incidence of breast cancer in exposed women. Data on toxicity to other species than laboratory mammals is scarce but suggests the environmental toxicity of hexabromobiphenyl is comparable to that of hexachlorobiphenyl.

Based on the available data, hexabromobiphenyl should be considered as a POP warranting global action. All in all, safe levels of exposure cannot be set for substances such as hexabromobiphenyl which are highly persistent and highly bioaccumulative because of the difficulties in assessing long-term effects of life-long exposure to even low concentrations.
Production and use of hexabromobiphenyl has ceased over the last decades but it cannot be excluded that it is still produced or used in some countries. In addition to emissions during manufacture or use, hexabromobiphenyl can enter the environment from the widespread use of flame-retarded products. A considerable part of the substance produced will probably reach the environment sooner or later because of the high stability of these compounds. Furthermore, some of these chemicals may form toxic polybrominated dibenzofurans during combustion processes.

CONCLUDING STATEMENT

It has been demonstrated that hexabromobiphenyl clearly meets all the criteria laid down in Annex D of the Stockholm Convention: It is very persistent in the environment. It has a great potential for bioaccumulation and in addition there is clear evidence of its biomagnification. Due to its physical and chemical properties and based on findings in environmental samples, it is verified that hexabromobiphenyl can be transported long distances in air, far from its sources. Hexabromobiphenyl is a possible human carcinogen and can also be regarded as a substance capable of disrupting the endocrine system.

As hexabromobiphenyl can travel in the atmosphere far from its sources, neither a single country nor group of countries alone can abate the pollution caused by this substance. Regional action has already been considered necessary and hexabromobiphenyl is totally banned under the Convention on Long-range Transboundary Air Pollution Protocol on Persistent Organic Pollutants. Although the production and use of hexabromobiphenyl seems to be ceased in most countries, its reintroduction remains possible. This could lead to increased releases and levels in the environment.

Based on the available evidence, it is thus likely that hexabromobiphenyl can, as result of long-range environmental transport, cause significant adverse effects on human health and/or the environment, such that global action is warranted.
REFERENCES


Thomas, G.O. (2005) Absorption of decabromodiphenyl ether and other organohalogen chemicals by grey seals...Environmental Pollution Volume 133 (2005), Issue 3 P. 581-586


Production and uses

According to the Risk Profile on Hexabromobiphenyl, the commercial production of PBBs began in 1970. Approximately 6 million kg of PBBs were produced in the United States from 1970 to 1976. Hexabromobiphenyl constituted about 5.4 million kg (ca 88%) of this total (Neufeld et. al., 1977 quoted from UNEP 2006). The production in the USA stopped in 1975 (Quoted from US ATSDR, 2004). Re-initiation of manufacture of PBBs would require approval from the EPA (Quoted from US ATSDR, 2004).

According to the information available, production and use of Hexabromobiphenyl has ceased in most, if not all, countries. However, it is possible that Hexabromobiphenyl is still being produced in some developing countries or in countries with economies in transition.

In the United States and Canada, Hexabromobiphenyl was used as a fire retardant in three main commercial products: acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business machine housings and in industrial (e.g. motor housing), and electrical (e. g. radio and TV parts) products; as a fire retardant in coatings and lacquers; and in polyurethane foam for auto upholstery (Neufeld et. al., 1977 quoted from UNEP 2006) (Modified from EHC 152 (IPCS, 1994) and US ATSDR, 2004).

Approximately 5 million tonnes of Hexabromobiphenyl were produced in the USA from 1970 to 1976 (Hesse and Powers, 1978 quoted from UNEP 2006). Of the estimated 2,200 tonnes Hexabromobiphenyl produced in 1974 (IARC, 1978), about 900 tonnes were used in ABS plastic products and an even larger amount in cable coatings. The exact quantity used in polyurethane foam for automobile upholstery was not published. The two larger consumers ceased using Hexabromobiphenyl (one of these in 1972) because PBBs did not decompose in the ultimate incineration of scrapped automobiles (Neufeld et. al., 1977 quoted from UNEP 2006) (Quoted from EHC 152 (IPCS, 1994)).

The Risk Management evaluation is primarily based on information that has been provided by Parties to the Convention and Observers. Responses regarding the information specified in Annex F of the Stockholm Convention (risk management) have been provided by the following countries:

**Table 0-1: Annex F questionnaires delivered by April 2007**

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<td>08.02.2007</td>
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<td>Ministry of Environment</td>
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<td>Thailand</td>
<td>Ministry of Public Health, Hazardous Substance Control Group</td>
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<td>NGO Observer</td>
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Besides answers to the questionnaire major information sources used have been the following:

Specific national and international risk management reports for Hexabromobiphenyl have not been available. However, there are a number of reports such as Danish EPA (1999), OSPAR (2001), BMU (2000), UBA (2003a, 2003b), USEPA (2005), which address the issue of control and substitution of brominated flame retardants at international or national scale.

General aspects of management for PBBs without further specification are reported in the Draft “Technical guidelines for the environmentally sound management of wastes consisting of, containing or contaminated with polychlorinated biphenyls (PCBs), polychlorinated terphenyls (PCTs) or polybrominated biphenyls (PBBs)”, Version 7 April 2006, available at http://www.basel.int/techmatters/index.html.

Status of the chemical under international conventions
Hexabromobiphenyl is listed in Annex I of the Protocol to the Convention on Long-range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants. The provisions of the Protocol oblige Parties to phase out all production and uses of Hexabromobiphenyl. Hexabromobiphenyl, together with other PBBs, is also included in the UNEP/FAO Rotterdam Convention on the Prior Informed Consent Procedure (PIC) for Certain Hazardous Chemicals and Pesticides in International Trade.

Under the OSPAR Convention, brominated flame retardants (including hexabromobiphenyl) are enumerated as part of the List of Chemicals for Priority Action (March 2002). A background document has been prepared by Sweden. It was first published 2001 and was updated in 2004 (OSPAR, 2006). The action recommended in the updated document is to support several measures of the European Community on Polybrominated Biphenyls and to develop an OSPAR monitoring strategy for several Polybrominated Biphenyls and to review the need for further OSPAR measures to supplement the eventual measures of the European Community.

Under the HELCOM Convention, Hexabromobiphenyl is listed as a selected substance for immediate priority action (Recommendation 19/5, Attachment, Appendix 3) and is scheduled for elimination (Annex I, part 2). HELCOM aims to move towards the target of the cessation of discharges, emissions and losses of hazardous substances by the year 2020.

Under the Basel Convention, PBBs are classified as hazardous in Annex VIII without further specification.

The Strategic Approach to International Chemicals Management (SAICM) includes POPs as a class of chemicals. An objective of SAICM is to ensure by 2020 that chemicals or chemical uses that pose an unreasonable and otherwise unmanageable risk to human health and the environment (among other POPs) based on a science-based risk assessment and taking into account the costs and benefits as well as the availability of safer substitutes and their efficacy, are no longer produced or used for such uses (SAICM 2006).

Any national or regional control actions taken
In the European Union, Hexabromobiphenyl is listed in Annex I to Regulation (EC) No 850/2004 on persistent organic pollutants with complete prohibition of production and use in all the 27 Member States. The EC Directive 2002/96/EC on Waste from Electric and Electronic Equipment (WEEE) requires that brominated flame retardants have to be removed from any separately collected WEEE prior to further treatment. EC Directive 2002/95/EC on Restrictions on Certain Hazardous Substances in Electric and

45 http://www.helcom.fi/environment2/hazsubs/action/en_GB/list
Electronic Equipment (ROHS) stipulates in article 4 that electric and electronic articles may not contain polybrominated biphenyls from July 2006.\(^{46}\)

The issue of Hexabromobiphenyl in waste is addressed at the European level in Regulation 850/2004/EC. As amended by regulation 1195/2006/EC POPs such as Hexabromobiphenyl in wastes have to be destroyed if concentration limits of 50 mg/kg are exceeded.

At the national level, legal control actions taken have been reported by Canada and the USA. In Canada all Polybrominated Biphenyls appear on Schedule 1 (List of Toxic Substances) of CEPA 1999, and are subject to prohibitions on their use. In addition, Polybrominated Biphenyls appear on Schedule 3, Part 1 (Export Control List – Prohibited Substances) of CEPA 1999, effectively prohibiting their export, except for the purpose of destroying the substance.

In the USA, Hexabromobiphenyl is subject to a TSCA Significant New Use Rule which would require notification to EPA prior to re-initiating manufacture or import for any use (63 FR 45955, August 28, 1998; 40 CFR 721.1790).

Identification of possible control measures

Control measures already widely implemented are elimination of production, use, export, and import. US EPA refers to the subjection of Hexabromobiphenyl to the Toxic Substances Control Act (TSCA) and the Significant New Use Rule which would require notification to EPA prior to re-initiation of manufacture or import for any use (63 FR 45955, August 28, 1998; 40 CFR 721.1790). Mauritius refers to the Dangerous Chemicals Control Act 2004 (DCCA) which subjects to control by the Dangerous Chemicals Control Board (DCCB) all importations of chemicals.

Exposure to Hexabromobiphenyl may occur in connection with the use of products, in the recycling of plastics containing PBBs and after disposal to landfills so that in general releases from articles in use and releases from waste should be considered when discussing the need for measures. (OSPAR, 2001).

Measures related to stocks and articles in use, for release control and clean-up are not addressed in the responses to the Annex F questionnaire.

As Hexabromobiphenyl is an intentionally produced chemical, the most evident and efficient control measure would be the prohibition of all production and uses of Hexabromobiphenyl and Hexabromobiphenyl containing products and articles. Alternatively, in accordance with Article 3(1), legal and administrative measures (e.g. withdrawal or denial of pre-production and pre-marketing authorisation of chemicals) necessary to eliminate Hexabromobiphenyl would have the same impact. As no remaining uses of Hexabromobiphenyl have been identified, listing of Hexabromobiphenyl in Annex A without any specific exemptions could be the primary control measure under the Convention. Listing of Hexabromobiphenyl in Annex A would also mean that the provisions of Article 3 on export and import and of Article 6 on identification and sound disposal of stockpiles and waste would apply.

Alternatives

The Hexabromobiphenyl Risk Profile describes three principal commercial products that contained Hexabromobiphenyl in the USA and Canada:

1. acrylonitrile-butadiene-styrene (ABS) thermoplastics used for business machine housings and electrical products such as radio and TV;
2. fire retardant in cable coatings and lacquers, and
3. fire retardant in polyurethane foam for auto upholstery.

Consequently evaluation and assessment of alternatives should focus on these uses as far as information is available.

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\(^{46}\) For use in article 5(1) a maximum concentration of 0.1% by weight in homogenous material of PBB shall be tolerated
A number of reports on risk assessment of alternative substances and processes are available. The OSPAR priority substances Series (OSPAR, 2001) provides summary information on alternatives for brominated flame retardants. The Danish Environmental Protection Agency has described alternative halogen-free flame retardants for a variety of uses including epoxies, phenolic resins, rigid and soft polyurethane foam, textiles, and a variety of plastics including ABS (Danish EPA, 1999). Both drop-in chemical substitutes and alternative materials are listed. US EPA has described process alternatives and chemical substitutes for polyurethane foam (USEPA, 2005). The German Federal Ministry of Environment has reported on alternatives for flame retardants used in electronics, upholstery, and other sectors (BMU, 2000).

As brominated flame retardants only account for about 15% of the global flame retardant consumption, principally a large number of compounds may be considered as alternatives (OSPAR, 2001). Substitution can take place at three levels:

1. brominated flame retardants can in some applications be replaced by another flame retardant without changing the base polymer; (major group of substitutes)
2. the plastic material, i.e. the base polymer containing flame retardants and other additives, can be replaced by another plastic material; (e.g. polysulfone, polyaryletherketone and polyethersulfone)
3. a different product can replace the product, e.g. the plastic material is replaced by another material (e.g. wool), or the function can be fulfilled by the use of a totally different solution.

Reported chemical substitutes (see indent 1) currently used in Europe comprise the group of (a) organophosphorus compounds, (b) inorganic fire retardants and (c) nitrogen containing compounds (Danish EPA, 1999).

(a) The group of organophosphorus contains the following main substances divided into the groups of:
   1) halogenated organophosphorus (Tris dichloropropyl phosphate, tris-chloropropylphosphate and tri-chloroethyl phosphate)
   2) non-halogenated organophosphorus (Triphenyl phosphate, Tricresyl phosphate, Resorcinol bis(diphenylphosphate), Phosphonic acid, (2-((hydroxymethyl)carbamyl)ethyl)dimethyl ester, Phosphorus and nitrogen constituents for thermosets
(b) The group of inorganics contains Aluminium trihydroxide, Magnesium hydroxide, Ammonium polyphosphate, Red phosphorus and Zinc borate
(c) The group of nitrogen containing compounds contains melamine and melamine derivatives, e.g. melamine cyanurate and melamine polyphosphate

In addition USEPA 2005 provides an assessment for Tribromoneopentyl alcohol, Chloroalkyl phosphate, other aryl phosphates, Tetrabromophthalate diol ester and reactive brominated flame retardants as potential substitutes for PBDE.

Tetrabromobisphenyl (TBBP) and reactive phosphorus polyols have been mentioned as potential alternatives as well.

As an overall assessment the Danish report on alternative flame retardants (Danish EPA, 1999) makes the following assessment:

1. Substitutes are available for most applications at relatively low extra cost;
2. Organophosphorus compounds can be released from products in significant amounts;
3. Inorganic phosphorus compounds are more positive than organophosphorus ones though a more comprehensive assessment is needed;
4. Aluminum hydroxide has desirable minimal toxicity characteristics presumable shared by magnesium hydroxide though no assessment is currently available; however high loading may be a disadvantage
5. Zinc borate and melamine may be desirable but require a more comprehensive assessment

The German report on substitution of environmentally relevant flame retardants (BMU, 2000) takes the following conclusions:

1) More data is needed to assess non-halogen phosphoric esters;
2) Melamine is problematic (due to lack of data, presence in environmental samples and moderate organ toxicity); and
3) Merely zinc borate, magnesium hydroxide and expandable graphite should not cause any problems when used.
In addition the report concludes that the substitution of alternatives for POPs provokes a deeper question about methods to evaluate and compare the hazards of various substances.

The OSPAR report concludes that available data show that there are less hazardous alternatives for use a flame retardant e.g. aluminium trihydroxide. But it cannot be disregarded that less suitable substances might be among the alternatives. When industry selects a method to inhibit fire, data has to be generated in order to allow a good choice from an industrial/product and environmental point of view (OSPAR, 2001).

It has to be stated that under HELCOM Triphenyl phosphate and Tricresyl phosphate are listed as potential substances of concern (Recommendation 19/5, Attachment, Appendix 2). A comparable procedure has been taken for TBBP-A which is enumerated in the list of chemicals for priority action (March 2002) under OSPAR.

The assessment of potential alternatives in the environmental profiles of chemical flame retardant alternatives for low-density polyurethane foam (USEPA, 2005) reveals an overall hazard of investigated substances being moderate for human health and ranging from low to high for the aquatic environment. Melamine, Tris (1,3-dichloro-2-propyl) phosphate (TDCPP) and Ammonium Polyphosphate (APP) are shortly addressed but assessed as less appropriate for low density polyurethane foam in the US report due to technical aspects (scorching).

Reported alternative technologies (ident 3) comprise barrier technologies, graphite impregnated foam and surface treatment (USEPA, 2005).

One screening guide focuses on evaluating environmentally preferable flame retardants for TV enclosures by developing and using a “Green Screen” (Rossi et al., 2007) The criteria used by the Green Screen include hazard endpoints with categories of high, medium, and low; criteria for determining each level of chemical concern; and consideration of degradation products and metabolites.

Lowell Center for Sustainable Production has developed an Alternatives Assessment Framework with the goal of “Creating an open source framework for the relatively quick assessment of safer and more socially just alternatives to chemicals, materials, and products of concern.” (Lowell, 2005). The Framework discusses goals, guiding principles, decision making rules, comparative and design assessment, and types of evaluation. Since the Framework is designed to be an open source tool, the Lowell Center encourages companies, NGOs, and governments to use, adapt, and expand on it.

In the light of parallel work on risk management evaluation of other brominated flame retardants under the Stockholm Convention and having in mind a certain convertibility of substances in a number of uses, consistency in recommendations for alternatives should be aimed at as far as appropriate.

**Description of alternatives (substances)**

According to OSPAR 2001, alternatives mentioned as chemical substitutes for electric and electronic equipment (e.g. aluminium trihydroxide, magnesium hydroxide, red phosphorus or organic phosphorus compounds) may not be viable for equipment which needs to meet certain demands with respect to technical and safety standards.

The flame retarded plastics used for switches, sockets and other applications where the material is in direct contact with live parts of electronic and electrical appliances are mainly made of thermoplastic polyester and polyamides. According to the Danish Environmental Protection Agency decaBDE has been substituted by Tetrabromobisphenol A (TBBP-A) and brominated styrene, but diarylphosphonate, melamine cyanurate or red phosphorus may be used as well.

Flame retardants for rigid polyurethane foams may be based on ammonium polyphosphates or red phosphorus. For flexible foams chlorinated phosphate esters, in some cases combined with melamine, ammonium polyphosphates and reactive phosphorus polyols are used.

Polysulfone, polyaryletherketone and polyethersulfone are plastics that are self-extinguishing and can be used without the addition of flame retardants. Less flammable materials e.g. wood and metals can also replace plastic material. Another example of the substitution of material is using wool instead of a more flammable fabric (OSPAR, 2001).

**Alternatives for ABS plastics**

Organic phosphorus compounds which are available as halogenated or non-halogenated substances can serve as alternatives for use in ABS plastics.

Halogenated organophosphorus compounds include tris-chloropropyl-phosphate (TCP), tris-chloroethyl-phosphate, and tris dichloropropyl phosphate (TDCPP) (BMU, 2000). According to (USEPA, 2005) TDCPP is often used in polyurethane foam in the US and abroad. However, tris dichloropropyl phosphate, tris-chloropropyl-phosphate and tri-chloroethyl phosphate entail moderate concern for carcinogenicity, reproductive toxicity, developmental toxicity, systemic toxicity, genotoxicity, acute and chronic ecotoxicity, and persistence. (WHO, 1998), (USEPA, 2005)

Tetrabromobisphenol A (TBBPA or TBBP-A) is regarded as very poisonous to water-living organisms and very persistent. This flame retardant is mainly used in printed circuit boards. Since TBBPA is chemically bound to the resin of the printed circuit board, there is no exposure of the aquatic environment and therefore no risk.

Non-halogenated organic phosphorus compounds as alternative flame retardants for High Impact Polystyrene (HIPS) and poly carbonate (PC) plastics include commonly used substances such as triphenyl phosphate (TPP), tricresyl phosphate (TCP), resorcinol bis(diphenylphosphate) (RDP), and phosphonic acid (2-((hydroxymethyl) carbamyl)ethyl)-dimethyl ester (Pyrovatex®) (Danish EPA, 1999).

(USEPA, 2005) reports moderate overall hazard for TPP while it is considered to be environmentally hazardous in Germany due to its toxicity to aquatic organisms (BMU, 2000) TCP toxicity apparently differs according to isomer. IPCS recommends the use of purified m- and p- isomers to prevent formation of the highly toxic o-isomer (Danish EPA, 1999). RDP is usually used in combination with TPP.

Pyrovatex® is not well-characterized though the Danish report notes that it is a weak inhibitor of acetyl choline esterase and the microsomal enzyme system and that high concentrations induced chromosome aberrations and reverse mutations. The German report notes that Pyrovatex easily separates formaldehyde and often is used together with ethylene carbamide to help trap released formaldehyde (BMU, 2000).

Both the German and Danish reports comment on the insufficiency of human and environmental toxicity data for RDP. Due to the absence of toxicity information and its possible transmission to humans from use of consumer products, the reports conclude that the data is insufficient to be able to make a recommendation.

**Alternatives in coatings and lacquers**

Halogen-free rubber cables can contain aluminium trihydroxide and zinc borate as flame retardant alternatives and incorporate the ethylene vinyl acetate polymer as well.

Aluminum trihydroxide is the most frequently used flame retardant (Danish EPA, 1999). Due to an endothermic reaction when decomposing and other properties it is highly effective and also suppresses smoke. Its functional disadvantage is that large amounts are required (up to 50%) which can affect the properties of the material. It would be extremely unlikely for its use in consumer products to cause adverse effects. Accumulation of the substance in food chains is not detectable (Danish EPA, 1999). Also the German alternatives report describes the use of aluminium trihydroxide as a flame retardant as “unproblematic.”

Magnesium hydroxide has comparable effects, however the environmental effects still have to be assessed (Danish EPA, 1999).
Zinc borate is often combined with aluminum trihydroxide and used to substitute for antimony trioxide. The German report describes the teratogenicity of boron along with its ability to irritate the eyes, respiratory organs, and skin at high levels. It assumes that its use as a flame retardant will not result in significant additional concentrations for humans. However, it concludes that it would be important to measure the ability for boron to be released in dust before its wide use in consumer products in homes.

**Alternatives for polyurethane foams**

Ammonium polyphosphate (APP) is an additive flame retardant currently used to flame retard flexible and rigid polyurethane foams, as well as intumescent laminations, molding resins, sealants and glues. APP formulations account for approximately 4-10% in flexible foam, and 20-45% in rigid foam (USEPA, 2005). APP is commonly used in combination with Aluminum hydroxide and Melamine. It metabolizes into ammonia and phosphate and is not thought to cause acute toxicity in humans (BMU, 2000). However, there are no analyses of long-term toxicity, teratogenicity, mutagenicity, or carcinogenicity. Ammonium polyphosphate breaks down rapidly and does not accumulate in the food chain. Skin irritation is possible due to the formation of phosphoric acids.

Red phosphorus mainly used in polyamids is easily ignited and poorly characterized toxicologically. There is no data available for red phosphorus on ecotoxicity, carcinogenicity, mutagenicity, long-term toxicity, or toxicokinetics and no data exists on concentrations of red phosphorus in indoor or outdoor air (from sewage sludge) as a consequence of incorporating red phosphorus into products. Eye and mucous membrane irritation can result due to the formation of phosphoric acid. Ecosystem accumulation is thought to be unlikely (BMU, 2000). US government researchers have noted that high levels of toxic phosphine were observed during long-term storage of red phosphorus (Anthony et al., 2006).

Information in (Danish EPA, 1999) confirms the observations made, and states that “smaller producers of plastic products avoid the use of red phosphorus”.

Melamine and its derivatives (cyanurate, polyphosphate) are currently used in flexible polyurethane foams, intumescent coatings, polyamides and thermoplastic polyurethanes (Special Chemicals, 2004). They are used effectively in Europe in high-density flexible polyurethane foams but require 30 to 40 percent melamine per weight of the polyol.

Melamine and its derivates display several toxic effects in animals. (USEPA, 1985, Danish EPA, 1999). In a fire, melamine cyanurate will release toxic fumes such as hydrocyanic acid and isocyanate (BMU, 2000).

However the Danish report notes that based on the results of the Swedish flame retardants project (Berglind, 1995) and a study from Stevens et al. (1999) there is no data on emission from products and that melamine appears to have low acute and chronic toxicity and concludes that, “…no adverse effects are envisaged from the level of exposure expected from the use of melamine as a flame retardant.”(Danish EPA, 1999).

In contrast, the German report describes the lack of data, presence in environmental samples and moderate organ toxicity of melamine and concludes it is a “problematic substance” (BMU, 2000).

According to IPEN specific reactive phosphorus polyols as potential alternative for soft polyurethane foam were not identified in the Danish report, though polyglycol esters of methyl phosphonic acid (CAS 676-97-1) have been used for flame retardants in polyurethane foam (e.g. CAS 294675-51-7) (OPCW, 2006). Researchers at the Oak Ridge National Laboratory in the US describe methyl phosphonic acid as one of degradation products of chemical weapons with “significant persistence.” (Munro et al., 1999) Other types of toxicity information are minimal but the substance reacts violently with water (USEPA, 1985). The phosphonic acid family also includes amino-methyl phosphonic acid (AMPA), a degradation product of the herbicide, glyphosate (also known as [carboxymethylamino] methyl phosphonic acid.) (Annex F responses, 2007, IPEN).

The US EPA Design for Environment report on flame retardant alternatives (USEPA, 2005) investigated the toxicological properties of 15 chemical substitutes for PentaBDE in low density foam. 12 of these substances have a moderate or high concern for persistence or would produce persistent
degradation products. An additional 6 substances have a moderate concern for the ability to bioaccumulate. All substances (including triphenyl phosphate, tribromoneopentyl alcohol and proprietary aryl phosphates) raised moderate overall concern for human health and ranged from low to high hazard for the aquatic environment.

**Description of alternatives (technologies)**

Three currently-available alternative technologies (barrier technologies, graphite impregnated foam and surface treatment) are shortly discussed in US EPA 2005.

Barrier technologies have the widest immediate commercial applicability and involve layers of materials that provide fire resistance. These include boric acid-treated cotton materials used in mattresses; blends of natural and synthetic fibers used in furniture and mattresses (VISIL, Basofil, Polybenzimidazole, KEVLAR, NOMEX and fiberglass); and high performance synthetic materials used in firefighter uniforms and space suits. As regards barrier technologies that use cotton and boric acid potential negative effects of boron (see above; BMU 2000) should be taken into account and it would be important to measure the ability for boron to be released in dust before its wide use in consumer products in homes.

More information on barrier fabrics or even eliminate the use of filling material can be found in (Lowell, 2005) and in (Posner, 2004) (USEPA, 2005).

Graphite impregnated foam and surface treatments have limited commercial uses. Graphite impregnated foam (GIF) can be considered an “inherently flame-resistant foam” that is self-extinguishing and highly resistant to combustion. It is a relatively new technology and is largely used in niche markets such as for general aircraft seating.

Surface treatments are also used in some applications and niche markets and may be appropriate for some textile and furniture manufacturing. However, surface treatments may not be viable as industry-wide replacements for use in low-density foam USEPA 2005):

**Technical feasibility**

All the alternatives described above are technically feasible and have been used in commercial applications (Annex F responses, 2007, IPEN). No specific comments on this topic have been provided by other parties.

**Costs, including environmental and health costs**

The prices of the alternatives are in general not higher than the BFRs but higher loading is often necessary. This is in particular true with respect to the inorganic compounds aluminum trihydroxide and magnesium hydroxide. Due to the low price of aluminum trihydroxide alternative materials may not be more expensive than BFR containing materials, but magnesium containing materials will usually be significantly more expensive. (Danish EPA, 1999)

As concerns alternative technologies (USEPA, 2005) describes the boric acid-treated cotton as “… the least expensive flame-retardant barrier materials available.” However, also GIF modified foams can be priced competitively by minimizing the expense associated with flame-retardant fabric. According to IPEN however, there are important points to consider when evaluating the costs of alternatives for any product as specified in (Ackermann et al., 2006):

- Alternatives with a higher initial purchase cost may actually be more cost effective over the life of the product when durability and other factors are taken into account.
- Mass-production of alternatives can significantly lower their costs.
- The costs of initiatives to protect health and the environment are frequently overestimated in advance and later decline rapidly after the regulation is implemented.

**Efficacy**

According to IPEN the alternatives described above meet US federal and state regulatory requirements along with standards bodies such as ASTM (American Society for Testing and Materials) and UL (Underwriters Laboratories). However, chemical manufacturers and foam manufacturing trade groups do not consider APP to be an alternative for brominated flame retardants
on a large scale. Reasons for this are that APP is typically incorporated as a solid, it has adverse effects on foam properties and processing and it is not considered to be as effective as a fire retardant compared to other alternatives (USEPA, 2002 quoted in USEPA 2005).

Melamine and TDCPP as two of the most commonly used chemicals to flame retard high-density, flexible polyurethane foam either result in scorching of the foam (an aesthetic effect unless severe) or a negative effect on the physical properties of foam if used in low-density flexible foams. Also, many formulations of these chemicals are available only as solids; making them less desirable as drop in substitutes for some brominated flame retardants (USEPA, 2005).

**Availability**
The alternatives described here are available since many are already in commercial use (Annex F responses, 2007, IPEN).

**Accessibility**
The alternatives described here are accessible since many are already in commercial use (Annex F responses, 2007, IPEN).

**Efficacy and efficiency of possible control measures in meeting risk reduction goals**

**Technical feasibility**
The essential phase-out of global production and use of Hexabromobiphenyl indicates that technically feasible alternatives have already been implemented (Annex F responses, 2007, IPEN).

**Costs, including environmental and health costs**

According to IPEN the considerable phase-out of Hexabromobiphenyl that has already occurred indicates that costs of alternatives have not inhibited their substitution. However, IPEN notes that there are inherent problems with using cost-benefit analysis to evaluate risk reduction and regulatory decisions. A fundamental problem is the difficulty of estimating the benefits attributed to a particular control measure. As stated in (Heinzerling et al., 2004) there is no meaningful way of assigning a dollar figure to human and environmental health (Annex F responses, 2007, IPEN).

No specific comments on this topic have been provided by other parties. As the phase out of Hexabromobiphenyl however, has taken place long ago already, significant costs from a global ban of the product would not be expected. Additional costs could arise from specific provisions concerning identification, collection, dismantling and disposal of remaining equipment.

According to IPEN any consideration of costs associated with the phase-out of Hexabromobiphenyl and proper management of its wastes must fully take into account the Polluter Pays Principle. Even after production of Hexabromobiphenyl ceases, it remains necessary for governments to ensure that wastes are managed and disposed in appropriate ways that do not significantly harm public health and the environment.

This too frequently results in external costs that society must bear unless appropriate economic instruments exist to properly internalize these costs within the importer/producer/user industry. Governments are entitled to take measures that result in the polluter bearing the administrative costs to governments of preventing and controlling pollution from Hexabromobiphenyl and including emissions from wastes.

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Mechanisms are needed to enable governments to recover from PBB-producing industries the costs that they and society incur associated with protective control measures that take effect when Hexabromobiphenyl is listed by the Stockholm Convention as a POP.

The European Union Risk Reduction Strategy for octaBDE contains an analysis of advantages and drawbacks of possible measures to reduce the risks from octa-BDE (RPA, 2002).

**Summary of information on impacts on society of implementing possible control measures**

**Health, including public, environmental and occupational health**
According to the German Federal Environment Agency impacts of a restriction/ban of Hexabromobiphenyl under the Stockholm Convention are expected to be rather low for the European region as Hexabromobiphenyl is already restricted for certain uses within the EU and as a critical level for water has lately been defined. On a global scale however, a positive impact on human health and on the environment can be expected from a ban of Hexabromobiphenyl (Annex F responses, 2007, Germany). Also the Czech Republic does not expect impacts of possible control measures (Annex F responses, 2007, Czech Republic).

According to IPEN elimination of Hexabromobiphenyl production, use, export, and import through a listing in Annex A of the Stockholm Convention would positively impact human health and the environment by preventing use of a persistent toxic substance. No discernible negative impacts on society have been reported from prohibition or phase-out of Hexabromobiphenyl as it is apparently not currently in use. A listing in Annex A would prevent future production and integration into products.

**Agriculture, including aquaculture and forestry**
There are no likely economic impacts on agriculture, as Hexabromobiphenyl has not been used in that sector. The positive environmental impacts in the form of reduced pollution could also have indirect positive impact on agriculture.

**Biota (biodiversity)**
As the persistent, bioaccumulative and toxic properties of Hexabromobiphenyl were shown under the POPs-Protocol and under the Stockholm Convention, a positive impact on biota from a ban/restriction of the substance can be expected according to the German Federal Environment Agency.

**Economic aspects**
According to IPEN cost competitive alternatives that do not exhibit POPs characteristics have already been implemented by companies for all uses of Hexabromobiphenyl.

**Movement towards sustainable development**
As the persistent, bioaccumulative and toxic properties of Hexabromobiphenyl as well as its potential for a long-range transboundary transport were shown under the POPs-Protocol and an by the POP RC of the Stockholm Convention which concluded that HBB meet the screening criteria listed in Annex D, a positive impact on a globally sustainable development from a ban/restriction of the substance is to be expected by the German Federal Environmental Agency (Annex F responses, 2007, Germany).

According to IPEN reduction and elimination of Hexabromobiphenyl is consistent with sustainable development plans that seek to reduce emissions of toxic chemicals. A relevant global plan is the Strategic Approach to International Chemicals Management (SAICM) that emerged from the World Summit on Sustainable Development\(^5\). The Global Plan of Action of SAICM contains specific measures to support risk reduction that include prioritizing safe and effective alternatives for persistent, bioaccumulative, and toxic substances.

**Social costs**
Since Hexabromobiphenyl has already been replaced with other substances or technologies, the impact on costs for consumers of an Annex A listing should be negligible according to IPEN.

\(^5\) [http://www.chem.unep.ch/saicm/](http://www.chem.unep.ch/saicm/)
Other impacts (Waste and disposal implications- stocks, contaminated sites)
Since Hexabromobiphenyl has already been largely phased-out, the impact on municipal waste and disposal according to IPEN should be minimal.

However, the Risk Profile outlines former consumer uses of Hexabromobiphenyl including ABS plastic used for business machine housings and electrical products such as radio and TV, cable coatings, and polyurethane foam.

In addition there are concerns over export of electronic waste to developing countries leading to Hexabromobiphenyl releases during recycling operations. Finally, burning or incineration of Hexabromobiphenyl-containing waste could lead to formation and release of brominated dibenzo-p-dioxins and -furans.

A listing of Hexabromobiphenyl in Annex A would subject wastes, products or articles containing the substance to Article 6 of the Stockholm Convention and require that they are disposed of in environmentally sound manner.

According to the US EPA approximately 11.8 million pounds (5.4 million kg) of Hexabromobiphenyl were used in commercial and consumer products in the U.S. with an estimated use life of 5-10 years. It is assumed that most of these products, such as TV cabinet and business machine housings must have been disposed of by land filling or incineration (US ATSDR, 2004). The sole U.S. producer depleted their remaining stocks in April 1975 (IARC Monographs, 1972 to present, V. 18, p. 110, quoted in TOXNET entry, http://toxnet.nlm.nih.gov)

There are no data on obsolete products and stocks in Zambia.

Other considerations
Access to information and public education
As Hexabromobiphenyl is prohibited within the EU and as a critical level for water has been defined, the need for public education programmes on the impacts of Hexabromobiphenyl is low in Germany. However, information can be obtained by everybody from the official websites of the Stockholm Convention and CLRTAP POPs-Protocol as well as from websites and helpdesks of national authorities dealing with chemicals (Annex F responses, 2007, Germany).

In the Czech Republic the issue of Hexabromobiphenyl is part of the SC/UN ECE CRLTAP education and awareness campaign under the national implementation plan. In Zambia access to environmental information is low, though it has increased in the recent past (ECZ 2001, State of the environment, Lusaka, Zambia).

Status of control and monitoring capacity
According to IPEN listing Hexabromobiphenyl in Annex A will involve control measures that are straightforward to communicate and therefore should be effective and suitable, even in countries that have limited chemical regulatory infrastructure.

Synthesis of information
According to the Risk Profile on Hexabromobiphenyl known commercial production (about 5,400 t) has mainly taken place in the USA from 1970 to 1975 by a sole producer Michigan Chemical Cooperation, St. Louis. In other regions of the world known production was restricted to octa- and decabromodiphenyl ethers which were regarded to be less toxic.

There is no information on potential Hexabromobiphenyl production in Russia, developing countries or countries with economies in transition. According to Danish EPA (1999), PBBs may still be in production in Asia.

Hexabromobiphenyl has mainly been used in ABS plastics and coated cables. Based on an expected lifetime of 5-10 years for electrical and electronical products it is expected that all of the products have already been disposed of (US ATSDR, 2004).
Hexabromobiphenyl is already listed in Annex I of the Protocol to the Convention on Long-range Transboundary Air Pollution (CLRTAP) on Persistent Organic Pollutants (Aarhus Protocol), requiring to phase out all production and uses. Hexabromobiphenyl, together with other PBBs, is also included in the UNEP/FAO Rotterdam Convention on the Prior Informed Consent Procedure (PIC) for Certain Hazardous Chemicals and Pesticides in International Trade. OSPAR lists Hexabromobiphenyl as chemicals of priority action since 1998.

At the European level Hexabromobiphenyl is listed in Annex I to Regulation (EC) No 850/2004 on persistent organic pollutants with complete prohibition of production and use. In addition Directive 2002/96/EC on Waste from Electric and Electronic Equipment (WEEE) requires that brominated flame retardants have to be removed from any separately collected WEEE prior to further treatment. EC Directive 2002/95/EC on Restrictions on Certain Hazardous Substances in Electric and Electronic Equipment (ROHS) stipulates in article 4 that electric and electronic articles may not contain polybrominated biphenyls from July 2006.

The issue of Hexabromobiphenyl in waste is addressed at the European level in Regulation 850/2004/EC. As amended by regulation 1195/2006/EC Hexabromobiphenyl in wastes has to be destroyed if concentration limits of 50 mg/kg are exceeded.

At the national level legal control actions taken have been reported by Germany, Canada and the USA. In Canada, Polybrominated Biphenyls appear on Schedule 1 (List of Toxic Substances) of CEPA 1999, and are subject to prohibitions on their use. In addition Polybrominated Biphenyls appear on Schedule 3, Part 1 (Export Control List – Prohibited Substances) of CEPA 1999, effectively prohibiting their export, except for the purpose of destroying the substance. In the USA Hexabromobiphenyl is subject to a TSCA Significant New Use Rule which would require notification to EPA prior to re-initiation of manufacture or import for any use.

Concerning chemical substitutes and technical alternatives reported data (although not specifically related to Hexabromobiphenyl but as overall alternatives to brominated flame retardants show that there are less hazardous alternatives e.g. aluminium trihydroxide. However, it cannot be disregarded that more harmful substances might be among the alternatives compared to the group of brominated flame retardants as such (e.g. halogenated phosphorus and partially non-halogenated phosphorus compounds). Providing guidance on criteria for selecting alternatives to Hexabromobiphenyl should be part of the risk management strategy for Hexabromobiphenyl elimination. This would help discourage substitution of Hexabromobiphenyl with other harmful substances. Criteria should include a non-hazardous synthetic pathway; minimum human and environmental toxicity; minimum release during product use; minimum formation of hazardous substances during incineration or burning; and the ability to be recycled or degrade into a non-hazardous substance.

As production of Hexabromobiphenyl has ceased some decades ago, availability of alternatives, efficacy and cost implications do not constitute a problem. Based on the same background significant negative impacts of listing of Hexabromobiphenyl in Annex A on society are not expected.

The Persistent Organic Pollutants Review Committee, has decided, in accordance with paragraph 7 (a) of Article 8 of the Convention, given the fact that Hexabromobiphenyl is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects such that global action is warranted.

A beneficial effect could be expected in case of currently unknown production in any part of the world, if management and disposal of potentially remaining stocks (e.g. coated cables, equipment exceeding average life time) would be further regulated and reintroduction of Hexabromobiphenyl would be prevented on a global scale.

51 For use in article 5(1) a maximum concentration of 0.1% by weight in homogenous material of PBB shall be tolerated
52 Danish Environmental Protection Agency, Brominated flame retardants: Substance flow analysis and assessment of alternatives, June 1999
Having evaluated the risk profile corresponding to Hexabromobiphenyl, and having prepared its risk management evaluation, the Committee concludes that this chemical is likely, as a result of long-range environmental transport, to lead to significant adverse effects on human health and/or the environment, such that global action is warranted. Although Hexabromobiphenyl is not known to be produced and used anymore, it is important to prevent re-introduction of this substance.

Therefore, in accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends the Conference of the Parties to the Stockholm Convention to consider listing and specifying the related control measures of Hexabromobiphenyl in Annex A. As no remaining production or uses of Hexabromobiphenyl have been identified, listing of Hexabromobiphenyl in Annex A without any specific exemptions is possible.
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- (USEPA, 1985)

- (USEPA, 2005)

- (WHO, 1990)

- (WHO, 1998)
### Table A.1 Concentrations of hexabromobiphenyl (PBB 153) in Arctic predators.

<table>
<thead>
<tr>
<th>Year of sampling</th>
<th>Location</th>
<th>Species</th>
<th>Tissue</th>
<th>Concentration µg/kg lipid</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999-2002</td>
<td>East Greenland</td>
<td>Polar bear ([<em>Ursus maritimus</em>] $^1$)</td>
<td>Blubber</td>
<td>33-44</td>
</tr>
<tr>
<td>1998</td>
<td>Faroe Islands</td>
<td>Fulmar ([<em>Fulmarus glacialis</em>] $^1$)</td>
<td>Fat</td>
<td>16-26</td>
</tr>
<tr>
<td>2001</td>
<td>Faroe Islands</td>
<td>Pilot whale ([<em>Globicephala melas</em>] $^1$)</td>
<td>Blubber</td>
<td>8.7-17</td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>Arctic Ocean</td>
<td>Guillemot ([<em>Uria aalge</em>] $^2$)</td>
<td>Muscle</td>
<td>50 $^6$</td>
</tr>
<tr>
<td>2002</td>
<td>East Greenland</td>
<td>Ringed seal ([<em>Phoca hispida</em>] $^1$)</td>
<td>Blubber</td>
<td>0.34-0.42</td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>Svalbard</td>
<td>Ringed seal ([<em>Phoca hispida</em>] $^2$)</td>
<td>Blubber</td>
<td>4 $^6$</td>
</tr>
<tr>
<td>1981</td>
<td>Svalbard</td>
<td>Ringed seal ([<em>Phoca hispida</em>] $^3$)</td>
<td>Blubber</td>
<td>0.42</td>
</tr>
<tr>
<td>&lt; 1988</td>
<td>Svalbard</td>
<td>Seal sp. $^9$</td>
<td>? (mean)</td>
<td>0.8</td>
</tr>
<tr>
<td>1998</td>
<td>East Greenland</td>
<td>Minke whale ([<em>Balaenoptera acutorostrata</em>] $^1$)</td>
<td>Blubber</td>
<td>0.56-1.2</td>
</tr>
<tr>
<td>1999-2001</td>
<td>Barents Sea</td>
<td>Arctic char ([<em>Salvelinus alpinus</em>] $^5$)</td>
<td>Muscle</td>
<td>n.d.-52</td>
</tr>
<tr>
<td>1986</td>
<td>Lapland</td>
<td>Whitefish ([<em>Coregonus sp.</em>] $^2$)</td>
<td>Muscle</td>
<td>0.29</td>
</tr>
<tr>
<td>2002</td>
<td>East Greenland</td>
<td>Shorthorn sculpin ([<em>Myoxocephalus scorpius</em>] $^1$)</td>
<td>Liver</td>
<td>n.d.</td>
</tr>
<tr>
<td>2002</td>
<td>West Greenland</td>
<td>Shorthorn sculpin ([<em>Myoxocephalus scorpius</em>] $^1$)</td>
<td>Liver</td>
<td>n.d.</td>
</tr>
</tbody>
</table>

n.d. = Not detected. Limits of detection are not well described in the references.

1: Vorkamp *et al.*, 2004,
2: Jansson *et al.*, 1987,
3: Jansson *et al.*, 1993,
4: Krüger, 1988 (Quoted from EHC 152),
6: FireMaster$^R$ BP-6
Table A.2  Concentrations of hexabromobiphenyl (PBB 153) in biota, collected in subarctic and temperate regions outside the vicinity of Michigan.

<table>
<thead>
<tr>
<th>Year of sampling</th>
<th>Location</th>
<th>Species</th>
<th>Tissue</th>
<th>Concentration µg/kg lipid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Aquatic species</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1979-85</td>
<td>Baltic Sea</td>
<td>Grey seal (Halichoerus grypus)</td>
<td>Blubber</td>
<td>26</td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>Baltic Sea</td>
<td>Harbour seal (Phoca vitulina)</td>
<td>Blubber</td>
<td>20</td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>~North Sea</td>
<td>Harbour seal (Phoca vitulina)</td>
<td>Blubber</td>
<td>3</td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>Baltic Sea</td>
<td>Guillemot (Uria aalge)</td>
<td>Muscle</td>
<td>160</td>
</tr>
<tr>
<td>1987-88</td>
<td>US mid Atlantic</td>
<td>Bottlenose dolphin (Tursiops truncatus)</td>
<td></td>
<td>14-20</td>
</tr>
<tr>
<td>&lt; 1999</td>
<td>North Sea</td>
<td>Whitebeaked dolphin (Lagenorhynchus albirostris)</td>
<td></td>
<td>13 (wwt)</td>
</tr>
<tr>
<td>1987</td>
<td>S. Sweden</td>
<td>Arctic char (Salvelinus alpinus)</td>
<td>Muscle</td>
<td>0.42</td>
</tr>
<tr>
<td>1986</td>
<td>Bothnian Bay</td>
<td>Herring (Clupea harengus)</td>
<td>Muscle</td>
<td>0.092</td>
</tr>
<tr>
<td>1987</td>
<td>Baltic Proper</td>
<td>Herring (Clupea harengus)</td>
<td>Muscle</td>
<td>0.16</td>
</tr>
<tr>
<td>1987</td>
<td>Skagerak</td>
<td>Herring (Clupea harengus)</td>
<td>Muscle</td>
<td>0.27</td>
</tr>
<tr>
<td>&lt; 1988</td>
<td>Germany</td>
<td>River fish (average)</td>
<td></td>
<td>0.60</td>
</tr>
<tr>
<td>&lt; 1988</td>
<td>Baltic Sea</td>
<td>Fish</td>
<td></td>
<td>2.39</td>
</tr>
<tr>
<td>&lt; 1988</td>
<td>North Sea</td>
<td>Fish</td>
<td></td>
<td>1.31</td>
</tr>
<tr>
<td>1997</td>
<td>USA, Great Lakes</td>
<td>Lake trout (Salvelinus nanaycush) (range of means)</td>
<td>Whole fish</td>
<td>0.19-2.08</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Predatory birds</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1987</td>
<td>Baltic Sea</td>
<td>White tailed sea eagle (Haliaeetus albicilla)</td>
<td>Muscle</td>
<td>280</td>
</tr>
<tr>
<td>1977</td>
<td>USA, 29 states</td>
<td>Baid eagle (Haliaeetus leucocephalus)</td>
<td>Carcass</td>
<td>&lt; 0.03 – 0.07 (wwt?)</td>
</tr>
<tr>
<td>1977</td>
<td>USA, 29 states</td>
<td>Baid eagle (Haliaeetus leucocephalus)</td>
<td>Brain</td>
<td>&lt; 0.03 – 0.05 (wwt?)</td>
</tr>
<tr>
<td>1982-86</td>
<td>S. Sweden</td>
<td>Osprey (Pandion haliaetus), corpses</td>
<td>Muscle</td>
<td>22</td>
</tr>
<tr>
<td>2003-2004</td>
<td>Belgium</td>
<td>7 species of predatory birds, corpses (range of medians)</td>
<td>Muscle</td>
<td>2-35</td>
</tr>
<tr>
<td>2003-2004</td>
<td>Belgium</td>
<td>7 species of predatory birds, corpses (range of medians)</td>
<td>Liver</td>
<td>2-43</td>
</tr>
<tr>
<td>1998-2000</td>
<td>Belgium</td>
<td>Little owl (Athene noctua)</td>
<td>Unhatched eggs</td>
<td>1-6</td>
</tr>
<tr>
<td>1991-2002</td>
<td>Norway</td>
<td>6 species of predatory birds (range of medians)</td>
<td>Unhatched eggs</td>
<td>0.2-9.4 µg/kg wwt</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Terrestrial herbivores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1986</td>
<td>N. Sweden</td>
<td>Reindeer (Rangifer tarandus)</td>
<td>Suet (fat)</td>
<td>0.037</td>
</tr>
</tbody>
</table>

n.d. = Not detected. Limits of detection are not well described in the references.

Table A.3. Summary of key toxicological studies on hexabromobiphenyl.

<table>
<thead>
<tr>
<th>Species (test material)</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat Fischer 344/N (FF-1)</td>
<td>Short-term/acute toxicity, 14-day repeat dose, 5 single daily doses per week</td>
<td>Body weight loss, emaciation, hepatotoxicity, renal &amp; adrenal changes, atrophy of thymus; necrosis of splenic lymphoblasts</td>
<td>1000 mg/kg/day (LOAEL)</td>
<td>Gupta and Moore 1979 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat</td>
<td>Short-term/acute toxicity10 day repeat dose gavage study</td>
<td>decreased thyroid serum T4 hormones</td>
<td>3 mg/kg bw/day (LOAEL) 1 mg/kg bw/day (NOAEL)</td>
<td>Allen-Rowlands et al. 1981 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat, Sprague Dawley (BP-6)</td>
<td>30-day dietary feeding study</td>
<td>increased number and decreased size of thyroid follicles</td>
<td>0.05 mg/kg/day (LOAEL)</td>
<td>Akoso et al. 1982 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Mouse B6C3F1 (FF-1)</td>
<td>Short-term/acute toxicity, 14-day repeat dose, 5 single daily doses per week</td>
<td>Hepatocyte enlargement and single-cell necrosis</td>
<td>0.3 mg/kg bw/day (NOAEL)</td>
<td>Gupta et al. 1981 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Guinea Pig (PBB not specified)</td>
<td>30-day dietary feeding study</td>
<td>vacuolation and fatty changes in liver</td>
<td>0.04 mg/kg bw/day</td>
<td>Sleight and Sanger 1976, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Balb/c) Mouse (BP-6)</td>
<td>Short-term/acute toxicit, 10 day oral dietary study</td>
<td>suppressed antibody-mediated response to SRBC, thymic atrophy</td>
<td>130 mg/kg bw/day (LOAEL)</td>
<td>Fraker and Aust 1978, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat Fischer 344/N (FF-1)</td>
<td>6 month gavage study, 5 single daily doses per week</td>
<td>decreased lymphoproliferative responses and decreased delayed hypersensitivity responses</td>
<td>3 mg/kg bw/day (LOAEL)</td>
<td>Luster et al. 1980 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rhesus Monkey (FF-1)</td>
<td>25-50 wk dietary feeding study</td>
<td>34% weight loss in adult male, 0% weight gain in juvenile, proliferation of mucosal cells, chronic inflammation, severe ulcerative colitis, alopecia, keratinization of hair follicles and sebaceous glands, clinical chemical and hepatic changes</td>
<td>0.73 mg/kg bw/day (LOAEL, males)</td>
<td>Allen et al. 1978; Lambrecht et al. 1978 (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat, Sprague Dawley (BP-6)</td>
<td>7 month dietary feeding study</td>
<td>decreased thyroid serum T3 and T4 hormones</td>
<td>0.45 mg/kg bw/day (LOAEL)</td>
<td>Byrne et al. 1987; (as quoted in US ATSDR, 2004).</td>
</tr>
</tbody>
</table>

Note: FF-1 and BP-6 in column 1 refer to FireMaster(R) FF-1 and FireMaster(R) BP-6, the PBBs used in the toxicity study described.
<table>
<thead>
<tr>
<th>Species (test material)</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat Fischer 344/N (FF-1)</td>
<td>25 wk gavage study, 5 single daily doses per week</td>
<td>gastric ulcers, decreased serum thyroid T4 hormone, hepatic, haematological disorders, thymic atrophy, progressive nephropathy</td>
<td>0.3 mg/kg bw/day (LOAEL), 0.1 mg/kg bw/day (NOAEL)</td>
<td>NTP 1983, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat Sprague-Dawley Holtzman (FF-1)</td>
<td>4 week gavage study, 5 single daily doses per week</td>
<td>decreased motor activity</td>
<td>6 mg/kg bw/day (LOAEL), 3 mg/kg bw/day (LOAEL)</td>
<td>Geller et al. 1979, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat, Sprague Dawley (BP-6)</td>
<td>6 month gavage study, 5 single daily doses per week</td>
<td>delayed acquisition of locomotion and reduced open field activity in offspring</td>
<td>2 mg/kg bw/day (LOAEL), 0.2 mg/kg bw/day (NOAEL)</td>
<td>Henck et al. 1994, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Monkey, Rhesus (FF-1)</td>
<td></td>
<td>increased menstrual cycle duration in 4/7; implantation bleeding in 2/7. 1/7 fetuses were aborted, 1/7 fetuses stillborn, 12% decreased birth weight and 22% decreased postnatal weight gain in 4/7 survivors</td>
<td>0.012 mg/kg bw/day (LOAEL)</td>
<td>Lambrecht et al. 1978; Allen et al. 1978; 1979, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat, Wistar (BP-6)</td>
<td>15-day reproductive toxicity study, dosing between gestational day 0-14</td>
<td>no implantations in 2/5 rats</td>
<td>28.6 mg/kg bw/day (LOAEL), 14.3 mg/kg bw/day (NOAEL)</td>
<td>Beaudoin 1979, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Rat, Sprague Dawley</td>
<td>Gavage study in pregnant rats, dosing between gestional day 7-15</td>
<td>Reproductive: Delayed vaginal opening in pups</td>
<td>0.04 mg/kg bw/day (NOAEL)</td>
<td>Harris et al. (1978) (as quoted in BKH Final Report 2000)</td>
</tr>
<tr>
<td>Rat, Sprague Dawley (BP-6)</td>
<td>40 day dietary feeding study</td>
<td>Reproductive deficits in learning behavior in offspring, 6 months after prenatal and lactational exposure</td>
<td>0.2 mg/kg bw/day (LOAEL)</td>
<td>Henck and Rech 1986, (as quoted in US ATSDR, 2004).</td>
</tr>
</tbody>
</table>

Note: FF-1 and BP-6 in column 1 refer to FireMaster(R) FF-1 and FireMaster(R) BP-6, the PBBs used in the toxicity study described.
Table A.3 (continued) Summary of key toxicological studies on hexabromobiphenyl.

<table>
<thead>
<tr>
<th>Species (test material)</th>
<th>Study type</th>
<th>Effect</th>
<th>LOAEL/NOAEL</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rat, Fischer 344/N (FF-1)</td>
<td>6 month gavage study, 5 single daily doses per week dosages of 0, 0.1, 0.3, 1, 3, or 10 mg/kg/day</td>
<td>hepatocellular adenoma and carcinoma, cholangiocarcinoma (females only)</td>
<td>3 mg/kg bw/day (LOAEL)</td>
<td>NTP 1983, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Mice B6C3F1 (FF-1)</td>
<td>6 month gavage study, 5 single daily doses per week dosages of 0, 0.1, 0.3, 1, 3, or 10 mg/kg/day</td>
<td>hepatocellular adenoma and carcinoma</td>
<td>10 mg/kg bw/day (LOAEL)</td>
<td>NTP 1983, (as quoted in US ATSDR, 2004)</td>
</tr>
<tr>
<td>Mice B6C3F1 (FF-1)</td>
<td>In utero and post partum exposure from Gd 0-ppd 56</td>
<td>hepatocellular adenoma and carcinoma in offspring</td>
<td>1.5 mg/kg bw/day (LOAEL) 0.15 mg/kg bw/day (NOAEL)</td>
<td>NTP 1992, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Humans</td>
<td>Females accidentally exposed in the Michigan incident</td>
<td>relationship between serum PBBs and risk of breast cancer</td>
<td>relationship between serum PBBs of &gt; 2 ppb and risk of breast cancer when compared with the reference group (&lt;2 ppb),</td>
<td>Henderson et al. 1995, (as quoted in US ATSDR, 2004).</td>
</tr>
<tr>
<td>Humans</td>
<td>Females accidentally exposed in the Michigan incident</td>
<td>Possible disturbance in ovarian function as indicated by menstrual cycle length and bleed length</td>
<td></td>
<td>Davis et al., 2005</td>
</tr>
<tr>
<td>Humans</td>
<td>Offspring of females accidentally exposed in the Michigan incident</td>
<td>breastfed girls exposed to high levels of PBB in utero had an earlier age at menarche</td>
<td>Effects at &gt; or =7 ppb in breast milk</td>
<td>Blanck et al., 2000, (as quoted in US ATSDR, 2004)</td>
</tr>
</tbody>
</table>

Note: FF-1 and BP-6 in column 1 refer to FireMaster(R) FF-1 and FireMaster(R) BP-6, the PBBs used in the toxicity study described.
### ANNEX B

**HEXABROMOBIPHENYL ISOMERS**

<table>
<thead>
<tr>
<th>IUPAC Number</th>
<th>Name</th>
<th>CAS Registry number</th>
</tr>
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<tr>
<td>128</td>
<td>Hexabromobiphenyl</td>
<td>36355-01-8</td>
</tr>
<tr>
<td>129</td>
<td>2,2',3,3',4,4' hexabromobiphenyl</td>
<td>82865-89-2</td>
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<td>130</td>
<td>2,2',3,3',4,5 hexabromobiphenyl</td>
<td>82865-90-5</td>
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<tr>
<td>131</td>
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<td></td>
</tr>
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<td>132</td>
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<td>119264-50-5</td>
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<td>55066-76-7</td>
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<td>2,2',3,4,4',5' hexabromobiphenyl</td>
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<td>119264-52-7</td>
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</tr>
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<td>69278-59-7</td>
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<td>84303-47-9</td>
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<td>158</td>
<td>2,3,3',4,4',6 hexabromobiphenyl</td>
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<td>163</td>
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<td></td>
</tr>
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</tr>
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<td>165</td>
<td>2,3,3',5,5,6 hexabromobiphenyl</td>
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<td>166</td>
<td>2,3,4,4,5,6 hexabromobiphenyl</td>
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</tr>
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<td>167</td>
<td>2,3,4,4,5,5' hexabromobiphenyl</td>
<td>67888-99-7</td>
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<tr>
<td>168</td>
<td>2,3,4,4,5,6 hexabromobiphenyl</td>
<td>84303-48-0</td>
</tr>
<tr>
<td>169</td>
<td>3,3',4,4',5,5' hexabromobiphenyl</td>
<td>60044-26-0</td>
</tr>
</tbody>
</table>

(US ATSDR (2004)\(^{55}\)

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\(^{53}\) Ballschmiter and Zell 1980

\(^{54}\) From EHC 152 (IPCS, 1994).

\(^{55}\) Note: the US ATSDR List does not include the two CAS numbers included in EHC 192 1997
## B.6. Lindane – SUMMARY

### SUMMARY

**6. Lindane**

Draft Risk Management Evaluation May 2007


Risk Profile UNEP/POPS/POPRC.2/17/Add4


<table>
<thead>
<tr>
<th>Composition</th>
<th>Lindane is the common name for the gamma isomer of 1,2,3,4,5,6-hexachlorocyclohexane (HCH). It is one of 5 stable HCH isomers in technical HCH. The gamma isomer is the only isomer showing strong insecticidal properties. The production of lindane is inefficient as for each ton of Lindane (gamma isomer) obtained; approximately 6-10 tons of other isomers are also obtained (IHPA, 2006).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Lindane has been used as a broad-spectrum insecticide for seed and soil treatment, foliar applications, tree and wood treatment and against ectoparasites in both veterinary and human applications. In the last years the production of lindane has rapidly decreased and it appears that only Romania and India are current producing countries. If the estimate of global usage of lindane of 600,000 tons between 1950 and 2000 is accurate, the total amount of possible residuals (if it is assumed that a mean value of 8 tons of waste isomers are obtained per ton of lindane produced) amounts to possibly 4.8 million tons of HCH residuals that could be present worldwide giving an idea of the extent of the environmental contamination problem (IHPA, 2006). Air releases of lindane can occur during the agricultural use or aerial application of this insecticide, as well as during manufacture or disposal. Also, lindane can be released to air through volatilization after application (Shen et al., 2004). Evaporative loss to air from water is not considered significant due to lindane's relatively high water solubility (WHO/Europe, 2003).</td>
</tr>
<tr>
<td>Releases</td>
<td>Considering every ton of lindane produced generates approximately 6 - 10 tons of other HCH isomers, a considerable amount of residues was generated during the manufacture of this insecticide. For decades, the waste isomers were generally disposed of in open landfills like fields and other disposal sites near the HCH manufacturing facilities. After disposal, degradation, volatilization, and run off of the waste isomers occurred (USEPA, 2006). If the estimate of global usage of lindane of 600,000 tons between 1950 and 2000 is accurate, the total amount of possible residuals (if it is assumed that a mean value of 8 tons of waste isomers are obtained per ton of lindane produced) amounts to possibly 4.8 million tons of HCH residuals that could be present worldwide giving an idea of the extent of the environmental contamination problem (IHPA, 2006). Air releases of lindane can occur during the agricultural use or aerial application of this insecticide, as well as during manufacture or disposal. Also, lindane can be released to air through volatilization after application (Shen et al., 2004). Evaporative loss to air from water is not considered significant due to lindane’s relatively high water solubility (WHO/Europe, 2003).</td>
</tr>
<tr>
<td>Fate</td>
<td>Once released into the environment, lindane can partition into all environmental media. Hydrolysis and photolysis are not considered important degradation pathways and reported half-lifes in air, water and soil are: 2.3 days, 3-300 days and up to 2 to 3 years, respectively. A half-life of 96 days in</td>
</tr>
</tbody>
</table>
Lindane can bio-accumulate easily in the food chain due to its high lipid solubility and can bio-concentrate rapidly in microorganisms, invertebrates, fish, birds and mammals. The bioconcentration factors in aquatic organisms under laboratory conditions ranged from approximately 10 up to 4220 under field conditions, the bioconcentration factors ranged from 10 up to 2600. Although lindane may bioconcentrate rapidly, bio-transformation, depuration and elimination are also relatively rapid, once exposure is eliminated.

<table>
<thead>
<tr>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatotoxic, immunotoxic, reproductive and developmental effects have been reported for lindane in laboratory animals. The US EPA has classified lindane in the category of “Suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential”. The most commonly reported effects associated with oral exposure to gamma-HCH are neurological. Most of the information is from case reports of acute gamma-HCH poisoning. Seizures and convulsions have been observed in individuals who have accidentally or intentionally ingested lindane in insecticide pellets, liquid scabicide or contaminated food (WHO/Europe, 2003). Lindane is highly toxic to aquatic organisms and moderately toxic to birds and mammals following acute exposures. Chronic effects to birds and mammals measured by reproduction studies show adverse effects at low levels such as reductions in egg production, growth and survival parameters in birds, and decreased body weight gain in mammals, with some effects indicative of endocrine disruption.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindane can be found in all environmental compartments, and levels in air, water, soil sediment, aquatic and terrestrial organisms and food have been measured worldwide. Humans are therefore being exposed to lindane as demonstrated by detectable levels in human blood, human adipose tissue and human breast milk in different studies in diverse countries. Exposure of children and pregnant women to lindane are of particular concern. Gamma-HCH has been found in human maternal adipose tissue, maternal blood, umbilical cord blood and breast milk. Lindane has also been found to pass through the placental barrier. Direct exposure from the use of pharmaceutical products for scabies and lice treatment should be of concern. Exposure from food sources is possibly of concern for high animal lipid content diets and subsistence diets of particular ethnic groups (USEPA, 2006 and CEC, 2005). Occupational exposure at manufacturing facilities should be of concern, because lindane production implies worker exposure to other HCH isomers as well, for example the alpha isomer is considered to be a probable human carcinogen (USEPA, 2006).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Status</th>
</tr>
</thead>
</table>
| Lindane is listed as a “substance scheduled for restrictions on use” in Annex II of the 1998 Protocol on Persistent Organic Pollutants of the Convention on Long-Range Transboundary Air Pollution. This means that products in which at least 99% of the HCH isomer is in the gamma form (i.e. lindane, CAS: 58-89-9) are restricted to the following uses: 1. Seed treatment. 2. Soil applications directly followed by incorporation into the topsoil surface layer 3. Professional remedial and industrial treatment of lumber, timber and logs. 4. Public health and veterinary topical insecticide. 5. Non-aerial application to tree seedlings, small-scale lawn use, and indoor and outdoor use for nursery stock and ornamentals. 6. Indoor industrial and residential applications. All restricted uses of lindane shall be reassessed under the Protocol no later than two years after the date of entry into force. The Protocol entered into force on October 23th, 2003. Lindane, as well as the mixture of HCH isomers, is listed in Annex III of the Rotterdam Convention on the Prior Informed Consent Procedure as “chemicals subject to the prior informed consent procedure”. Hexachlorocyclohexane isomers, including Lindane, the gamma isomer, are included in the List of Chemicals for Priority Action (Updated 2005) under the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic. HCH (including lindane) is listed as a Level II substance in
the Great Lakes Binational Toxics Strategy between the United States and Canada, which means that one of the two countries has grounds to indicate its persistence in the environment, potential for bioaccumulation and toxicity. Lindane is also listed under the European Water Framework Directive. This Directive is a piece of water legislation from the European Community. It requires all inland and coastal water bodies to reach at least “good status” by 2015. Lindane is one of the listed priority hazardous substances for which quality standards and emission controls will be set at EU level to end all emissions within 20 years. Lindane is banned for use in 52 countries, restricted or severely restricted in 33 countries, not registered in 10 countries, and registered in 17 countries (CEC, 2006).

**Alternatives**

Chemical and non-chemical alternatives for the agricultural, veterinary and pharmaceutical uses of lindane in the United States, Canada and Mexico have been reviewed in the North American Regional Action Plan on Lindane and Other HCH Isomers developed by the North American Commission for Environmental Cooperation (CEC, 2006). Among cultural methods currently known to effectively prevent harm to seeds and crops are: Crop rotation (alfalfa, soybeans and clover), where small grains need to be rotated with a non-host species every year to reduce the severity of infestation and maintain low levels of pests; Site selection and monitoring in order to determine if wireworms are present; Fallowing, starving wireworms by allowing the area to fallow for a few years before planting; Re-seeding with resistant crops such as buckwheat or flax; Timing of seeding and planting, trying to plant in warm, dry conditions, usually later in the season for small grains where larvae are deeper in the soil and giving seedlings a greater chance of survival; Shallow cultivation to starve hatchlings, expose eggs for predation and damage larvae; and Soil packing to impede wireworm travel (CEC, 2006). Biological methods are also considered as non-chemical alternatives to lindane. Current research at Pacific Agri-Food Research Centre, in Canada is examining the use of *Metarhizium anisopliae*, an insect fungal pathogen to control wireworm. Additional biological control methods employed in Costa Rica include *Trichodama* spp, *Piper aduncum*, *Trichogram* wasps, and *Bacillus thuringiensis* (Annex F information provided by IPEN, 2007).
Candidate for POPs List

LINDANE

(gamma hexachlorocyclohexane)
Introduction

Lindane, the common name for the gamma isomer of hexachlorocyclohexane, is a white crystalline solid, stable in light, heat, air, carbon dioxide and strong acids. Technical hexachlorocyclohexane contains mainly five isomers: alpha hexachlorocyclohexane (53%–70%), beta hexachlorocyclohexane (3%–14%), gamma hexachlorocyclohexane (11%–18%), delta hexachlorocyclohexane (6%–10%) and epsilon hexachlorocyclohexane (3%–5%). The mixture of isomers was largely used as an inexpensive insecticide, but as the gamma isomer is the only isomer showing strong insecticidal properties, it was purified from the mixture and commercialized under the name Lindane (technical grade purity >99%).

Lindane (and hexachlorocyclohexane) is listed as a persistent organic pollutant with restricted uses under the Protocol on Persistent Organic Pollutants to the Convention on Long-range Transboundary Air Pollution under the United Nations Economic Commission for Europe. Its restricted uses have recently undergone a mandatory review under the Protocol.

1. Identification of the chemical

1.1. Names and registry numbers

**CAS chemical name:**
gamma, 1,2,3,4,5,6-hexachlorocyclohexane

**Synonyms/abbreviations:**
gamma benzene hexachloride; gamma-BHC

**Trade names:**

**CAS registry number:**
58-89-9

1.2. Structure of alpha, beta, gamma, delta and epsilon HCH isomers
Modified from Buser et al., 1995.
Molecular formula: 
C₆H₆Cl₆

Molecular weight: 
290.83

The term “benzene hexachloride (BHC)” is also commonly used for HCH, but according to IUPAC rules this designation is incorrect. Nevertheless the term is used and therefore, gamma-BHC also designates lindane. In the present document, lindane refers to at least 99% pure gamma-HCH and the BHC term is not used.

1.3. Physical and Chemical properties of gamma-HCH

<table>
<thead>
<tr>
<th>Physical state</th>
<th>Crystalline solid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melting point</td>
<td>112.5 °C</td>
</tr>
<tr>
<td>Boiling point at 760 mmHg</td>
<td>323.4 °C</td>
</tr>
<tr>
<td>Vapor pressure at 20°C</td>
<td>4.2x10⁻³ mmHg</td>
</tr>
<tr>
<td>Henry's Law constant at 25°C</td>
<td>3.5x10⁻⁶ atm m³/mol</td>
</tr>
</tbody>
</table>

ATSDR, 2005

Lindane is the common name for the gamma isomer of 1,2,3,4,5,6-hexachlorocyclohexane (HCH). Technical HCH is an isomeric mixture that contains mainly five forms differing only by the chlorine atoms orientation (axial or equatorial positions) around the cyclohexane ring. The five principal isomers are present in the mixture in the following proportions: alpha-hexachlorocyclohexane (53%–70%) in two enantiomeric forms ((+)-alpha-HCH and (-)-alpha-HCH), beta-hexachlorocyclohexane (3%–14%), gamma-hexachlorocyclohexane (11%–18%), delta-hexachlorocyclohexane (6%–10%) and epsilon-hexachlorocyclohexane (3%–5%). The gamma isomer is the only isomer showing strong insecticidal properties.

2. Persistence

Lindane has a half-life of 2.3 to 13 days in air, 30 to 300 days in water, 50 days in sediments and two years in soil. It is stable to light, high temperatures and acid but it can be hydrolysed at high pH. Lindane degrades very slowly by microbial action. Lindane is more water-soluble and volatile than other chlorinated organic chemicals, which explains why it is found in all environmental media (water/snow, air, soil/sediments).

3. Bioaccumulation

Although lindane has the potential to bioaccumulate easily in the food chain as a result of its high lipid solubility, biotransformation and elimination are also relatively rapid. Lindane has a logKow of 3.5 and a log bioconcentration factor of 2.26 to 3.85. Nevertheless, lindane can be found in seabirds, fish and mammals in the Arctic and in other regions of the world.

4. Potential for long-range environmental transport

Lindane is a volatile compound (vapour pressure 3.83 x 10⁻³ Pa) which can undergo vaporization and condensation cycles and can be found in remote regions where it is not used, such as the Arctic. Estimated annual loading of lindane to the Arctic is 13,000 kg per year.

The transport and deposition processes have led to the accumulation of lindane in all environmental niches in the Arctic. Lindane is measured consistently in Arctic air, sea water and freshwater bodies. Lindane and alpha hexachlorocyclohexane have made up about 75 per cent of the organochlorine compounds measured in snowpack in the Canadian Arctic.
5. Adverse effects

Lindane is moderately to highly toxic in acute rat studies, with an oral LD$_{50}$ ranging from 55 to 480 mg/kg body weight. Acute effects include central nervous system excitation, convulsions, respiratory failure, pulmonary oedema and dermatitis.

Several animal studies indicate an association between chronic lindane exposure and aplastic anaemia, convulsions, liver and kidney effects, reduced ability to fight infection and injury to testes and ovaries. The International Agency for Research on Cancer has classified Lindane as carcinogenic in mice.

The most common human exposure pathway to Lindane is through food. A direct correlation exists between food intake, especially fish, meat and dairy products, and marine mammals, and lindane concentrations in body fat and human milk.

Lindane is reported to be highly toxic to some fish and other aquatic species.

REASONS FOR CONCERN

Lindane is persistent and is frequently found in environmental compartments. Due to its physicochemical properties it has the potential to be transported long distances. It is ecotoxic and has acute and chronic adverse effects in humans. Pharmaceutical uses, mainly in children, should be of particular concern.

Adding to these data on the harmful effects of Lindane to human health and the environment, is the fact that in the production and purification process to obtain 99% pure gamma hexachlorocyclohexane, for every ton of Lindane produced there are 6–10 metric tonnes of the other isomers that must be disposed of or otherwise managed. Lindane being the only isomer in the mixture that has insecticidal properties, there is very limited to no commercial value for the other isomers obtained. Because of this waste isomer problem, the production of hexachlorocyclohexane/Lindane has been a worldwide problem for years. The International Hexachlorocyclohexane and Pesticide Forum exists in order to bring together experts to solve the problems associated with the clean-up of former hexachlorocyclohexane/lindane production sites.

Other hexachlorocyclohexane isomers can be as toxic, persistent and contaminant as lindane, or even more so. The continued use of lindane in the world is causing this important polluting source. Global action is therefore needed to halt the pollution caused worldwide by lindane and its production.”

Data Source

Overview

International initiatives on Lindane include the Protocol on Persistent Organic Pollutants of the Convention on Long Range Transboundary Air Pollution; the Rotterdam Convention; the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic, the Great Lakes Binational Toxics Strategy between the United States and Canada, and a North American Regional Action Plan on Lindane and Other Hexachlorocyclohexane Isomers under the Commission for Environmental Cooperation between Canada, United States and Mexico.

For each ton of lindane produced, around 6-10 tons of other isomers are also obtained. In the last years the production of lindane has rapidly decreased and it appears that only Romania and India are current producing countries. Lindane has been used as a broad-spectrum insecticide for seed and soil treatment, foliar applications, tree and wood treatment and against ectoparasites in both veterinary and human applications.

Once released into the environment, lindane can partition into all environmental media. Hydrolysis and photolysis are not considered important degradation pathways and reported half-lifes in air, water and soil are: 2.3 days, 3-300 days and up to 2 to 3 years, respectively. A half-life of 96 days in air has also been estimated.

Lindane can bio-accumulate easily in the food chain due to its high lipid solubility and can bio-concentrate rapidly in microorganisms, invertebrates, fish, birds and mammals. The bioconcentration factors in aquatic organisms under laboratory conditions ranged from approximately 10 up to 4220 under field conditions, the bioconcentration factors ranged from 10 up to 2600. Although lindane may bioconcentrate rapidly, bio-transformation, depuration and elimination are also relatively rapid, once exposure is eliminated.

Many studies have reported lindane residues throughout North America, the Arctic, Southern Asia, the Western Pacific, and Antarctica. HCH isomers, including lindane, are the most abundant and persistent organochlorine contaminants in the Arctic where they have not been used, pointing at evidence of their long-range transport.

The hypothesis that isomerization of gamma HCH to alpha HCH in air emerged as a possible explanation for higher than expected alpha HCH/gamma HCH ratios in the Arctic. However no conclusive experimental evidence of isomerization taking place in air has been produced to date. Also, although there is evidence that bioisomerization of lindane can take place through biological degradation, it seems that this process may play an insignificant role in the overall degradation of gamma-HCH.

Lindane can be found in all environmental compartments, and levels in air, water, soil sediment, aquatic and terrestrial organisms and food have been measured worldwide. Humans are therefore being exposed to lindane as demonstrated by detectable levels in human blood, human adipose tissue and human breast milk in different studies in diverse countries. Exposure of children and pregnant women to lindane are of particular concern.

Hepatotoxic, immunotoxic, reproductive and developmental effects have been reported for lindane in laboratory animals. The US EPA has classified lindane in the category of "Suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential". Lindane is highly toxic to aquatic organisms and moderately toxic to birds and mammals following acute exposures. Chronic effects to birds and mammals measured by reproduction studies show adverse effects at low levels such as reductions in egg production, growth and survival parameters in birds, and decreased body weight gain in mammals, with some effects indicative of endocrine disruption.

These findings and the evidence of its long range transport, as well as the fact that lindane is currently the object of local and global action initiatives, that also include thorough analysis and selection procedures, should be sufficient to warrant global action under the Stockholm Convention.
Summary information relevant to the risk profile

1. SOURCES

a) Production, trade, stockpiles

The manufacture of technical-HCH involves the photochlorination of benzene, which yields a mixture of five main isomers. This mixture of isomers is subject to fractional crystallization and concentration to produce 99% pure lindane, with only a 10-15 percent yield. The production of lindane is therefore inefficient as for each ton of lindane (gamma isomer) obtained, approximately 6-10 tons of other isomers are also obtained (IHPA, 2006). According to the International HCH and Pesticide Association (IHPA) (report and Annexes), there have been variations in the production methods for HCH and lindane, as well as for HCH isomers destruction or re-use. However, most of the methods to process or re-use the waste HCH isomers have been given up over the years and consequently, most of the waste products have been dumped over the last 50 years (IHPA, 2006). The lindane industry claims that modern production technology processes the waste isomers into TCB (trichlorobenzene) and HCl (hydrochloric acid) thereby reducing or eliminating environmental contamination from these byproducts (Crop Life, 2006).

Historical production of technical HCH and lindane occurred in many European countries, including the Czech Republic, Spain, France, Germany, United Kingdom, Italy, Romania, Bulgaria, Poland, and Turkey, and took place mainly from 1950 or earlier and stopped in 1970 to the 1990s. According to a research by IHPA, technical HCH and lindane have also been produced in other countries including Albania, Argentina, Austria, Azerbaijan, Brazil, China, Ghana, Hungary, India, Japan, Russia, Slovakia and the United States. Exact information is difficult to obtain, as many countries do not keep records of historical pesticides production, sales and usage or the industry considers this to be proprietary information (IHPA, 2006).

It is estimated that global lindane usage from 1950 to 2000 for agricultural, livestock, forestry, human health and other purposes amounts to around 600 000 tons. The next table shows agricultural lindane usage in different continents in the period from 1950 to 2000 (IHPA, 2006).

<table>
<thead>
<tr>
<th>Continent</th>
<th>Usage (tons)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Europe</td>
<td>287,160</td>
</tr>
<tr>
<td>Asia</td>
<td>73,200</td>
</tr>
<tr>
<td>America</td>
<td>63,570</td>
</tr>
<tr>
<td>Africa</td>
<td>28,540</td>
</tr>
<tr>
<td>Oceania</td>
<td>1,032</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>435,500</strong></td>
</tr>
</tbody>
</table>

It appears that in the last years the production of lindane has rapidly decreased leaving only a small number of producing countries. Romania, India, and possibly Russia are the only countries in the world still currently producing Lindane (IHPA, 2006 and USEPA, 2006, CEC, 2005 Annex A). Other sources indicate that Russia (Li et al., 2004) and China (USEPA, 2006) have stopped producing lindane. India produces and uses lindane for the control of mites in sugarcane at 200 tonnes per year.

Global lindane production between 1990 and 1995 was around 3 222 tons per year. In Europe, the top 10 countries with highest lindane usage between 1950 and 2000, representing 96% of the total usage in Europe, were: Czechoslovakia, Germany, Italy, France, Hungary, Spain, Russia, Ukraine, Yugoslavia and Greece (IHPA, 2006).

The 1998 Food and Agriculture Organization Inventory of Obsolete, Unwanted and/or Banned Pesticides found a total of 2785 tons of technical-grade HCH, 304 tons of lindane, and 45 tons of unspecified HCH material scattered in dumpsites in Africa and the Near East (Walker et al., 1999).
According to the information from the Arctic Council’s Arctic Contaminants Action Program (ACAP) project on obsolete pesticides, possibly up to 1,000 tonnes of obsolete stockpiles of technical HCH and lindane still exist in the Russian Federation after the ban of production in the beginning of the 1990s.

b) Uses

Lindane has been used as a broad-spectrum insecticide, which acts by contact, for both agricultural and non-agricultural purposes. Lindane has been used for seed and soil treatment, foliar applications, tree and wood treatment and against ectoparasites in both veterinary and human applications (WHO, 1991).

As a consequence of its toxic, suspected carcinogenic, persistent, bioaccumulative and suspected endocrine disrupting properties, lindane became a substance of scrutiny for countries in the European Community. All uses of HCH including lindane have been banned, but Member States may allow technical HCH for use as an intermediate in chemical manufacturing and in products with at least 99% of the isomer content in the gamma form (lindane) for public health and veterinary topical use only, until December 31st 2007 (UNECE, 2004). Currently, the only registered agricultural use for lindane in the United States is for seed treatment and for lice and scabies treatment on humans (CEC, 2005). In Canada the major use of lindane has been on canola and corn, but the only current allowable use of lindane is for public health purposes, as a lice and scabies treatment (CEC, 2005).

c) Releases to the environment

Considering every ton of lindane produced generates approximately 6 - 10 tons of other HCH isomers, a considerable amount of residues was generated during the manufacture of this insecticide. For decades, the waste isomers were generally disposed of in open landfills like fields and other disposal sites near the HCH manufacturing facilities. After disposal, degradation, volatilization, and run off of the waste isomers occurred (USEPA, 2006).

If the estimate of global usage of lindane of 600,000 tons between 1950 and 2000 is accurate, the total amount of possible residuals (if it is assumed that a mean value of 8 tons of waste isomers are obtained per ton of lindane produced) amounts to possibly 4.8 million tons of HCH residuals that could be present worldwide giving an idea of the extent of the environmental contamination problem (IHPA, 2006).

Air releases of lindane can occur during the agricultural use or aerial application of this insecticide, as well as during manufacture or disposal. Also, lindane can be released to air through volatilization after application (Shen et al., 2004). Evaporative loss to air from water is not considered significant due to lindane’s relatively high water solubility (WHO/Europe, 2003).

2. ENVIRONMENTAL FATE

a) PERSISTENCE

A half-life for lindane in air of 2.3 days was estimated, based on the rate constant for the vapor-phase reaction with hydroxyl radicals in air; a tropospheric lifetime of 7 days due to gas-phase reaction with hydroxyl radicals was estimated, and a lifetime of 13 days was estimated for atmospheric reaction with OH radicals in the tropics (Mackay, 1997). Brubaker and Hites (1998) estimated a lifetime in air of 96 days for lindane. Lindane has half-lives of 3-30 days in rivers and 30 to 300 days in lakes. Other studies report calculated or experimental hydrolysis half-lives ranging from 92 to 3090 hours depending on the study; a persistence of about 2 to 3 years in soil is also reported (Mackay et al., 1997).
Once released into the environment, lindane can partition into all environmental media, but it is demonstrated that evaporation is the most important process in the distribution of lindane in the environment. Several studies focusing on the adsorption-desorption characteristics of lindane have shown that mobility of lindane is very low in soils with a high content of organic material, and higher in soils with little organic matter. The diffusion of lindane has also been investigated, showing it is strongly influenced by the water content of the soil and by temperature. The International Program on Chemical Safety states that when lindane suffers environmental degradation under field conditions, its half-life varies from a few days to three years depending on many factors including climate, type of soil, temperature and humidity (WHO, 1991).

Hydrolysis is not considered an important degradation process for lindane in aquatic environments under neutral pH conditions. Lindane is stable to hydrolysis at pH 5 and 7 with a half-life of 732 days and a half-life of 43 to 182 days at pH 9. Also, different estimated and calculated half-life values for lindane have been reported to be: 1.1 years at pH 8 and 20°C in seawater; 42 years at pH 7.6 and 5°C in Lake Huron, and 110 years in the Arctic Ocean at pH 8 and 0°C (USEPA, 2006).

Lindane is stable to light. Since lindane does not contain chromophores that absorb light, direct photolysis either in air, water or soil is not expected to occur. Even when indirect photolysis could occur with a photosensitizing agent, there is no clear evidence of lindane photodegradation. Lindane degrades very slowly by microbial action with a calculated half-life in soil of 980 days under laboratory aerobic conditions. Degradation takes place faster under anaerobic conditions than in the presence of oxygen. Possible degradation products are pentachlorocyclohexene, 1,2,4-trichlorobenzene, and 1,2,3-trichlorobenzene (USEPA, 2006).

b) BIOACCUMULATION

The bioconcentration factors (BCF) in aquatic organisms under laboratory conditions ranged from approximately 10 up to 6000; under field conditions, the bioconcentration factors ranged from 10 up to 2600 (WHO, 1991). Other studies report bioconcentration factors (log BCF) ranging from 2.26 in shrimp to 3.85 in rainbow trout in early life stages on lipid basis and 4.3 in zooplankton and a bioaccumulation factor (log BAF) up to 4.1 in rainbow trout (Mackay et al., 1997). Also, uptake and elimination rate constants ranging from 180 – 939 h⁻¹ and 0.031 – 0.13 h⁻¹ respectively have been reported for rainbow trout in early life stages on lipid basis (Mackay et al., 1997).

Lindane can bio-accumulate easily in the food chain due to its high lipid solubility and can bio-concentrate rapidly in microorganisms, invertebrates, fish, birds and mammals. Bioconcentration factors (BCF) within aquatic species vary considerably, with experimental data revealing bioconcentration factors of 3-36 (Berny, 2002); 43-4220 on a wet weight basis, and a mean BCF of 11,000 on a lipid basis (Geyer et al., 1997); and also 1200-2100 (Oliver et al., 1985).

An average log BCF of 2.28 in invertebrate species and an average log BCF of 2.87 in vertebrate species can be calculated from different studies (Donkin et al., 1997, Renberg et al., 1985, Thybaud et al., 1988, Yamamoto et al., 1983, Butte et al., 1991, Carlberg et al., 1986, Kanazawa et al., 1981, Kosian et al., 1981 La Rocca et al., 1991, Oliver et al., 1985, Vigano et al., 1992). In the same way, an average log BAF of 2.94 in invertebrate species, and an average log BAF of 3.80 in vertebrate species can be calculated from other studies (Oliver et al., 1988, Chevreuil et al., 1991, Hartley et al., 1983, Caquet et al., 1992). Bioconcentration factors of 780 for fillet, 2500 for viscera and 1400 for whole fish tissues have also been reported (USEPA, 2002).

In an experiment carried out by Geyer et al. (1997), bioconcentration factors are shown to be dependent on the fish species and their lipid content; additionally, different modes of uptake, metabolism, sources of contamination and even experimental conditions, taken together could explain the significant variation observed for BCF values. Also, most data
suggest that, although lindane may bioconcentrate rapidly, bio-transformation, depuration and elimination are relatively rapid once exposure is eliminated. (WHO, 1991).

The bioaccumulation of lindane has been observed for most taxonomic groups, from plants and algae to vertebrates. The environmental consequences of the combination of this bioaccumulation potential with a high toxicity – no-observed-adverse-effect levels (NOAELs) as low as 0.3 mg/kg body weight/day – and ecotoxicity – aquatic ecosystem no-observable-effect concentration (NOEC) below 1 µg/l (Environmental Health Criteria No. 124, 1991; and Brock et al., 2000) – should be considered. For example, when measured field levels in earthworms (0.3 mg/kg for a soil containing 80 µg/kg) are weighed against mammalian toxicity data (Environmental Health Criteria No. 124, 1991;) using a realistic food intake ratio of 0.63 (Guidance document on risk assessment for birds and mammals 2002.) the comparison indicates an area of ecotoxicological concern which should be further explored.

Lindane has been reported in seabirds, fish and mammals in the Arctic (ATSDR, 2005). Lindane concentrations in marine mammals are found at equivalent or even higher levels than some of the more hydrophobic contaminants such as polychlorinated biphenyls (PCBs) and DDT (ATSDR, 2005). In addition, lindane has been reported in human breast milk among Inuit in the Arctic and in marine mammals (Arctic Monitoring and Assessment Programme, 2002).

c) POTENTIAL FOR LONG-RANGE ENVIRONMENTAL TRANSPORT

Many studies have reported HCH residues, particularly alpha and gamma isomers throughout North America, the Arctic, Southern Asia, the Western Pacific, and Antarctica. HCH isomers, including lindane, are the most abundant and persistent organochlorine insecticide contaminants in the Arctic, and their presence in the Arctic and Antarctic, where technical HCH and lindane have not been used, is evidence of their long-range transport. HCH isomers, including lindane, are subject to “global distillation” in which warm climates at lower latitudes favor evaporation into the atmosphere where the chemicals can be carried to higher latitudes. At midlatitudes, deposition and evaporation vary with season. At high latitudes, cold temperatures favor deposition (Walker et al., 1999).

Use of lindane in countries such as Canada, where usage was ~ 500 tons in 2000, and certain European countries, such as France, has contributed to gamma-HCH levels present in the Arctic air, Concentrations of lindane were detected at Alert in the Arctic and varied from 10-11 pg/m³ in 1993 decreasing to 6.4 pg/m³ in 1997 (CACAR, 2003).

In a study completed by Shen et al. in 2004, 40 passive air sampling stations were located along transects from the Canadian Arctic, down the east coasts of Canada and the U.S., along the Canada - U.S. border and in southern Mexico and Central America for one year. The elevated alpha-HCH levels (sampler volumetric air concentrations between 1.5 and 170 pg/m³) in eastern Canada were explained by outgassing of alpha-HCH from cold arctic water flowing south, warming, and releasing the alpha-HCH back to the atmosphere. High concentrations of gamma-HCH (sampler volumetric air concentrations between 5 and 400 pg/m³) were found in the Canadian prairies, north of Lake Ontario, southern Québec, the middle Atlantic states and southern Mexico, reflecting the influence of regional lindane usage (Shen et al., 2004). Transport over the Pacific Ocean of lindane was measured at a sampling site in Yukon and ranged 4-18 pg/m³ (Bailey et al., 2000). HCH isomers, including lindane, were measured at a mountain site at Tenerife Island from June 1999 to July 2000. Air concentrations of gamma-HCH at this site ranged 18 - 31 (mean 26) pg/m³ (Van Drooge et al., 2002).

Lindane is very prevalent in the marine environment and soils, and its atmospheric long range transport potential has been demonstrated for the European Union, (WHO/Europe, 2003) especially by the European Monitoring and Evaluation Program (EMEP). High concentrations of gamma-HCH in air occurred in France, Portugal, Spain, the Netherlands and Belgium. These can be explained by the high emission densities of
lindane in these countries. Relatively high air concentrations were also found in Germany, Italy, Switzerland and Luxembourg, despite the lower lindane emission densities in these countries. These elevated air concentrations were probably explained by atmospheric transport from the former high-density emission European countries (Shatalov and Malanichev, 2000; Shatalov et al., 2000).

- **Isomerization**

The hypothesis that isomerization of gamma-HCH to alpha-HCH could be taking place in air emerged as a possible explanation for alpha-HCH/ gamma-HCH ratios that were found in the 80's as high as 18, when this ratio was expected to be around 5 according to the fraction of these two isomers found in the technical HCH mixture. (Oehme et al 1984a, Oehme et al., 1984b, Pacyna et al., 1988) However no conclusive experimental evidence of isomerization taking place in air has been produced to date.

In the same line, Walker et al. (1999) noted that if photochemical transformation of gamma-HCH to alpha-HCH in air takes place, one should see significant concentrations of alpha-HCH in the Southern Hemisphere air. However, recent measurements have found alpha-HCH levels are dropping over time in the Southern Hemisphere as well as in the Arctic Ocean, which is not consistent with the isomerization theory and a continued use of lindane. The ratio of alpha-HCH/gamma-HCH in air sampled in the Southern Hemisphere during the 1980s - 1990s was generally 1 to 2.3 (Ballischmiler et al., 1991, Bidleman et al., 1993, Iwata et al., 1993, Kallenborn et al., 1998, Lakaschus et al., 2002; Schreitmüller et al., 1995) and was 0.81 in the most recent study in Antarctica (Dickhut et al., 2005).

Other studies have suggested that differential air-sea gas exchange rates could lead to fractionation of the HCH isomers and preferential accumulation of alpha-HCH in air during long range transport over the oceans. This could account for some portion of the elevated alpha-HCH/gamma-HCH ratios observed during wintertime, but not for the very high ratios found in summer in the early studies. (Pacyna et al., 1988 and Oehme et al., 1991). Walker et al. (1999) concluded that even when the experiments show that photoisomerization is possible, evidence that this process is a substantial contributor to the high alpha/gamma ratios observed in the Arctic is indirect and subject to several interpretations.

Several studies have also reported photolytic isomerization of gamma-HCH to alpha-HCH. However, these studies have demonstrated isomerization in condensed media, but there is no evidence that isomerization takes place in the gas phase under ambient atmospheric conditions. Laboratory evidence shows that gamma-HCH can be transformed into other isomers in soil or sediments through biological degradation, but although the biosisomerization of lindane can take place, it seems that this process may play an insignificant role in the overall degradation of gamma-HCH (Walker et al., 1999 and Shen et al., 2004).

- **Environmental monitoring data**

Poland reported concentrations of gamma-HCH in river sediments ranging from 2.4 to 9.4 µg/kg. Results from the National Veterinary Residue Control Programme in Poland indicate that food of animal origin contains levels of gamma-HCH below the level of action of 1000 µg/kg (Annex E information provided by Poland, 2006).

The Ministry of Environment in Japan has monitored Lindane in water finding a concentration of Lindane of 32 to 370 pg/l in 60 surveyed water specimens across the country in 2003. A total of 186 bottom sediment specimens were also surveyed in 2003 and Lindane was detected in all the specimens, with a
concentration of Lindane from traces (1.4) to 4000 pg/g dry, with a geometric mean of 45 pg/g dry. A recent survey in 2003 on shellfish, fish and birds shows that Lindane was detected in all the specimens with concentrations ranging from 5.2 to 130 pg/g-wet for shellfish, 130 pg/g-wet for fish, and 1,800 to 5,900 pg/g-wet for birds. Lindane was detected in all 35 specimens from 35 sites in Japan for ambient air in the warm season in 2003 with a concentration of Lindane ranging from 8.8 to 2,200 pg/m$^3$ with a geometric mean of 63pg/m$^3$. The survey on the same sites excluding one site during the cold season in year 2003 indicates a concentration of 3.1 to 330 pg/m$^3$ with a geometric mean of 14pg/m$^3$ (Annex E information provided by Japan, 2006).

Australia reported that none of the meat and crop samples monitored for residues in the country contained detectable levels of lindane (Annex E information provided by Australia, 2006).

The United States reported that gamma-HCH, was below the level of detection in all samples analyzed for the Third National Report on Human Exposure to Environmental Chemicals. Lindane was detected in fish tissue from lakes and reservoirs in the US EPA national Lake Fish Tissue Study, with levels ranging from 0.652 to 8.56 ppb. Lindane is being monitored in air and precipitation with the Integrated Atmospheric Deposition Network in the Great Lakes region with average concentration of 15-90 pg/m$^3$ in the early 90s, decreasing to 5-30 pg/m$^3$ since 2000. Average concentrations in precipitation (volume-weighted mean) at seven main sites during the years 1997-2003 were 690-1400 pg/L for lindane. The most recent years of available analytical data in the U.S. EPA's Great Lakes Fish Monitoring Program indicate the concentration of Lindane in sport fish fillets (Chinook and Coho Salmon and Steelhead Trout) have ranged between trace detection and 0.005 ppm between 1982 and 2000. The National Oceanic and Atmospheric Administration's National Status and Trends (NS&T) Program has measured lindane in the tissues of bivalves throughout the coastal US and Great Lakes from 1986 to present. Over the Program's history, a total of 283 sites throughout the contiguous US, Alaska, Hawaii, and Puerto Rico have been sampled, with a total of 4,990 records for the gamma isomer. Median measured concentration for gamma-HCH was 0.56 (range 0-71.0) ng/g dry weight. A trends assessment using data pooled for the entire USA, indicates that there has been a statistically significant decline in lindane levels from 1986 through 2003. (Annex E information provided by the United States of America, 2006).

In Canada, a project was undertaken in 1999-2000 by Alberta Environment to characterize the pesticides found in a number of Alberta locations, and to determine their relative levels and seasonality. Lindane was detected in ambient air at Lethbridge in all samples starting from May to August. Lindane levels peaked on June 15 at 1.15 ng/m$^3$, while the low level of 0.23 ng/m$^3$ was present in ambient air on June 22, 1999. As lindane is used on treated seed that is planted in April and early May, lindane is then released into the atmosphere following seeding and hence the higher levels in May followed by a slow decline to low and/or undetectable levels in August and September (Kumar, 2001).

3. EXPOSURE

Lindane can be found in all environmental compartments and levels in air, water, soil, sediment, aquatic and terrestrial organisms and food have been measured worldwide. Humans are therefore being exposed to lindane as demonstrated by detectable levels in human blood, human adipose tissue and human breast milk (WHO/Europe, 2003).

A special area of concern is the fact that HCH isomers, including lindane, accumulate in colder climates of the world. High concentrations of HCH isomers, including lindane, are found in the Beaufort Sea and Canadian Archipelago (CEC, 2005). Through environmental exposure, gamma-HCH can enter the food chain and accumulate in fatty animal tissue constituting an important
exposure pathway for Arctic or Antarctic animals as well as for humans who rely on these animals for their subsistence diets (USEPA, 2006)

General population exposure to gamma-HCH can result from food intake, particularly from animal origin products like milk and meat, as well as water containing the pesticide. Lindane was found to be 10 times higher in adipose tissue of cattle than in the feed (ATSDR, 2005) showing that animals may be exposed to the compound through food and even through ectoparasite treatment. Lindane has been detected in cow’s milk in countries that still use the chemical as a pesticide. In a study performed in Uganda, Africa, the concentrations of gamma-HCH in cow’s milk was 0.006–0.036 mg/kg milk fat, respectively. Mean levels of gamma-HCH analyzed in cow’s milk samples from two separate areas in India were 0.002 and 0.015 mg/kg. A monitoring study of 192 samples of cow’s milk from Mexico revealed 0.002–0.187 mg/kg of gamma-HCH (ATSDR, 2005).

Determinations of the lindane content in body tissues in the general population have been made in a number of countries. The content in blood in the Netherlands was in the order of < 0.1–0.2 µg/l. In the early 1980s, mean concentrations of gamma-HCH in human adipose tissue in Czechoslovakia, the Federal Republic of Germany and the Netherlands were 0.086, 0.024–0.061 and 0.01–0.02 mg/kg, respectively, on a fat basis. In total-diet and market-basket studies to estimate daily human intake of gamma-HCH, clear differences were observed with time: intake in the period around 1970 was up to 0.05 µg/kg body weight per day, whereas by 1980 intake had decreased to 0.003 µg/kg body weight per day or lower (WHO/Europe, 2003).

Individuals living in rural areas and on a non-vegetarian diet are more likely to be exposed to gamma-HCH as shown by a study performed in India, where women who consumed red meat, eggs and chicken had higher pesticide levels, including lindane, in blood than vegetarian women (ATSDR, 2005). Other sources of direct exposure include facilities at which lindane is still being produced, abandoned pesticide plants, and hazardous waste sites (USEPA, 2006).

Exposure of children to lindane is a particular concern. Gamma-HCH has been found in human maternal adipose tissue, maternal blood, umbilical cord blood and breast milk. Lindane has also been found to pass through the placental barrier. Mean breast milk concentration of lindane was 0.084 mg/l in a study in India. An average level of 6 ppb lindane in breast milk was obtained in a study in Alberta, Canada (ATSDR, 2005). In a study looking at organochlorine pesticides in human breast milk collected from 12 regions in Australia, lindane was detected in all samples with a mean of 0.23 ng/g lipid and a range of 0.08-0.47 ng/g lipid (Annex E information provided by Australia, 2006).

Lindane levels have also been found in human breast milk from different countries including Canada, Germany, the Netherlands and the United Kingdom. Lindane levels ranged from <0.001 to 0.1 mg/kg on a fat basis (WHO/Europe, 2003).

An additional exposure route for children exists in regions where lindane is applied directly to milk and meat producing livestock for pest control. On a body weight basis, children consume more milk per unit body weight than adults, and thus may be exposed to significant concentrations of lindane residues through drinking milk (CEC, 2005). Medical use of products to treat head lice and scabies is also of concern when applied to children, although most adverse effects have been observed after misuse. Another exposure to possibly significant amounts of lindane might occur through household dust in certain conditions, and are also of concern especially for children (ATSDR, 2005).

4. HAZARD ASSESSMENT FOR ENDPOINTS OF CONCERN

Lindane is the most acutely toxic HCH isomer affecting the central nervous and endocrine systems. In humans, effects from acute exposure at high concentrations to lindane may range from mild skin irritation to dizziness, headaches, diarrhea, nausea, vomiting, and even convulsions and death (CEC, 2005). Respiratory, cardiovascular, hematological, hepatic and endocrine effects have also been reported for humans, following acute or chronic lindane inhalation. Hematological alterations like leukopenia, leukocytosis, granulocytopenia,
granulocytosis, eosinophilia, monocytosis, and thrombocytopenia, have been reported, following chronic human occupational exposure to gamma-HCH at production facilities (ATSDR, 2005).

Additionally, gamma-HCH has been detected in the blood serum, adipose tissue and semen of occupationally and environmentally exposed individuals (ATSDR, 2005). Serum luteinizing hormone levels were significantly increased in men occupationally exposed to gamma-HCH. Also, the mean serum concentration of follicle stimulating hormone was increased and testosterone was decreased in exposed individuals, but these trends were not statistically significant compared to unexposed controls (ATSDR, 2005).

The most commonly reported effects associated with oral exposure to gamma-HCH are neurological. Most of the information is from case reports of acute gamma-HCH poisoning. Seizures and convulsions have been observed in individuals who have accidentally or intentionally ingested lindane in insecticide pellets, liquid scabicide or contaminated food (WHO/Europe, 2003).

In India, blood levels of gamma-HCH were significantly higher in 135 breast cancer patients, 41-50 years of age, compared to a control group without the disease. However, in similar studies in other countries, a correlation between breast cancer incidence and elevated levels of gamma-HCH in blood was not observed (ATSDR, 2005).

Rats exposed to various concentrations of gamma-HCH through inhalation for 4 hours exhibited concentration-related neurological effects when observed for up to 22 days after exposure. Slight-to-moderate sedation was observed after exposure to 101 mg/m$^3$; slight-to severe sedation was noted after exposure to 378 mg/m$^3$; restlessness, excitement, and ataxia were seen after exposure to 642 and 2,104 mg/m$^3$; and spasms were also noted at the highest concentration of 2,104 mg/m$^3$ (ATSDR, 2005).

Hepatotoxic effects of lindane have been demonstrated in laboratory animals by numerous studies. Increases in cytochrome P-450 levels after inhalation of lindane aerosol at 5 mg/m$^3$ for 90 days and increases in cytochrome P-450 activity cytoplasmic superoxide dismutase, lipid peroxidation in rats after being fed 1.8 mg/kg body weight for 15 and 30 days, have been demonstrated. Chronic studies with a dose of 7-8 mg/kg body weight of lindane in the diet showed liver necrosis and fatty degeneration in rats exposed for 38 to 70 weeks, and hypotrophy in Wistar rats exposed for 104 weeks (WHO/Europe, 2003). Rats exposed to 15 mg gamma-HCH/kg/day for 5 days and 2.5 mg gamma-HCH/kg/day for 21 days, showed significant increases in absolute liver weight, P-450 and EROD activity in a dose- and time-dependent manner (ATSDR, 2005).

Some evidence is available for immunotoxic effects, like immunosuppression and suppressed antibodies responses, caused by lindane in laboratory animals. Immunosuppression was observed in rats exposed to 6.25 and 25 mg/kg body weight for 5 weeks. Primary antibody response was suppressed in albino mice being exposed to 9 mg/kg body weight per day in the diet for 12 weeks, and secondary antibody response suppression was observed after 3 weeks at the same dose (WHO/Europe, 2003).

Reproductive effects of lindane have been recorded in laboratory animals: female rats exposed orally to 10 mg/kg body weight per day for 15 weeks presented anti-estrogenic properties. Female rabbits exposed to gamma-HCH at 0.8 mg/kg body weight per day, 3 days per week for 12 weeks had a reduced ovulation rate (WHO/Europe, 2003). In male rats, reductions in the number of testicular spermatids and epididymal sperms were observed after an oral dose of 6 mg/kg body weight for 5 days, or a single dose of 30 mg/kg body weight of gamma-HCH. Testicular atrophy, seminiferous tubules degeneration and disruption of spermatogenesis were also reported in male rats fed 75 mg/kg body weight per day for 90 days (WHO/Europe, 2003). Lindane has therefore characteristics of an endocrine disrupting compound. Exposure to lindane during gestation with a single dose of 30 mg/kg of body weight at day 15 of pregnancy, induced altered libido and reduced testosterone concentration in male offspring rats (USEPA, 2006).

Developmental effects of lindane have also been reported. Decreased fetal weight, fetal thymic weight, and placental weight were observed in mice treated at 30 and 45 mg/kg by gastric intubation at day 12 of gestation. Fetotoxic effects of lindane were also observed and may be due
to induced oxidative stress, enhanced lipid peroxidation and DNA single strand breaks in the fetal and placental tissues (WHO/Europe, 2003). Rats exposed to 1.7, 3.4 and 6.8 µM corresponding to exposure doses that might be encountered in contaminated vegetables (80-250 µg/kg) or contaminated drinking water (0.02 µg/l) for 12 weeks, showed an affected growth rate, decreased spermatozoid count, as well as decreased testosterone levels during gestation, lactation or weaning (WHO/Europe, 2003). Evidence of increased susceptibility of the young animal was noted in a rat multi-generation reproduction study and rat developmental neurotoxicity study (USEPA, 2002).

The available genotoxicity data indicate that gamma-HCH has some genotoxic potential. Gamma-HCH has been shown to increase chromosome clastogeny in bone marrow cells in mice exposed to 1.6 mg per kg body weight per day by gavage for 7 days (ATSDR, 2005). Nevertheless, lindane is not classified as genotoxic by the European Union (WHO/Europe, 2003). DNA damage was observed in cultures of rat nasal and gastric mucosa cells, and human nasal mucosa cells exposed to gamma-HCH and induced unscheduled DNA synthesis in certain types of cells, like human peripheral lymphocytes (ATSDR, 2005).

The International Agency for Research on Cancer (IARC) has classified lindane as possibly carcinogenic to humans; it has also classified technical HCH and alpha-HCH as possible human carcinogens (ATSDR, 2005). The US EPA has recently reclassified lindane in the category “Suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential”. USEPA has classified technical-grade HCH and alpha-HCH as probable human carcinogens while beta-HCH is a possible human carcinogen (ATSDR, 2005).

Carcinogenicity of lindane has been tested by oral administration in different experiments. Some studies have shown no significant increases in endocrine, thyroid, pituitary, adrenal gland, liver, or ovary tumors in rats fed 10.8–33 mg/kg/day in the diet for 80 weeks, or 0.07–32 mg gamma-HCH/kg/day in the diet for 104 weeks, but poor survival rates limited the significance of such results (WHO/Europe, 2003). While other studies have reported hepatocellular carcinomas in mice exposed to 13.6–27.2 mg/kg/day in the diet for 80 or 104 weeks, and in mice exposed to 27.2 mg/kg/day in the diet for 96 weeks, these results were obtained in a strain of mouse that has a dominant mutation resulting in an increased susceptibility to formation of strain-specific neoplasms.

Lindane is highly toxic to aquatic organisms and moderately toxic to birds and mammals following acute exposures. Chronic effects to birds and mammals measured by reproduction studies show adverse effects at low levels such as reductions in egg production, growth and survival parameters in birds and decreased body weight gain in mammals, with some effects indicative of endocrine disruption. Acute aquatic toxicity data on lindane indicate that it is highly toxic to both freshwater fish (LC₅₀ ranges of 1.7 to 131 ppb) and aquatic invertebrates (LC₅₀ ranges of 10.0 to 520 ppb). Chronic aquatic toxicity data for freshwater organisms show reduction in larval growth in freshwater fish at a NOAEC of 2.9 µg/l, and decreased reproduction in aquatic invertebrates at a NOAEC of 54 µg/l (CEC, 2005 and USEPA, 2006).

Lindane produced statistically significant sex ratio effects (71% males) in frogs at a level of 0.1 ppb and estrogenic activity as well as altered sperm responsiveness to progesterone and induced expression of vitellogenin and estrogen receptors in in vitro tests (USEPA, 2006). Reproductive and population effects were found at a LOAEL of 13.5 µg/l lindane in invertebrate in a 35 day study. Lindane at 100 ppm and 25 ppm caused reduced hatchability in both laying hens and Japanese quails, respectively (USEPA, 2006).

In 2002, USEPA published a dietary risk assessment for indigenous people in the Arctic for lindane. This dietary risk assessment is based on a number of hazard and exposure assumptions, and estimates risk to communities in Alaska and others in the circumpolar Arctic region who depend on subsistence foods, such as caribou, seal and whale. The total dietary intakes for adults ranged from 0.000055 to 0.00071 mg/kg/day. For non-cancer effects, the Level of Concern was (LOC) =0.0016 mg/kg/day. The dietary risks for lindane did not exceed the LOC (USEPA, 2002).
Although the decision to include lindane in the Stockholm Convention would be based on the gamma isomer alone, the POPRC agreed that discussions could include the alpha and beta isomers. Therefore, information from a 2006 USEPA risk assessment on the alpha and beta isomers is included below.

In February 2006, USEPA published for public comment a risk assessment that discussed risks from lindane and the alpha- and beta-HCH isomers, by-products of the lindane manufacturing process (USEPA, 2006). Total dietary intakes were estimated for adults and children and ranged from 0.00057 to 0.051 mg/kg/day for alpha-HCH, and from 0.00037 to 0.01 mg/kg/day for beta-HCH. These dietary intakes were compared to USEPA’s chronic level of concern (LOC). For non-cancer effects, the LOC is cRfD=0.00006 mg/kg/day for beta-HCH and a cRfD=0.001 mg/kg/day for alpha-HCH, based on the dose at which USEPA has concluded will result in no unreasonable adverse health effects. The cancer LOC is when the estimated upper bound cancer risk exceeds one in one million. The dietary risk assessment indicates that the chronic and cancer dietary risk estimates for alpha- and beta-HCH are above the USEPA levels of concern (LOC) for these Arctic populations based on high-end dietary intake estimates.

**Synthesis of information**

Lindane has been shown to be neurotoxic, hepatotoxic, immunotoxic and to have reproductive effects in laboratory animals. Human acute intoxication data show that lindane can cause severe neurological effects, and chronic data suggest possible haematological effects. The International Agency for Research on Cancer (IARC) has classified lindane as possibly carcinogenic to humans (ATSDR, 2005). The US EPA classified lindane in the category “Suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential”.

Human exposure to lindane, particularly in pregnant women and children, is a concern heightened by the ongoing presence of HCH isomers, including lindane, in human tissues and breast milk. Direct exposure from the use of pharmaceutical products for scabies and lice treatment should be of concern. Exposure from food sources is possibly of concern for high animal lipid content diets and subsistence diets of particular ethnic groups (USEPA, 2006 and CEC, 2005). Occupational exposure at manufacturing facilities should be of concern, because lindane production implies worker exposure to other HCH isomers as well, for example the alpha isomer is considered to be a probable human carcinogen (USEPA, 2006).

Lindane is very prevalent in the marine environment and soils, with higher concentrations often found in colder regions. The atmospheric long range transport potential of lindane has been demonstrated for the European Region (WHO/Europe, 2003).

Although current production of lindane seems to be declining with only a few producing countries remaining, the inefficient production process used to manufacture this insecticide over the years has been a world wide contamination problem which has left, and might still be leaving behind, an enormous legacy of contaminating waste products (IHPA, 2006).

The evaluation of laboratory experimental data of lindane would suggest a lower potential of bioaccumulation and biomagnification than that expected for other organochlorine pesticides. In fact, lindane should be considered a border case in terms of its potential for bioaccumulation. Fortunately, there is a large amount of monitoring data on biota allowing a real estimation of the risk profile of lindane in comparison with other organochlorine pesticides. The information provided by this huge amount of real field data is conclusive: lindane concentrations in biota samples collected far away from use areas is similar to that observed for other organochlorine pesticides, confirming the concern for persistence, bioaccumulation and long-range transport.

As the toxicity of lindane is also similar or even higher than that observed for other organochlorine pesticides, it should be considered that the concern related to the POP characteristics of lindane is equivalent to that observed for other chemicals already included in the Stockholm Convention. For example, Weisbrod et al., (2000) found lindane levels in pilot whales similar or just slightly lower than those found for aldrin, endrin, heptachlor or mirex. Also Sørmo et al. (2003) and Kannan et al. (2004)
found equivalent levels for the sum of HCHs and for the sum of chlordanes in gray seal and sea otters respectively.

**Concluding Statement**

Lindane has been the subject of numerous risk assessment reports by different agencies, diverse country regulations and international initiatives, indicating the general concern raised by this organochlorine compound and indicating global action has already been undertaken.

The information contained in the numerous risk assessment reports published on lindane, indicate that lindane is persistent, biaccumulative and toxic, and is found in environmental samples all over the world as well as in human blood, human breast milk and human adipose tissue in different studied populations, especially impacting Arctic communities that depend on subsistence foods. These findings indicate that lindane is likely as a result of its long-range environmental transport to lead to significant adverse human health and environmental effects such that global action is warranted.

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The following lindane assessment reports are publicly available through the internet:

- Assessment of Lindane and other Hexachlorocyclohexane Isomers. USEPA. February 2006
- USEPA Reregistration Eligibility Decision (RED) for Lindane. 2002. See RED and supporting health and eco assessments included in the docket.
  [http://www.epa.gov/oppsrrd1/REDs/lindane_red.pdf](http://www.epa.gov/oppsrrd1/REDs/lindane_red.pdf)
  [http://www.inchem.org/documents/hsg/hsg/hsg054.htm](http://www.inchem.org/documents/hsg/hsg/hsg054.htm)
RISK MANAGEMENT EVALUATION

Overview

Mexico proposed that lindane be added to Annex A of the Stockholm Convention on June 29, 2005. The POPs Review Committee evaluated Annex D information at its first meeting and concluded that "the screening criteria have been fulfilled for lindane". The Review Committee at its second meeting evaluated the risk profile for lindane in accordance with Annex E, and concluded that "lindane is likely, as a result of its long range environmental transport, to lead to significant adverse human health and environmental effects such that global action is warranted".

International initiatives on lindane include the Protocol on Persistent Organic Pollutants of the Convention on Long-Range Transboundary Air Pollution; the Rotterdam Convention; and the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic.


Lindane control measures currently implemented in several countries include: Production, use, sale and imports prohibition, registrations and use cancellations, clean-up of contaminated sites, and public health advisories and hazard warnings issuing for pharmaceutical uses.

The assessment of the efficacy and efficiency of control measures is country dependent; however, all countries consider that control measures currently implemented are technically feasible. There are several chemical alternatives for lindane for seed treatment, livestock, and veterinary uses. Alternatives that are currently in use are considered, in general, technically feasible, efficient, available and accessible by the countries that are already using them. A different scenario exists for pharmaceutical alternatives for lindane, where alternatives are available, but failures have been reported for scabies and lice treatments producing a big concern in relation to the limited number of available alternative products on the market. Non-chemical alternatives for lindane agricultural uses have also been reviewed. Some information has been received about the cost of replacing lindane with alternative pesticides in agricultural applications.

Lindane meets several internationally accepted criteria for persistence, bioaccumulation and toxicity. Therefore, the implementation of control measures is expected to reduce the risks from exposure of humans and the environment to lindane. Implementation of control measures is expected to have positive impacts on biota due to the ease with which lindane accumulates in wildlife, especially in Arctic wildlife. There are potential risks identified from dietary exposure, particularly to people in Alaska who depend on foods such as caribou, seal and whale.

Several countries that have already prohibited or restricted lindane use, consider the use of existing stockpiles for a set time period as feasible, leaving a limited amount of waste for disposal. Contaminated sites of former lindane producers, old storages and dumps have to be addressed by several countries.

Canada, the United States, the Czech Republic, the Republic of Zambia and Brazil have mechanisms to monitor and control lindane. Other countries also have programs to share information concerning lindane uses, alternatives and regulations.

A thorough review of existing control measures that have already been implemented in several countries, shows that risks from exposure of humans and the environment to lindane can be reduced significantly. Control measures are also expected to support the goal agreed at the 2002 Johannesburg World Summit on Sustainable Development of ensuring that by the year 2020, chemicals are produced and used in ways that minimize significant adverse impacts on the environment and human health.
Having evaluated the risk profile corresponding to Lindane, and having prepared its risk management evaluation, the POPs Review Committee of the Stockholm Convention concludes that this chemical is likely, as a result of long-range environmental transport, to lead to significant adverse effects on human health and/or the environment, such that global action is warranted. Therefore, in accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends the Conference of the Parties to the Stockholm Convention to consider listing and specifying the related control measures of Lindane in Annex A.

1. STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS

Lindane is listed as a “substance scheduled for restrictions on use” in Annex II of the 1998 Protocol on Persistent Organic Pollutants of the Convention on Long-Range Transboundary Air Pollution. This means that products in which at least 99% of the HCH isomer is in the gamma form (i.e. lindane) are restricted to the following uses: 1. Seed treatment. 2. Soil applications directly followed by incorporation into the topsoil surface layer. 3. Professional remedial and industrial treatment of lumber, timber and logs. 4. Public health and veterinary topical insecticide. 5. Non-aerial application to tree seedlings, small-scale lawn use, and indoor and outdoor use for nursery stock and ornamentals. 6. Indoor industrial and residential applications. All restricted uses of lindane shall be reassessed under the Protocol no later than two years after the date of entry into force. The Protocol entered into force on October 23th, 2003. There are currently 28 Parties to this Protocol. 56

Lindane, as well as the mixture of HCH isomers, is listed in Annex III of the Rotterdam Convention on the Prior Informed Consent Procedure as “chemicals subject to the prior informed consent procedure”. The Rotterdam Convention entered into force 24 February 2004. There are currently 116 Parties to this Convention. 57

Hexachlorocyclohexane isomers, including lindane, are included in the List of Chemicals for Priority Action (Updated 2005) under the OSPAR Commission for the Protection of the Marine Environment of the Northeast Atlantic. Under this initiative, the Hazardous Substance Strategy sets the objective of preventing pollution of the maritime area by continuously reducing discharges, emissions and losses of hazardous substances, with the ultimate aim of achieving concentrations in the marine environment near background values for naturally occurring substances and close to zero for man-made synthetic substances. The OSPAR Convention entered into force on 25 March 1998. 58

HCH (including lindane) is listed as a Level II substance in the Great Lakes Binational Toxics Strategy between the United States and Canada, which means that one of the two countries has grounds to indicate its persistence in the environment, potential for bioaccumulation and toxicity. 59

1.5 Any national or regional control actions taken

Lindane is banned for use in 52 countries, restricted or severely restricted in 33 countries, not registered in 10 countries, and registered in 17 countries (CEC, 2006).

56 Convention on Long-range Transboundary Air Pollution http://www.unece.org/env/lrtap/
The three Parties (Mexico, Canada and the United States) of the North American Commission for Environmental Cooperation (CEC) have recently signed a North American Regional Action Plan (NARAP) on Lindane and Other Hexachlorocyclohexane Isomers, under the Sound Management of Chemicals project. The goal of the NARAP is to reduce the risks associated with exposure of humans and the environment to these substances.

Lindane is also listed under the European Water Framework Directive 200/60/EC. This Directive is a piece of water legislation from the European Community. It requires all inland and coastal water bodies to reach at least “good ecological status” and “good chemical status” by 2015. Lindane is one of the listed priority hazardous substances for which quality standards and emission controls will be set at EU level to end all emissions within 20 years.

Lindane is listed under the European Union Regulation 850/2004/EC, that specifies Member States may allow until September 2006 professional remedial and industrial treatment of lumber, timber and logs, as well as indoor industrial and residential applications; and until December 31, 2007 the use of technical HCH as an intermediate in chemical manufacturing and the restriction of products containing at least 99% of the HCH gamma isomer for use as public health and veterinary topical insecticide (Annex F information provided by Germany, 2007).

HCH is listed in Annexes IB (banned substances) and Annex IV (waste regulation) of European Council Directive 850/2004/EEC. Regulation 850/2004/EC was lately amended by regulation 1195/2006/EC in order to include thresholds for POPs containing waste. Article 7 applies to waste containing >50 mg/kg of the sum of alpha, beta and gamma HCH (Annex F information provided by Germany, 2007).

2. SUMMARY INFORMATION RELEVANT TO THE RISK MANAGEMENT EVALUATION

2.1 Identification of possible control measures

Lindane control measures currently implemented in several countries include: Production, use, sale and imports prohibition, use restrictions, registrations and use cancellations, clean-up of contaminated sites and public health advisories and hazard warnings issued for pharmaceutical uses.

Production, sale and use of lindane are prohibited for all pesticide use in Canada. Stocks that existed at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable, after which their sale or use became a violation of the Pest Control Products Act (PCPA) (Annex F information provided by Canada, 2007).

Lindane is also prohibited for agricultural uses in Mauritius (Annex F information provided by Mauritius, 2007). In the Czech Republic lindane use was banned in 1995, and the site of former producer (Spolana Neratovice) was successfully cleaned-up (Annex F information provided by the Czech Republic, 2007). In Germany, lindane has not been used in agriculture and forestry since 1989. In the European Union, lindane can still be used as public health and veterinary topical insecticide until the end of 2007 (Annex F information provided by Germany, 2007). In Sweden, lindane has not been used for scabies in humans or animals since the 1980s (Annex F information provided by Sweden, 2007).

In Brazil, lindane use and imports are prohibited. Imports and formulation of lindane were allowed until 2006. Commercialization of products with lindane and use of products legally commercialized are allowed until 2007 (Annex F information provided by Brazil, 2007).

Lindane is severely restricted in Switzerland under the Ordinance on Risk Reduction related to Chemical Products. The only legal use is in medicinal products. Until the coming into force of the

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60 Commission for Environmental Cooperation. [www.cec.org/lindane](http://www.cec.org/lindane)

Ordinance, the only legal use of lindane other than medicinal products was in seed dressings for agricultural purposes (Annex F information provided by Switzerland, 2007).

In Thailand, the use of lindane-containing products is restricted. Lindane and lindane-containing products are regulated by the Hazardous Substances Control Act B.E. 2535 (1992). According to the Act, registration and permission of any activities, including production, import, export or possession, are required. Only the use in household and public health programs is permitted under the supervision of the Hazardous Substances Control Group, Food and Drug Administration (Annex F information provided by Thailand, 2007).

In 1998 the United States Environmental Protection Agency (USEPA) cancelled the livestock uses of lindane. In 2006, the United States announced the cancellation of the remaining agricultural uses of lindane, effective July 1, 2007. However, the United States Food and Drug Administration (USFDA) determined that lindane products have benefits that outweigh the risks for individual patients when used as directed as second-line treatment of scabies and lice when other treatments fail or cannot be tolerated. In 2003, USFDA issued a public health advisory, a medication guide, a boxed warning, and limited package sizes to reduce risks from the use of lindane. (Annex F information provided by the United States of America, 2007).

Methods for the clean-up of sites contaminated with lindane include: a) Hazardous waste incinerators and rotary kilns with Gas Phase Chemical Reduction (GPCR), b) Base-catalyzed decomposition, c) Sodium dispersion (alkali metal reduction), d) Subcritical water oxidation, e) Supercritical water oxidation, f) Mechanochemical method and g) GeoMelt. According to technical proofs conducted by the Ministry of Agriculture, Forestry and Fisheries of Japan, all the methods have destruction efficiencies greater than 99.999% (Annex F information provided by Japan, 2007).

2.2 Efficacy and efficiency of possible control measures in meeting risk reduction goals

The efficacy and efficiency of implemented control measures is country dependent.

The Czech Republic considers clean-up of contaminated sites feasible by application of Base-catalyzed decomposition (BCD) technology. The estimated remediation cost of a former lindane production site is 100 000 000 € (Annex F information provided by the Czech Republic, 2007).

In Mauritius, lindane is already listed as a prohibited agricultural chemical in the Dangerous Chemicals Control Act 2006. All imports of chemicals are subject to control by the Dangerous Chemicals Control Board (DCCB) under the Dangerous Chemicals Control Act 2004. The law provides that no person shall import, manufacture, use or possess lindane (Annex F information provided by Mauritius, 2007).

The United States considers it technically feasible to cancel registrations and eliminate agricultural uses. In 2002 lindane was registered as a seed treatment on wheat, barley, corn, sorghum, oats and rye crops. Until 2006, there were no alternatives for use on oats and rye. However, in 2006, Imidacloprid was registered for these two uses, and currently alternatives exist for all six seed treatment uses. The scenario for pharmaceutical uses is quite different in the United States, where at this time it is not technically feasible to withdraw lindane as a treatment for scabies and lice (Annex F information provided by the United States of America, 2007).

Canada has established post-registration monitoring and compliance programs for the agricultural use of lindane to ensure compliance with federal and provincial legislation. Federal, provincial and territorial hazardous waste programs address small quantities of retired material in the possession of consumers and have collected and safely disposed of pesticide products that are no longer registered (Annex F information provided by Canada, 2007).

In Japan, the distribution of lindane was banned by the Minister of Agriculture, Forestry and Fisheries in 1971. Lindane is listed as a deleterious substance under Poisonous and Deleterious Substances Control Law. Manufacturers, importers and sellers are required to register themselves to handle lindane. There are also regulations for labelling containers and packages, and for handling and disposal of lindane (Annex F information provided by Japan, 2007).
In Thailand, banning of medical use of lindane is still in question because the current substitutes for the treatment of head lice and scabies appeared not to be as effective as lindane. For medical purpose, lindane is in the National List of Essential Medicines (2004). It is the second-line drug of choice for head lice or scabies treatment (Annex F information provided by Thailand, 2007).

2.3 Information on alternatives (products and processes)

a) Description of alternatives

Chemical and non-chemical alternatives for the agricultural, veterinary and pharmaceutical uses of lindane in the United States, Canada and Mexico have been reviewed in the North American Regional Action Plan on Lindane and Other HCH Isomers developed by the North American Commission for Environmental Cooperation (CEC, 2006).

In the United States, at least one of the following active ingredients is registered for seed treatment for corn, barley, wheat, oat, rye and sorghum: Clothianidin, Thiamethoxam, Imidacloprid, Permethrin and Tefluthrin. For uses on livestock, Amitraz, Carbaryl, Cymaphos, Cyfluthrin, Cypermethrin, Diazinon, Dichlorvos, Fenvalerate, Lambda-cyhalothrin, Malathion, Methoxychlor, Permethrin, Phosmet, Pyrethrin, Tetrachlorvinfosin, and Trichlorfon are registered. Veterinary Drugs include: Eprinomectin, Ivermectin, Doramectin, Moxidectin, and Methoprene. For pharmaceutical uses, approved treatments for head lice include: Pyrethrum/Piperonyl butoxide, Permethrin, and Malathion. Lice nit combs are also recommended for use in conjunction with these treatments. For scabies, Permethrin and Crotamiton (Eurax) are approved treatments (Annex F information provided by the United States of America, 2007).

Canadian alternatives for pharmaceutical uses of lindane include: Permethrin (1% cream), Bioallethrin and piperonyl butoxide, Pyrethrin and piperonyl butoxide, Permethrin (5% cream), Precipitated sulphur 6% in petrolatum and Crotamiton 10% (Eurax). Canadian registered alternatives for agricultural uses include: for canola: Acetamiprid, Clothianidin, Thiamethoxam and Imidacloprid; for corn: Clothianidin, Imidacloprid (only for field corn grown for seed) and Tefluthrin; and for sorghum: Thiamethoxam and Imidacloprid. Alternatives for livestock treatments include: Carbaryl, Diazinon, Dichlorvos, Malathion, Phosmet, Tetrachlorvinphos, Trichlorfon, Cyfluthrin, Cypermethrin, Fenvalerate, Permethrin, Pyrethrin, Rotenone, Eprinomectin, Ivermectin, Abamectin, Doramectin, Moxidectin and Phosmet (CEC, 2006).


In Germany, alternatives against Atomaria linearis include: Thiamethoxam, Imidacloprid, Imidacloprid / Tefluthrin, Clothianidin, Clothianidin / Beta-Cyfluthrin, Alpha-Cypermethrin and Deltamethrin; against Elateridae: Clothianidin, Imidacloprid and Thiamethoxam; against leaf-cutting insects: Lambda-Cyhalothrin, Acadirachtin, Pyrethrin / Rapsöl, Beta-Cyfluthrin, Alpha-Cypermethrin, Lambda-Cyhalothrin, Acadirachtin, Pyrethrin / Rapsöl and Methamidophos. Alternatives for use as a wood protection product include: 3-Iodo-2-propynyl butylcarbamate (IPBC), (E)-1-(2-Chloro-1,3-thiazol-5-ylmethyl)-3-methyl-2-nitro guanidine / Clothianidin, 1-(4-(2-Chloro-alpha,alpha,alpha-trifluorotolyloxy)-2-fluorophenyl)-3-(2,6-difluorobenzolyl)urea / Flufenoxuron, Cyclopropenecarboxylic acid, 3-[(Z)-2-chloro-3,3,3-trifluoro-1-propenyl]-2,2-dimethyl-, (2-methyl[1,1'-biphenyl]-3-ylmethyl ester, (1R,3R)-rel / Bifenithrin, 3-Phenoxybenzyl-2-(4-ethoxyphenyl)-2-methylpropylether / Etofenprox, m-Phenoxybenzyl 3-(2,2-dichlorovinyl)-2,2-dimethylcyclo propanecarboxylate / Permethrin, alpha.-cyano-3-phenoxybenzyl 3-(2,2-dichlorovinyl)-2,2-dimethylcyclo propanecarboxylate / Cypermethrin, Dazomet, Thiamethoxam and 4-Bromo-2-(4-chlorophenyl)-1-(ethoxymethyl)-5-(trifluoromethyl)-1H-pyrrole-3-carbonitrile / Chlorfenapyr. The alternative used for public health and veterinary topical insecticide is: Infectopedicical solution (Permethrin) (Annex F information provided by Germany, 2007).
In Thailand, alternatives for the treatment of head lice and scabies include: *Permethrin*, *Cabaryl*, *Stemona* root extract and benzyl benzoate. The alternatives for use on pets are: *Permethrin*, *Flumethrin* and *Cypermethrin*; and for termite control: *Alpha-cypermethrin*, *Bifenthrin*, *Cypermethrin* and *Delta-methrin* (Annex F information provided by Thailand, 2007).

In Sweden, *Malation*, *Permethrin* and *Disulfiram* with benzylbenzoate have been used as alternatives against scabies and lice in humans. In veterinary applications, *Flumethrin*, *Foxim*, *Fipronil*, *Ivermectin* and *Moxidectin* have been used (Annex F information provided by Sweden, 2007).

Alternatives used in Brazil include: *Cypermethrin* for termite control in compacted wood, *Cypermethrin* and 3-iodo-2-propynyl butylcarbamate (IPBC) for control of insects and fungi in dry wood, *Cyfluthrin* for wood used in construction or furniture fabrication, *Deltamethrin* for control of termite and drill, *Endosulfan* for termite control in wood, *Fipronil* for termite control in manufacture of compacted agglomerated wood, and TBP for fungal control in just-sawed wood (Annex F information provided by Brazil, 2007).

Alternatives used in Switzerland for seed treatment are *Fipronil* and *Thiamethoxam* (Additional information provided by Switzerland, 2007)

Besides the chemical alternatives, there are also non-chemical alternatives to agricultural seed treatment uses of lindane. Among cultural methods currently known to effectively prevent harm to seeds and crops are: Crop rotation (alfalfa, soybeans and clover), where small grains need to be rotated with a non-host species every year to reduce the severity of infestation and maintain low levels of pests; Site selection and monitoring in order to determine if wireworms are present; Fallowing, starving wireworms by allowing the area to fallow for a few years before planting; Re-seeding with resistant crops such as buckwheat or flax; Timing of seeding and planting, trying to plant in warm, dry conditions, usually later in the season for small grains where larvae are deeper in the soil and giving seedlings a greater chance of survival; Shallow cultivation to starve hatchlings, expose eggs for predation and damage larvae; and Soil packing to impede wireworm travel (CEC, 2006).

Biological methods are also considered as non-chemical alternatives to lindane. Current research at Pacific Agri-Food Research Centre, in Canada is examining the use of *Metarhizium anisopliae*, an insect fungal pathogen to control wireworm. Additional biological control methods employed in Costa Rica include *Trichodama* spp, *Piper aduncum*, *Trichogram* wasps, and *Bacillus thuringiensis* (Annex F information provided by IPEN, 2007).

**b) Technical feasibility**

Chemical alternatives for seed and livestock treatments and approved alternatives for pharmaceutical uses in the United States are technically feasible and are currently in use. However there have been reported treatment failures for all the approved pharmaceutical alternatives for treatment of scabies and lice. Some physicians prescribe off-label oral *Ivermectin* for scabies even when it is not approved by USFDA for treatment for scabies. The manufacturer of oral *Ivermectin* does not have an approved application for its use in scabies and does not advocate its use in scabies. USFDA is currently reviewing potential new treatments for lice from a number of companies and is exploring mechanisms to encourage pharmaceutical companies to submit candidate treatments for scabies (Annex F information provided by the United States of America, 2007).

In Canada, alternative pesticide products are also currently being used. Technical feasibility is a requirement of registration by Canada’s Pest Management Regulatory Agency (PMRA) (Annex F information provided by Canada, 2007).

In Sweden, alternatives are all technically feasible, available, freely accessible and effective if used as prescribed. There are no reports of major resistance problems (Annex F information provided by Sweden, 2007).

**c) Costs, including environmental and health costs**
Only the United States has information available regarding costs associated with alternatives. The information provided includes data from 2002 to 2006.

In 2006, the usage of lindane in the United States was less than 150,000 lbs of active ingredient applied annually to about 9.7 million acres. In 2002, lindane was used in the United States as a seed treatment on wheat, barley, oats, rye, corn, and sorghum. Imidacloprid and Thiamethoxam were the primary seed treatment alternatives to lindane for barely, corn, sorghum and wheat. Moreover, since 2002, additional alternatives have been registered on corn and sorghum. The alternatives are as effective as lindane but costlier to use. For wheat and barley, the estimated increase in treatment cost would be $0.36 to $1.71 per acre ($5 million for all US acreage). For corn, the estimated increase in treatment cost would be $1.82 per acre ($8.7 million dollars for all US acreage). For sorghum, the estimated increase in treatment cost would be $3.70 to $4.69 per acre (about $386,000 for all US acreage) (USEPA, 2006).

In 2002, there were no registered alternatives for oats and rye. If these two uses had been cancelled at that time, there would have been a major impact on growers of those crops, estimated at a 9% yield loss. The total aggregate increase in treatment costs is $14 million. Total aggregate value of yield loss on oats and rye is $354,000. By 2006, Imidacloprid was registered for use on oats and rye (Annex F information provided by the United States of America, 2007).

Regarding health costs associated with pharmaceutical alternatives, scabies can be a serious problem in long-term care facilities, crowded living environments and economically poor conditions in general. Scabies may be complicated by secondary bacterial infections of the lesions, and scabies has been identified as a risk factor for development of post-streptococcal glomerulonephritis. In the United States, children cannot return to school with untreated lice or scabies (Annex F information provided by the United States of America, 2007).

d) Efficacy, including benefits and limitations of alternatives versus nominated substance and identification of any critical uses for which there is at present no alternative

In the United States, resistance has been reported for all of the approved treatments for head lice. For scabies, treatment failures have been reported with all of the approved treatments, and resistance has been reported for Permethrin and oral Ivermectin although the latter is not approved for this indication (Annex F information provided by the United States of America, 2007).

In Thailand, current substitutes for the treatment of head lice and scabies appeared not to be as effective as lindane (Annex F information provided by Thailand, 2007).

e) Risk, including information on whether the proposed alternative has been tested/evaluated and any information on potential risks associated with untested alternatives over the life-cycle of the alternative

USEPA conducts risk assessments for pesticide products as a routine part of the registration process; therefore, the USEPA has conducted risk assessments for the alternative products and uses of those products (Annex F information provided by the United States of America, 2007).

Alternatives have been reviewed in Canada by the PMRA and their efficacy and the environmental and health risks associated with their uses have been considered acceptable (Annex F information provided by Canada, 2007).

f) Availability

Alternatives for the agricultural, livestock and pharmaceutical uses of lindane in the United States are available and currently in use.
The feasibility of using alternatives in the Republic of Zambia remains undetermined. *Gaucho* is readily available and easily accessible on the local market (Annex F information provided by the Republic of Zambia, 2007).

g) Accessibility

The availability and accessibility of alternatives in Canada is market dependant (Annex F information provided by Canada, 2007).

Alternative products in Thailand are on the market. Particularly the alternatives for use on pets or for termite control are widely used (Annex F information provided by Thailand, 2007).

2.4 Summary of information on impacts on society of implementing possible control measures

h) Health, including public, environmental and occupational health

Considering that lindane meets several internationally accepted criteria for persistence, bioaccumulation and toxicity, the implementation of control measures is expected to reduce the risks from exposure of humans and the environment to lindane.

Lindane can be found in all environmental compartments and levels in air, water, soil sediment, aquatic and terrestrial organisms and food have been measured worldwide (WHO/Europe 2003). Lindane can bio-accumulate easily in the food chain due to its high lipid solubility and can bio-concentrate rapidly in microorganisms, invertebrates, fish, birds and mammals. HCH isomers, including lindane, accumulate in colder climates of the world (CEC, 2006). General population exposure to gamma-HCH can result from food intake particularly from animal origin products like milk and meat, as well as water containing the pesticide (ASTDR, 2005). There is potential dietary exposure particularly to people in Alaska who depend on subsistence sea foods such as caribou, seal and whale (USEPA, 2006).

At high doses lindane has been shown to be neurotoxic, hepatotoxic, immunotoxic and to have reproductive effects in laboratory animals. Human acute intoxication data show that lindane can cause severe neurological effects and chronic data suggest possible haematological effects. Although there is some evidence for toxicity of lindane when used topically as a pharmaceutical, this has been generally associated with inappropriate use. Most of the side effects of lindane have been associated with chronic inhalation by seed treatment workers (Annex F information provided by Canada, 2007). The carcinogenicity of lindane is less clear. The International Agency for Research on Cancer (IARC) has classified lindane as possibly carcinogenic to humans (ATSDR, 2005). USEPA reclassified lindane in the category “suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential.” The US Center for Disease Control’s Agency for Toxic Substances and Registry concurs with USEPA’s classification. However, the Joint Meeting on Pesticide Residues (JMPR) of the World Health Organization concluded that “…lindane is not likely to pose a carcinogenic risk to humans” (CEC, 2006).

The implementation of control measures applied to lindane pharmaceutical uses has a positive impact to the environment since lindane application as a lice treatment shampoo or topical lotion that must be washed off, end up in waste water (Annex F information provided by the United States of America, 2007). In 2002, the state of California banned the sale of lindane for lice and scabies treatments in order to reduce the levels in drinking water supplies. In May 2000, the California Toxics Rule (CTR) established a new water quality criterion of 19 parts per trillion (ppt) for lindane in existing or potential drinking water supplies for protection of public health based on potential cancer risk to humans. As available treatment technologies were unable to remove lindane to meet the new California state water quality criterion, a preventive strategy to allow compliance was required. A bill was then sponsored in the California assembly, which passed without opposition, to ban the sale of all pharmaceutical lindane in the state of California beginning in January 2002. Since the ban, lindane concentrations in waste water have declined to almost non-detectable levels (CEC, 2006).
Since the California lindane ban, four scabies outbreaks were reported by four counties to the California Department of Health Services (CDHS) Surveillance and Statistics Section. Prior to the ban, CDHS issued guidelines to all physicians to use Malathion instead of lindane to control head lice. For scabies outbreaks, CDHS developed and distributed to healthcare facilities a guideline where CDHS recommends the use of Ivermectin to treat patients with severe scabies. Although Ivermectin has not been approved by the FDA for use for scabies and it is not recommended by CDHS for typical scabies or prophylaxis, Ivermectin has been used in outbreaks in California for treatment of symptomatic cases and for mass prophylaxis because of its ease of use and probable greater compliance and efficacy compared to Permethrin (CEC, 2006).

On the topic of pharmaceutical uses restriction there is a general concern. In the United States, if lindane products were not available, approved treatment options for lice and scabies would be very limited. Cases of lice and scabies could remain untreated or harmful home remedies might be used (Annex F information provided by the United States of America, 2007). A similar concern exists in Canada where it is felt that lindane should be available for use in cases where an alternative therapy is inappropriate (Annex F information provided by Canada, 2007). In the European Union, lindane can be used as public health and veterinary topical insecticide until end of 2007, and only a limited number of alternative products (based on Permethrin) are currently on the market (Annex F information provided by Germany, 2007).

i) Agriculture, including aquaculture and forestry

There are no impacts of implementing possible control measures reported for this sector.

j) Biota (biodiversity)

Due to the ease of lindane to accumulate in wildlife, implementation of control measures is expected to have only positive impacts on biota, especially in Arctic wildlife. Several studies in the Arctic have monitored HCH levels in Steller sea lion, beluga whales, bowhead whales, and polar bears (Annex F information provided by IPEN, 2007).

k) Economic aspects, including costs and benefits for producers and consumers and the distribution of costs and benefits

Information regarding costs of implementing possible control measures and alternatives is provided in this document in sections 2.2 and 2.3.

2.4 Other considerations

a) Access to information and public education

In Sweden, extensive information on treatment regimes for all available drugs against scabies and lice may be found on the Swedish Medical Products Agency website http://www.lakemedelsverket.se or in the list of pharmaceutical products in Sweden http://www.fass.se (Annex F information provided by Sweden, 2007).


Mexico developed and published in 2004 a National Diagnostic Report on Lindane. The report includes information on production, imports, exports, commercial names, prices, selling patterns, quantities used and possible alternatives.
The Czech Republic has an education and awareness POPs campaign (SC/UN ECE CRLTAP) based on the Czech National Implementation Plan (Annex F information provided by the Czech Republic, 2007).

b) Status of control and monitoring capacity

In Canada, control and monitoring capacity of pesticide uses is managed by the Pest Management Regulatory Agency (PMRA) through compliance mechanisms in place at border crossings and entry points to prohibit the import of lindane to Canada. Compliance issues within Canada may be reported by PMRA through PMRA compliance activities, reporting of suspected infractions and/or results reported from other government agencies (Annex F information provided by Canada, 2007).

In the United States, in December 2006 USEPA announced the cancellation of all agricultural pesticide products containing lindane under the authority of the Federal Insecticide, Fungicide and Rodenticide Act, effective July 1, 2007. USEPA works with its federal, state and tribal regulatory partners to assure compliance with pesticide laws and regulations in order to protect human health and the environment (Annex F information provided by the United States of America, 2007).

Control and monitoring institutions in the Czech Republic include: RECETOX MU for monitoring in ambient air, surface waters, sediments, soils, mosses and needles, Water Research Institute for monitoring of surface and ground waters and sediments, Central Institute for Supervising and Testing in Agriculture (CISTA), Research Institute of Amelioration and Soil Conservation (RIASC), State Veterinary Inspection and Czech Food Inspection for food control, and National Institutes of Public Health for human exposure and dietary studies (Annex F information provided by the Czech Republic, 2007).

The control and monitoring capacity of the Republic of Zambia is addressed through the Environmental Protection and Pollution Control Act which is enforced by the Environmental Council of Zambia (Annex F information provided by the Republic of Zambia, 2007).

The Brazilian Institute for the Environment and Renewable Natural Resources (IBAMA) controls the stockpiles, the adequate destination of obsolete products and the illegal entrance of products (Annex F information provided by Brazil, 2007).

c) Waste and disposal implications

Current production of lindane seems to be declining with only a few producing countries remaining, but former production and the inefficient production process over the years, have left an enormous amount of waste products.

For the United States it would be technically feasible to use agricultural and pharmaceutical existing stocks for a set time period. USEPA will allow the use of lindane products in agriculture until October 1, 2009. Therefore it is expected that there will be minimal costs associated with disposal of unusable stocks (Annex F information provided by the United States of America, 2007).

In Switzerland, about 3000 contaminated sites would require remediation. Specially two sites, Bonfol (Canton Jura) and Kölliken (Canton Aargau) which served as chemical waste disposal sites contain around 114 000 and 350 000 tons respectively of special waste, probably containing POPs chemicals. The exact amount of POPs chemicals in these disposal sites is still unknown. The current estimate is that the now initiated full remediation (including on-site incineration in a high tech oven) will require about CHF 200 and 500 million for Bonfol and Kölliken, respectively (Annex F information provided by Switzerland, 2007).
In the Czech Republic, waste problems represent old contaminated sites of former producer Spolana Neratovice, old storages, unknown illegal stores and dumps. Spolana Neratovice is now successfully cleaned-up using the BCD technology. Plans for future remediation of other contaminated sites are under development (Annex F information provided by Czech Republic, 2007).

In Canada there is no commercial reason to maintain stockpiles given that any stocks that existed at the time that pesticide registration was discontinued or suspended were to be sold, used or disposed of in accordance with an established timetable. Federal, provincial and territorial hazardous waste programs address small quantities of retired material in the possession of consumers and have collected and safely disposed of pesticide products that are no longer registered (Annex F information provided by Canada, 2007).

Chemical disposal methods for lindane as well as costs of disposal and management of HCH isomer wastes in different countries have been reviewed in the report: The legacy of Lindane HCH Isomer Production (Vijgen, 2006).

3. SYNTHESIS OF INFORMATION

Published risk assessment reports on lindane indicate that lindane is persistent, bioaccumulative and toxic. Lindane has been found in environmental samples all over the world as well as in human blood, human breast milk and human adipose tissue in different studied populations, especially in Arctic communities that depend on subsistence foods.

Lindane has been shown to be neurotoxic, hepatotoxic, immunotoxic and to have reproductive effects in laboratory animals. Human acute intoxication data show that lindane can cause severe neurological effects, and chronic data suggest possible haematological effects. The International Agency for Research on Cancer (IARC) has classified lindane as possibly carcinogenic to humans (ATSDR, 2005).

Implementation of control measures is expected to reduce the risks from exposure of humans and the environment to lindane, especially in the Arctic where lindane accumulates easily in the wildlife, and where communities depend on subsistence foods.

Lindane control measures that have shown to be technically feasible, efficient and accessible include: Production, use, sale and imports prohibition, use restrictions, registrations and use cancellations and clean-up of contaminated sites. Therefore, they can be proposed as potential control measures to be implemented by countries. When lindane registrations are cancelled, allowing the use of stocks for a reasonable time period is a recommended strategy in order to reduce the amount of waste generated and the costs associated with disposal.

Lindane chemical alternatives that have been reviewed in the present document for agricultural, livestock and veterinary uses are considered efficient, technically feasible and accessible. However, alternatives for pharmaceutical uses have often failed for scabies and lice treatment and the number of available alternative products for this use is scarce. For this particular case, a reasonable alternative would be to use lindane as a second-line treatment when other treatments fail, while potential new treatments are assessed. Issuing public health advisories would be recommended to control lindane use patterns and reduce risks associated.

CONCLUDING STATEMENT

Having evaluated the risk profile corresponding to Lindane, and having prepared its risk management evaluation, the POPs Review Committee of the Stockholm Convention concludes that this chemical is likely, as a result of long-range environmental transport, to lead to significant adverse effects on human health and/or the environment, such that global action is warranted.

A thorough review of existing control measures that have already been implemented in several countries, shows that risks from exposure of humans and the environment to lindane can be reduced significantly. Control measures are also expected to support the goal agreed at the 2002
Johannesburg World Summit on Sustainable Development of ensuring that by the year 2020, chemicals are produced and used in ways that minimize significant adverse impacts on the environment and human health.

Therefore, in accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends the Conference of the Parties to the Stockholm Convention to consider listing and specifying the related control measures of Lindane in Annex A.

References


B.7. Octabromodiphenyl ether – SUMMARY

SUMMARY
7. OctaBDE

Draft Risk Profile May 2007

<table>
<thead>
<tr>
<th>Composition</th>
<th>Commercial mixture contains bromodiphenyl ethers including: hexa/penta (1.4 – 12%), hepta (43 – 58%), octa (26 – 35%), nona (8-14%), and deca (0 – 3%). Always used in conjunction with antimony trioxide.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>Flame retardant primarily used in acrylonitrile-butadiene-styrene (ABS) polymers at 12-18% weight loadings in the final product. Other minor uses, accounting for the remaining 5% use, include high impact polystyrene (HIPS), polybutylene terephthalate (PBT) and polyamide polymers, at typical loadings of 12- 15% weight in the final product. The flame retarded polymer products are typically used for the housings of office equipment and business machines. Other uses that have been reported for octabromodiphenyl ether include nylon and low density polyethylene (WHO, 1994), polycarbonate, phenol-formaldehyde resins and unsaturated polyesters (OECD, 1994) and in adhesives and coatings (WHO, 1994).</td>
</tr>
<tr>
<td>Releases</td>
<td>Although there are some figures on annual production of this mixture, there are no accurate values on the amount of the commercial octa and/or the individual homologues in articles in service and disposed at the world-wide level, but considering the estimated figure of 6 000 tonnes/year (WHO, 1994) the total amount should be expected in the 105 – 106 tonnes range. According to the BSEF, OctaBDE was commercialized sometime in the mid 70’s. By the early 2000’s global production was &lt;4000 tonnes/year and by the time production ceased, demand was &lt;500 tonnes. While Thus, assuming 30 years of production at 6000 tonnes per year gives 180,000 tonnes, a figure within the proposed range.</td>
</tr>
<tr>
<td>Fate</td>
<td>The persistence of these PBDE in the environment is well documented. Bioaccumulation potential is confirmed at least for some isomers, as well as biomagnification in some food chains. As debromination into other POP-like chemicals is expected to be a relevant contribution to the dissipation of hexa to nonaBDE, the absence of food-chain biomagnification for a specific congener on a specific taxonomic group does not necessarily decrease the overall concern. The data available for lower and higher brominated congeners (some of them also present in c-octaBDE) show that they have potential for long-range environmental transport.</td>
</tr>
<tr>
<td>Effects</td>
<td>Unfortunately, the available information on the toxicity and ecotoxicity of hexa to nonaBDE [which make up commercial OctaBDE] is very limited. Effects on mammals and birds include slight fetotoxicity, increased liver weights, and delayed skeletal ossification. Other observed effects include immunotoxicity and neurotoxicity. There is an increasing evidence suggesting similar toxicological profiles and therefore, equivalent hazards and concerns, between PBDEs and PCBs. The potential for formation of polybrominated dibenzo-p-dioxins and furans (See European Communities, 2003 for a general discussion), although not considered in this risk profile, should also be taken into account.</td>
</tr>
<tr>
<td>Exposure</td>
<td>Exposure to components of c-OctaBDE in remote areas is confirmed and</td>
</tr>
</tbody>
</table>
Based on the available information, OctaBDE should be attributed to a combination of releases and transport of c-OctaBDE, c-pentaBDE (for hexaBDE) and c-DecaBDE (for nonaBDE), and to the debromination of DecaBDE in the environment and biota. Despite its large molecular size, the evidence demonstrates the capability of c-OctaBDE components to cross the cellular membranes and to accumulate in biota. The exposure route is mainly via food.

| Status | Octa-BDE takes part of the list of selected substances for the OSPAR lists (no 236). Under the reviewed list, Octa-BDE is put under section C – about the substances put on hold because they are not produced and/or used in the OSPAR catchment or are used in sufficiently contained systems making a threat to the marine environment unlikely. C-OctaBDE is being considered under UNECE Convention on Long range Transboundary Air Pollution (LRTAP) and its Protocol on Persistent Organic Pollutants (POPs). |
| Alternatives | Will be discussed in Annex F evaluation if OctaBDE advances. |
Candidate for POPs List

Octabromodiphenyl ether (commercial mixture) (c-OBDE)
Background

The Stockholm Convention is a global treaty to protect human health and the environment from persistent organic pollutants (POPs), of which twelve are currently listed under the Convention. POPs are chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in living organisms and can cause harm to humans and the environment. The European Union and its Member States, which are Parties to the Stockholm Convention, submitted a proposal in July 2006 for listing octabromodiphenyl ether in Annex A of the Stockholm Convention pursuant to paragraph 1 of Article 8 of the Convention, and the POPRC agreed that the commercial product Commercial octabromodiphenyl ether – actually a mixture as described below - met the screening criteria of Annex D to the Convention.

Introduction

Commercial octabromodiphenyl ether (c-octaBDE) is a mixture of several polybrominated diphenyl ethers and congeners. These synthetic brominated compounds have mainly been used as flame retardants. In addition to octaBDE isomers, c-octaBDE contains significant amounts of other component groups (such as pentabromodiphenyl (pentaBDE) and hexabromodiphenyl ethers) with persistent organic pollutant (POP) characteristics. Specifically, the Persistent Organic Pollutants Review Committee concluded that pentaBDE meets all the criteria specified in Annex D of the Stockholm Convention on Persistent Organic Pollutants and therefore should be considered as a persistent organic pollutant.

Data Sources
c) As penta- and hexabromodiphenyl ethers (which have POP characteristics) occur in c-octaBDE, relevant information about these two compounds is also provided, where appropriate.

1. Identification of the chemical

It is believed that little if any c-OctaBDE is produced since the major supplier located in North America stopped production in 2004. The commercially supplied OctaBDE was complex mixture consisting (as of 2001 within the EU Member States) typically of ≤0.5% Pentabromodiphenyl ether isomers, ≤12% Hexabromodiphenyl ether isomers, ≤45% Heptabromodiphenyl ether isomers, ≤33% OctaBDE isomers, ≤10% Nonabromodiphenyl ether isomers and ≤0.7% Decabromodiphenyl ether. The composition of older products or products from non-EU countries may be different from this.

The c-OctaBDE is sold as a technical grade under the Chemical Abstracts Service (CAS) Registry number for the OctaBDE isomer.

IUPAC Name: Diphenyl ether, octabromo derivative (octabromodiphenyl ether, OctaBDE)

Synonyms: octabromobiphenyl oxide, octabromodiphenyl oxide, octabromo phenoxybenzene and benzene, 1,1’ oxybis-, octabromo derivative

CAS Number: 32536-52-0

Molecular formula: C12H2Br8O

Molecular weight: 801.38

Chemical structure:
Three polybrominated diphenyl ether flame retardants were historically available commercially. They are referred to as penta, octa and decabromodiphenyl ether, but each product is a mixture of diphenyl ethers with varying degrees of bromination. Several synonyms and abbreviations for polybrominated diphenyl ethers exist and these are shown below:

- polybrominated biphenyl ethers ≡ polybromobiphenyl ethers – PBBEs
- polybrominated biphenyl oxides ≡ polybromobiphenyl oxides - PBBOs
- polybrominated diphenyl ethers ≡ polybromodiphenyl ethers - PBDPEs
- polybrominated diphenyl oxides ≡ polybromodiphenyl oxides – PBDPOs

The compositions of the commercial polybrominated diphenyl ethers based on composite samples from the EU suppliers are shown in Table 1. These are the substances that have been used in the recent tests and used as a basis for the EU risk assessment reports for the three commercial substances. These data indicate that if tetra- and pentabromodiphenyl ethers are present in the commercial octabromodiphenyl ether or Decabromodiphenyl ether products, they must be present only at very low levels.

### Table 1. Composition of commercial polybrominated diphenyl ethers as described in the EU RAR.

<table>
<thead>
<tr>
<th>Component</th>
<th>% Composition of commercial product</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tri bromodiphenyl ether</td>
<td>0.23</td>
</tr>
<tr>
<td>Tetra bromodiphenyl ether</td>
<td>33.7</td>
</tr>
<tr>
<td>Penta bromodiphenyl ether</td>
<td>54.6</td>
</tr>
<tr>
<td>Hexa bromodiphenyl ether</td>
<td>11.7</td>
</tr>
<tr>
<td>Hepta bromodiphenyl ether</td>
<td>42.3</td>
</tr>
<tr>
<td>Octa bromodiphenyl ether</td>
<td>36.1</td>
</tr>
<tr>
<td>Nona bromodiphenyl ether</td>
<td>13.9</td>
</tr>
<tr>
<td>Deca bromodiphenyl ether</td>
<td>2.1</td>
</tr>
</tbody>
</table>

There is some discrepancy between the composition of octa bromodiphenyl ether given in the OECD Voluntary Industry Commitment (VIC) and the composition more recently supplied (Table 1), particularly with regard to the levels of the pentabromodiphenyl ether congener. The composition given in the VIC is as follows:

- Hexa/penta bromodiphenyl ether 1.4-12.0%<sup>62</sup>
- Hepta bromodiphenyl ether 43.0-58.0%  
- Octa bromodiphenyl ether 26.0-35.0% 
- Nona bromodiphenyl ether 8.0-14.0% 
- Deca bromodiphenyl ether 0.0-3.0%

<sup>62</sup>The Bromine Science and Environmental Forum (BSEF) suggest that the discrepancies are related to analytical limitations at the time, this represents a total of PentaBDE and HexaBDE congeners, however the majority of this % is believed by BSEF to be the hexaBDE fraction.
In the VIC it is not clear how much if any pentabromodiphenyl ether actually present. No details of the analyses used were provided. Also, at the time the VIC was set up, production of octabromodiphenyl ether was carried out in the EU. Since then, production moved to sites outside the EU, and some producers have stopped producing octabromodiphenyl ether altogether. This may have had some effect on the composition. From the information presented in Table 1 above, it is clear that if pentabromodiphenyl ether is present in the commercial product, it will be at much lower levels than the 12% indicated by the VIC. La Guardia et al (2006) have recently reported additional information on the composition of commercial mixtures.

The commercial mixture covered by this entry is therefore a complex combination of isomers and congeners, as defined at POPRC.

There is a tendency in scientific literature to present the identities of polybrominated diphenyl ether congeners using the numbering system based on the polychlorinated biphenyl system. This risk profile will focus on the series of hexa, hepta, octa and nona homologues:

- Hexabromodiphenyl ethers (benzene, 1,1'-oxybis-, hexaBDE) (CAS No. 36483-60-0; IUPAC No. between BDE-128 and BDE-169)
- Heptabromodiphenyl ethers (benzene, 1,1'-oxybis-, heptaBDE) (CAS No. 68928-80-3; IUPAC No. between BDE-170 and BDE-193)
- Octabromodiphenyl ethers (benzene, 1,1'-oxybis-, octaBDE) (CAS No. 32536-52-0; IUPAC No. between BDE-194 and BDE-205)
- Nonabromodiphenyl ethers (benzene, 1,1'-oxybis-, nonaBDE) (CAS No. 63936-56-1; IUPAC No. between BDE-206 and BDE-208)

2. Persistence

OctaBDE has been found to photodegrade rapidly in a mixture of organic solvents, with a half-life of around 5 hours, but the environmental significance of such a finding is uncertain (European Commission, 2003). Besides, octaBDE is predicted to adsorb strongly onto sediment and soil, which means that only a fraction of this PBDE will be exposed to sunlight, thus having the potential to photodegrade. No information is available on the hydrolysis of octaBDE, but it is not expected to be an important process for octaBDE in the environment.

Regarding biotic degradation, octaBDE is not readily biodegradable in standard tests (no degradation seen over 28 days) and is not expected (by analogy with other brominated diphenyl ethers) to degrade rapidly under anaerobic conditions. Nevertheless, other more highly brominated congeners (deca and nonabromodiphenyl ether) have been found to degrade anaerobically in sewage sludge, although at a very slow rate (Gerecke et al. 2005). The evidence seems to indicate that there is little significant biotic or abiotic degradation of octaBDE.

It's worth noting that degradation of polybrominated diphenyl ethers (PBDEs) can yield byproducts that are lower brominated congeners. For instance, Ahn et al. (2006) showed that decaBDE immobilised on specific soil/sediment and mineral aerosols yielded a number of penta to triBDEs, via octaBDE as an intermediate step. This may pose an additional environmental concern, as these lower brominated diphenyl ethers are usually more toxic and much more bioaccumulative.

3. Bioaccumulation

The log octanol-water partition coefficient (log \(K_{ow}\)) value for the commercial product has been determined to be around 6.29 (European Commission, 2003). Based on its log \(K_{ow}\), octaBDE congener would be expected to be bioaccumulative. However, the experimental result indicates that octaBDE does not bioconcentrate (BCF<9.5), probably due to its large size, which may preclude the crossing of cell walls in organisms.

Nevertheless, other brominated diphenyls present in c-octaBDE have been found to have higher BCFs, for example:
- 11 700 – 17 700 for pentaBDE (European Commission, 2003);
- Up to 5 600 for hexaBDE (European Commission, 2003).
Thus, lower brominated diphenyls have BCF that meet perfectly the accumulation criteria. As they are not only present in c-octaBDE (penta and hexaBDE make up to 12% of the commercial product) but may also appear as a result of the degradation of the higher brominated diphenyls, c-octaBDE can be considered to be bioaccumulative.

The EU Risk Assessment Report (European Commission, 2003) reports that brominated diphenyl ethers with bromine contents both lower and higher than octaBDE have been detected in some biota samples, notably predatory birds' eggs. Theoretically, higher brominated congeners shouldn't accumulate, as they are large molecules which are not likely to go through cell walls. However, the work of Sellström et al. (2005) shows a noticeable accumulation of these substances (hepta and decaBDE, amongst other BDEs) in wild falcons. Verreault et al. (2005) found accumulation of several octaBDE congeners (both higher and lower brominated) in several environmental samples of two Arctic top predators, and De Wit et al. (2006) reported a variety of PBDEs in the Arctic. Therefore, a similar behaviour could be expected from octaBDE. In addition, other studies (Tomy et al. 2004, Stapleton et al. 2004) mention that biotransformation of PBDEs via debromination can lead to bio-accumulation parameters higher than expected, and a consequent biomagnification risk.

By using the benchmark approach proposed by Scheringer (1997) and Beyer et al. (2000) (which suggests that the intrinsic properties of a substance may be evaluated by studying those of similar substances for which more data exist), it is likely that octaBDE is bioaccumulative.

4. Potential for long-range environmental transport

In the EU Risk Assessment Report (European Commission 2003), the vapour pressure of c-octaBDE is reported to be 6.59 x 10^{-6} Pa at 21 °C. Brominated diphenyl ethers as a group all have low vapour pressures, the vapour pressure tending to decrease with increasing bromination. In the same report, the atmospheric half-life for octaBDE is estimated to be 76 days which means that long-range transport is possible for this substance.

Table 1: Water solubility (WS), vapour pressure (VP) and Henry’s Law Constant (HLC) (at 25 °C) for c-octaBDE and currently listed POPs

<table>
<thead>
<tr>
<th>Substance</th>
<th>WS mg/L</th>
<th>VP Pa</th>
<th>HLC Pa m^3/mol</th>
</tr>
</thead>
<tbody>
<tr>
<td>c-octaBDE</td>
<td>0.0005</td>
<td>6.59 x 10^{-6}</td>
<td>10.6</td>
</tr>
<tr>
<td>POP-min (DDT)</td>
<td>0.0012</td>
<td>2.5 x 10^{-5} (DDT)</td>
<td>0.04 (endrin)</td>
</tr>
<tr>
<td>POP-max (toxaphene)</td>
<td>3.0</td>
<td>27 (toxaphene)</td>
<td>3726 (toxaphene)</td>
</tr>
<tr>
<td>POP-2nd max (dieldrin)</td>
<td>0.5</td>
<td>0.04 (heptachlor)</td>
<td>267 (heptachlor)</td>
</tr>
</tbody>
</table>

* EU Risk Assessment Report

Table 1 shows the water solubility, vapour pressure and Henry’s law constant for c-octaBDE, in comparison with the maximum and the minimum for currently listed POPs. Henry's law constant, a key property to determine if there is risk of long-range environmental transport for a substance, is well inside the range set by the other POPs. Considering this fact together with its half-life, it can be concluded that c-octaBDE is quite likely to undergo long-range environmental transport.

There are no monitoring data from remote locations available for octaBDE itself. In general, PBDE concentrations have increased exponentially in arctic biota over the past two decades. The lower brominated congeners (e.g. pentabromodiphenyl ethers and hexabromodiphenyl ethers) present in c-octaBDE appear to be subject to long-range environmental transport, possibly via the atmosphere, as they are widely found in sediment and biota in remote areas (Environment Canada, 2004).

For other brominated congeners, hepta and decaBDE have been demonstrated to occur in airborne particles in the high arctic (Wang et al., 2005). The modelling study by Wania and Dugani (2003, as reviewed in European Commission 2004) concluded that decabromodiphenyl ether was likely to be almost exclusively adsorbed to atmospheric particulates that would effectively control the long-range transport behaviour of the substance. Besides, the presence of decaBDE in moss in relatively remote regions of Norway, and in birds and mammals in Polar Regions, has been attributed to long-range particulate transport (European Commission, 2004).
In summary, the data available for lower and higher brominated congeners (some of them also present in c-octaBDE) show that they have potential for long-range environmental transport. Analysis of c-octaBDE’s chemical properties seems to support this conclusion, as Henry's law constant is very similar to those of acknowledged POPs. Therefore, it can be expected that c-octaBDE is subject to long-range environmental transport.

5. Adverse effects

The available ecotoxicity data for the c-octaBDE product show little or no effect on aquatic organisms (short-term fish study and a longer-term Daphnia magna study), sediment organisms (Lumbriculus variegatus) and soil organisms (three species of plant and earthworms Eisenia fetida) (European Commission 2003). However, the EU Risk Assessment Report identifies a risk of secondary poisoning in other species (via ingestion of earthworms) for the hexabromodiphenyl ether component in the c-octaBDE product (from use in polymer applications).

The EU Risk Assessment Report (European Commission 2003) reviews the available toxicological studies on octaBDE. In that report, the lowest no observed adverse effect level (NOAEL) from the available mammalian toxicity data for the c-octaBDE product is determined as 2 mg/kg bw/day in a developmental study with rabbits. Using this data, a predicted no-effect concentration (PNEC) of 6.7 mg/kg food was derived in the EU Risk Assessment Report. Within the EU, c-octaBDE has been classified as “Toxic”, due to its effects on human health, with the risk phrases "may cause harm to unborn child", and "possible risk of impaired fertility".

The presence of lower brominated diphenyl ethers in the c-octaBDE products is of concern also from the human health point of view as they are likely to have a higher potential to cause adverse effects. WHO (1994) and more recently Birnbaum and Staskal (2004) have reviewed the toxicological data on PBDEs in general.

All the abovementioned studies and assessments provide evidence that c-octaBDE causes adverse effects. The possible formation of brominated dibenzo-p-dioxins and dibenzofurans during combustion or other high temperature processes involving articles containing c-octaBDE is another cause of concern (European Commission, 2003).

Statement of the reasons for concern

“The fact that c-octaBDE consists of several polybrominated diphenyl ethers and congeners makes the assessment of POP characteristics more difficult than in the case of a single compound. However, it can be concluded that c-octaBDE meets the criteria for persistence, potential for long range environmental transport and potential to cause adverse effects. The situation with regards to the screening criteria for bioaccumulation is not so clear cut but the commercial product does contain at least a component group that has been confirmed by the POPRC to meet all the screening criteria (pentabromodiphenyl ether). It also contains hexaBDE, another congener with POP characteristics.

A second aspect of concern is that although the higher brominated BPDEs are persistent, there is evidence that they can degrade under some conditions. Lower brominated diphenyl ether congeners have been identified among the degradation products. Since some of the products may be more bioaccumulative and toxic than the parent compound, any significant formation would be a cause for concern.

An additional risk is the possible formation of brominated dibenzo-p-dioxins and dibenzofurans during combustion and other high temperature processes involving articles treated with c-octaBDE flame retardants.

Marketing and use of octaBDE has been prohibited recently in the EU but it is assumed still to be produced and used as a flame retardant in many countries. As octaBDE and its congeners can move far from their sources, single countries or groups of countries alone cannot abate the pollution caused by it. Due to the harmful POP properties and risks related to its possible continuing production and use, international action is warranted to eliminate this pollution.”
EXECUTIVE SUMMARY

The European Union and its Member States, which are Parties to the Stockholm Convention, submitted a proposal in July 2006 for listing octabromodiphenyl ether in Annex A of the Stockholm Convention pursuant to paragraph 1 of Article 8 of the Convention, and the POPRC agreed that the commercial product Commercial octabromodiphenyl ether – actually a mixture as described below - met the screening criteria of Annex D to the Convention.

The polybrominated diphenyl ethers in general are used as flame retardants of the additive type. They are physically combined with the material being treated rather than chemically combined (as in reactive flame retardants). The commercial products cover several congeners and bromination levels. The information provided by the bromine industry indicates that the octa commercial product has been produced in The Netherlands, France, USA, Japan, UK and Israel, but since 2004, it is no longer produced in the EU, USA and the Pacific Rim and there is no information that indicates it is being produced in developing countries. According to the Bromine Science and Environmental Forum (BSEF), OctaBDE was commercialized sometime in the mid 70’s. By the early 2000’s global production was <4000 tonnes/year and by the time production ceased, demand was <500 tonnes; assuming 30 years of production at 6000 tonnes per year total production volume would be around 180,000 tonnes.

Assuming that the commercial octaBDE is no longer produced, the releases to the environment must be associated to historical processes, as well as to releases during the service life of articles containing the commercial mixtures and at the end of article service life during disposal operations. Switzerland reported for this country diffuse emission from the use of products containing OctaBDE of about 0.37 t/a (based on worst-case estimations) for a total stock of 680 tons.

The persistence of these PBDE in the environment is well documented. The only relevant degradation pathways identified until now are photolysis, anaerobic degradation and metabolism in biota, acting through debromination and producing other BDE which may have higher toxicity and bioaccumulation potential.

The bioaccumulation potential for sediment exposure and particularly for exposure via food is well documented for some c-OctaBDE components. There is also enough toxicokinetic information demonstrating that elimination rates in some vertebrate and invertebrate species are equivalent to those observed for other POPs, with values in the range of 0.01 to 0.5 days⁻¹, equivalent to a half life of about 14 to 70 days assuming first order kinetic. Thus a bioaccumulation potential is confirmed at least for some isomers, as well as biomagnification in some food chains. As debromination into other POP-like chemicals is expected to be a relevant contribution to the dissipation of hexa to nonaBDE, the absence of food-chain biomagnification for a specific congener on a specific taxonomic group does not necessarily decrease the overall concern.

In fact, biota monitoring data in remote areas offer the best demonstration on the potential for long range transport of c-OctaBDE components, in particular for Hexa and HeptaBDE. Theoretically this presence could also be explained by the transport of DecaBDE and its subsequent debromination. However, the comparative analysis of the available information on the physical-chemical properties of the different PBDE homologues indicates that debromination from DecaBDE might contribute to the process but it is not realistic to assume that this explains the process without additional transport from other congeners. Thus, based on the available information a long-range transport is expected for the c-OctaBDE components, and the role of atmospheric transport is confirmed at least for Hexa and HeptaBDE based on its detection in alpine lakes.

Unfortunately, the available information on the toxicity and ecotoxicity of hexa to nonaBDE is very limited and does not offer enough information for presenting sound toxicological and ecotoxicological profiles for each isomer, mixtures of isomers and commercial mixtures.

No relevant effects have been observed in aquatic, sediment and soil laboratory studies; however, this information cannot be used to conclude that Hexa to NonaBDE are not toxic for these organisms.
as the measured endpoints and the exposure conditions, employed in these assays are clearly insufficient for a proper assessment of chemicals such as hexa to nonaBDE.

The available information on mammals and birds offer relevant information. The lowest reported NOAEL for traditional endpoints is 2-5 mg/kg bw/d. The effects are relevant for the health and the ecological assessments and therefore useful for assessing risks for humans and wildlife. In addition, immuno-toxicological effects and particularly delayed neurotoxic effects observed after a single dose require specific attention. A critical body burden for hexa BDE 153 of 2000 µg/kg lipid has been estimated based on a NOEL of 0.45 mg/kg; it should be noted that hexa BDE 153 concentrations close to these value have been found in several species and geographic sites and total PBDE concentrations frequently exceed largely this threshold.

The evaluation of the human and environmental risk of commercial OctaBDE associated to its potential for long range transport is not an easy task as the commercial product is a mixture of components with different properties and profiles, which may also be released to the environment due to its presence as components of other PBDE commercial products and also produced in the environment by debromination of commercial decaBDE.

The greatest difficulty appears for the estimation of the potential hazard of the commercial mixture and its components. There are traditional ecotoxicological and toxicological studies where no effects have been observed even at unrealistically high concentrations. However, an in-depth assessment of these studies considering in particular the properties and toxicokinetic of PBDE indicates that the test design, exposure conditions and measured endpoints are not appropriate for a sound assessment of these types of chemicals. Thus, the lack of effects reported in those tests should be considered with care.

In addition, specific studies have reported particular hazards such as delayed neurotoxicity and immunotoxicity which may be particularly relevant in the assessment of both human health and ecosystem risks.

Based on the existing evidence, additional concerns related to the debromination into toxic BDEs, the increasing evidence relating these chemicals with other POPs (similarities between PBDEs and PCBs; relationships with dioxins and furans), and that under Article 8, paragraph 7(a) of the Convention the lack of full scientific certainty shall not prevent a proposal from proceeding, it is concluded that the components of c-OctaBDE, Hexa to NonaBDE, are likely, as a result of LRET, to lead to significant adverse human health and/or environmental effects, such that global action is warranted.

1. INTRODUCTION

The complexity for setting a risk profile for a complex mixture has been discussed by the POPRC with reference to the commercial mixture of pentabromodiphenyl ether, and the situation is similar for the commercial mixture of octabromodiphenyl ether. Briefly, there are three main conceptual issues:

- Each isomer and congener may have different physicochemical properties, persistence, bioaccumulation potential, toxicological and ecotoxicological profiles and potential for long range transport
- All, most or several isomers and congeners in the mixture may act through the same mechanism of action and the assessment of the individual risk profiles may not be enough for a proper estimation of the overall risk of the commercial mixture due to additive and synergistic effects.
- The debromination in the environment and biota represents an additional source of bromodiphenyl ethers and related metabolites. The metabolites may be more bioavailable and/or toxic than the parent compounds.

The current complexity when analysing this situation is increased by the reduced availability of information as the physical-chemical, fate, and (eco)toxicological information covers in some cases assays with the commercial mixtures, while in other cases the studies focused on mixtures of isomers
and/or homologues, or individual compounds. A full data set for conducting a risk profile is not available for the commercial mixture or for the individual components. Thus the available pieces of information have been combined in this risk profile. The report will present whenever possible the risk expected for the commercial mixture.

Evaluation of commercial octabromodiphenyl ether against the criteria of Annex D

(a) Chemical identity:
   (i) Adequate information was provided in the proposal and supporting information. The proposal relates to commercial octabromodiphenyl ether;
   (ii) The chemical structure for the pure compound octabromodiphenyl ether was provided. Commercial octabromodiphenyl ether is a mixture of several polybrominated diphenyl ethers and congeners (pentabromodiphenyl ether isomers, hexabromodiphenyl ether isomers, heptabromodiphenyl ether isomers, octabromodiphenyl ether isomers, nonabromodiphenyl ether isomers and decabromodiphenyl ether isomers);

   The chemical identity of commercial octabromodiphenyl ether and the pure compound octabromodiphenyl ether is adequately established;

(b) Persistence:
   (i) There was no degradation in an OECD test (301D) over 28 days (Ref. 3);
   (ii) Elevated concentrations of polybromodiphenyl ethers, including octa and hepta bromodiphenyl ether congeners, were found in agricultural soil more than 20 years after treatment of the soil with contaminated sewage sludge, which is consistent with very long half-lives for components of commercial octabromodiphenyl ether (Ref. 2);

   There is sufficient evidence that commercial octabromodiphenyl ether meets the persistence criterion;

(c) Bioaccumulation:
   (i) The log Kow value for the commercial product has been determined to be around 6.29 (Ref. 3). Experimental results presented in the European Union risk assessment report indicates that octa and heptabromodiphenyl ethers have low bioconcentration factors (less than 10–36); these results have been confirmed by data presented and peer reviewed by the Japanese Government. Nevertheless, other brominated diphenyl ethers present in commercial octabromodiphenyl ether have been found to have higher bioconcentration factors, for example 11,700–17,700 for pentabromodiphenyl ethers (Ref. 3) and 1,000–5,600 for hexabromodiphenyl ethers (Ref. 3);

   (ii) and (iii) Field data provide evidence for the potential for bioaccumulation of heptabromodiphenyl ether. Concentrations of 220–270 ng/g lipid weight in eggs of the peregrine falcon in northern Sweden and Greenland have been reported (Refs. 4 and 5). This evidence demonstrates that, despite its large molecular weight, the molecule is found in top predators at levels similar to those of bioaccumulable tetra and penta bromodiphenyl ether. In addition, the estimated half-life in humans is 100 days (Ref. 6), suggesting a potential for bioaccumulation. In soil biota, the soil organism accumulation factor for octabromodiphenyl ether 197 has been calculated as 2 (Ref. 2).

   There is sufficient evidence that commercial octabromodiphenyl ether meets the bioaccumulation criterion;

(d) Potential for long-range environmental transport:
   (i) and (iii) The vapour pressure of commercial octabromodiphenyl ether is reported to be 6.59 x 10^-6 Pa at 21°C (Refs. 1 and 3). The atmospheric half-life of the pure compound octabromodiphenyl ether is estimated to be 76 days, which means that long-range transport is possible for the substance;
(ii) Monitoring data show that the hexa and hepta bromodiphenyl ether congeners are present in biota in remote regions (Refs. 7 and 8) and in Arctic air (Ref. 9); there is sufficient evidence that commercial octabromodiphenyl ether meets the criterion on potential for long-range environmental transport;

(e) Adverse effects:

(i) There are no data provided on the direct toxicological effects of commercial octabromodiphenyl ether or polybromodiphenyl ether congeners in humans;

(ii) There is evidence of reproductive toxicity in mammals. The lowest no observed adverse effect level (NOAEL) from the available mammalian toxicity data for the commercial octabromodiphenyl ether product was determined as 2 mg/kg bw/day in a developmental study in rabbits (Ref. 3). Additional information on the developmental toxicity of octabromodiphenyl ether has been published recently (Ref. 10);

There is sufficient evidence that commercial octabromodiphenyl ether meets the criterion on adverse effects;

The Committee concluded that commercial octabromodiphenyl ether meets the screening criteria specified in Annex D.

Data sources

The EU risk assessment report (European Commission 2003), the Canadian assessment (Environment Canada, 2004), and references from the WHO (1994) report were the main source of information used by the POP RC in Annex D screening.

Additional information has been submitted by Canada, the Czech Republic, Germany, Lithuania, Norway, Switzerland, Turkey, UK, USA, the NGO Environmental Health Fund on behalf of the International POPs Elimination Network (IPEN), and the industry organization Bromine Science and Environmental Forum. Considering the large amount of new scientific information produced nowadays, a review of recent scientific literature has also been conducted and used as an essential data source in this report.

2. STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS

- OSPAR Convention: Octa-BDE takes part of the list of selected substances for the OSPAR lists (No 236). Under the reviewed list, Octa-BDE is put under section C – about the substances put on hold because they are not produced and/or used in the OSPAR catchment or are used in sufficiently contained systems making a threat to the marine environment unlikely.
- UNECE, Convention on Long-range Transboundary Air Pollution (LRTAP) and its Protocol on Persistent Organic Pollutants (POPs): c-OctaBDE is being considered under Protocol procedures for inclusion.

3. SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

3.1 Sources

The information provided by the bromide industry indicates that the commercial product has been produced in The Netherlands, France, USA, Japan, UK and Israel, but since 2004, it is no longer produced in the EU, USA and the Pacific Rim and there is no information that indicates it is being produced in developing countries.

The polybrominated diphenyl ethers in general are used as flame retardants of the additive type. They are physically combined with the material being treated rather than chemically combined (as in
reactive flame retardants). This means that there is the possibility that the flame retardant may diffuse out of the treated material to some extent.

Industry indicates that octabromodiphenyl ether is always used in conjunction with antimony trioxide. In Europe, it is primarily used in acrylonitrile-butadiene-styrene (ABS) polymers at 12-18% weight loadings in the final product. Around 95% of the total octabromodiphenyl ether supplied in the EU is used in ABS. Other minor uses, accounting for the remaining 5% use, include high impact polystyrene (HIPS), polybutylene terephthalate (PBT) and polyamide polymers, at typical loadings of 12-15% weight in the final product. In some applications, the flame retardant is compounded with the polymer to produce pellets (masterbatch) with slightly higher loadings of flame retardant. These are then used in the polymer processing step to produce products with similar loadings as given above.

The flame retarded polymer products are typically used for the housings of office equipment and business machines. Other uses that have been reported for octabromodiphenyl ether include nylon and low density polyethylene (WHO, 1994), polycarbonate, phenol-formaldehyde resins and unsaturated polyesters (OECD, 1994) and in adhesives and coatings (WHO, 1994).

Assuming that the commercial octaBDE is not longer produced, the releases to the environment must be associated to historical processes, as well as to releases during the service life of articles containing the commercial mixtures and at the end of article service life during disposal operations.

The information review by La Guardia et al. (2006) allows estimations of the relative contribution of each congener in different markets and time periods. As an example, Figure 1 presents the calculations for European commercial products in 2001.

![Relative contribution of PBDEs in European commercial formulas from 2001](image)

Figure 1. Estimated relative contribution for the different BDE congeners in products in the European market in 2001. Calculated from data published by La Guardia et al., 2006. Note the logarithmic scale.

Although there are some figures on annual production of this mixture, there are no accurate values on the amount of the commercial octa and/or the individual homologues in articles in service and disposed at the world-wide level, but considering the estimated figure of 6 000 tonnes/year (WHO, 1994) the total amount should be expected in the $10^3 - 10^6$ tonnes range. According to the BSEF, OctaBDE was commercialized sometime in the mid 70’s. By the early 2000’s global production was <4000 tonnes/year and by the time production ceased, demand was <500 tonnes. While Thus, assuming 30 years of production at 6000 tonnes per year gives 180,000 tonnes, a figure within the proposed range.

In a 2002 document (Switzerland info) Switzerland reported for this country diffuse emission from the use of products containing OctaBDE of about 0.37 t/a (based on worst-case estimations) for a total stock of 680 tons.

### 3.2 Environmental fate

#### 3.2.1 Persistence
No aerobic biodegradation of the hexa- to NonaBDEs is expected based on BIOWIN estimates as recalcitrant with respect to biodegradation, and no degradation, based on oxygen uptake, occurred in a 28-day closed bottle test (OECD 301D).

Gerecke et al. (2005) in a study on DecaBDE reported the degradation of nonaBDE 206 and 207 under anaerobic conditions using sewage sludge inoculum to OctaBDEs; and this degradation has been confirmed in other studies (Gaul et al., 2006; He et al., 2006).

AOPWIN predicts half-lives for reaction with atmospheric hydroxyl radicals ranging from 30.4 to 161.0 d for hexa- to NonaBDEs, respectively. In the atmosphere, octaBDE is expected to strongly adsorb to suspended particles in the air and be removed via wet and/or dry deposition. Note that predicted half-lives have not been empirically substantiated, but are provided for reference purposes.

The photodecomposition of several BDEs has been studied in different matrices such as methanol/water 80:20 (Eriksson et al. 2001) a sealed polyethylene tube exposed to natural sunlight for up to 120 min (Peterman et al. 2003); or water (Sanchez-Prado et al., 2006); in general degradation was faster for the higher brominated DEs than for the lower brominated congeners. Rayne et al. (2006) suggest a short photochemical half-life for the hexa BDE153 in aquatic systems, with rapid photolytic debromination to some of the most prevalent penta- and tetra-brominated diphenyl ether congeners.

### 3.2.2 Bioaccumulation

Bioconcentration factors were reported by European Communities (2003) based on the results of a study by CBC (1982), in which carp, Cyprinus carpio, were exposed for 8 weeks to commercial c-octaBDE at 10 or 100 µg/L using polyoxyethylene hydrogenated castor oil as a dispersing agent. If it is assumed that the actual concentrations of the c-octaBDE components were at or around the reported water solubility for the substance of 0.5 µg/L, then the BCF for octaBDE would be <3.5 while the BCF for heptaBDE would be about <1.1-3.8 and the BCF for c-octaBDE would be about <10-36 (European Communities 2003). These BCF values are lower than would be expected from the substance’s octanol-water partition coefficients. This was potentially justified by a reduced bioavailability due to the inability of the large molecule to cross cell membranes (European Communities 2003).

The UK has re-analyzed the CITI (1982b) bioconcentration data and suggests BCFs of up to ~5,640 l/kg and ~ 2,580 l/kg for components D and E (both hexaBDPE).

However, toxicokinetic studies and monitoring data on humans and wildlife reviewed by European Communities 2003, Canada Info 2 and others clearly demonstrate the capability of the large BDE molecules to cross cell membranes. In fact UK info indicates that studies on the levels of hexa- to octaBDE in the environment confirm the presence of hepta and octaBDE isomers in some biota samples. Further data (see below) confirms the bioavailability of large BDE molecules. Thus, the discussion on the molecular size is useless and the evaluation should be related to the capability of BCFs to quantify the bioaccumulation potential of these types of molecules (EU_SCHER, 2005).

In fact, oral exposure is expected to be the most relevant exposure pathway for these chemicals. Van Beusekom et al. (2006) reported biota-sediment accumulation factors between 1 and 3 for hexa and heptaBDE on two freshwater fish species in Spain and concluded that 100% of the exposure was associated to food or food plus sediment for bleak (Alburnus alburnus) and barbel (Barbus graellsii), respectively. The potential for biomagnification has been demonstrated for hexa and heptaBDE (Burreau et al., 2004; Sormo et al., 2006), and more recently suggested for the DecaBDE (Law et al., 2006).

The potential for bioaccumulation and biomagnification of these types of molecules can be calculated using toxicokinetic models, based on metabolism and elimination. Differences among isomers and the reported debromination processes introduce additional uncertainty when reviewing field data.

Ciparis and Hale (2005) have reported a rapid bioaccumulation of hexaBDE in the aquatic oligochaete, Lumbricus variegatus, exposed via sediment, with differences between isomers and in
the contamination pathway. A biota-sediment accumulation factor of 9.1± 1.1 was observed for BDE 154, the highest concentration was found on day 15 and the depuration rate constant was 0.032 ± 0.016 days\(^{-1}\).

Stapleton et al. (2004) in a dietary study on carps found depuration rates of 0.051± 0.036 days\(^{-1}\) and assimilation efficiencies of 4% ± 3 for the hexaBDE 153.

Stapleton and Baker (2003) and Stapleton et al. (2004b) in dietary studies on common carp (Cyprinus carpio) found significant and rapid debromination of heptaBDE183 to hexaBDE154 and to another unidentified hexaBDE congener within the intestinal tissues of the carp after consuming its food. In \textit{in vitro} studies have demonstrated the microsomal debromination in fish (Stapleton et al. (2006) and mammals (McKinney et al., 2006).

The role of exposure levels in the elimination rate of several chemicals including hexaBDE 153 has been studied by the LPTC, Université Bordeaux I and the INIA’s Laboratory for Ecotoxicology within the context of LRI-Cefic Research Project ECO-1AINIA-1100. Depuration rates of 0.03-0.05 for Sparus aurata and Mytilus edulis, were obtained (Alonso et al., 2006).

A recent study (Drouillard et al., 2007) has reported a depuration rate constant for the hexaBDE 0.016 days\(^{-1}\) in juvenile American kestrels (Falco sparverius), with a retention of about 50% of the administered dose.

A controlled feeding trial assessed transfer and accumulation of PBDEs from feed to farmed Atlantic salmon (Salmo salar). On average, 95% of the total PBDE content in the feed accumulated in whole salmon including heptaBDE 183 (Isosaari, et al. 2005).

3.2.3 Long range environmental transport

The presence of components of commercial octa BDE in remote areas (e.g. Norway info, Norway Info 2; Canada info 2; Switzerland info2) is considered the best demonstration for the potential for long range transport of these chemicals. As demobromination in the environment and biota has been demonstrated, hypothetically, the presence of hexa to nonaBDEs could be explained by a long range transport of decaBDE and its subsequent debromination, however, it is very unlikely to assume a long range transport for decaBDE and not for the nona to hexa congeners.

Previous model predictions suggested a low potential for long-range atmospheric transport for highly brominated BDEs (e.g. Wania and Dugani, 2004). However, in a recent paper on DecaBDE, Breivik et al., (2006) have reported that chemicals that are both sorbed to particles and potentially persistent in the atmosphere, such as BDE-209, may have a larger potential for LRT than anticipated on the basis of earlier model evaluations. This explanation could be also applied to c-OctaBDE components.

Recently Wegmann, et al (2007) applied the OECD Pov and LRTP Screening Tool to the current POPs candidates, including c-octaBDE. The authors noted that they believed that the substance property values for c-octaBDE in Wania and Dugani (2003) were more accurate than the values in the POPRC document and therefore included the Wania and Dugani values in their Monte Carlo uncertainty analysis. Although there were considerable uncertainties, the results indicated that c-octaBDE has Pov and LRTP properties similar to those of several known POPs.

3.3 Exposure

- Summary of relevant information concerning exposure in local areas (both near the source and in remote areas)

3.3.1 Atmosphere

Little information is available about concentrations of c-OctaBDE in the atmosphere. A concentration of 52 000 pg/m\(^3\) was predicted for c-OctaBDE in the local atmosphere resulting from emissions from polymer processing (EUSES predictions in European Communities 2003).

Some measurements are available for PBDE congeners present in c-OctaBDE. Strandberg et al. (2001) analyzed air samples from urban, rural and remote sites in the United States near the Great
Lakes. The average total c-OctaBDE-related congeners (i.e., sum of BDEs 153, 154 and 190) present in the samples ranged from approximately 0.2 to 0.9 pg/m^3.

PBDEs (ranging from tri- to OctaBDEs) were detected in deposition samples collected from sites in The Netherlands, Germany and Belgium, confirming their presence in precipitation (Peters 2003). The PBDE composition of the samples could be linked to the commercial penta- and OctaBDE mixes, with BDEs 47, 99 and 154 the predominant congeners.

Bergander et al. (1995) analyzed air samples from two areas of Sweden remote from industry for the presence of c-OctaBDE. No OctaBDE was detected in either the particulate or gas phase samples (the detection limit not stated), but indications of the presence of hexaBDE and heptaBDE were found in the particulate phase samples.

3.3.2 Water

Luckey et al. (2002) measured total PBDE (mono- to heptaBDE congeners) concentrations of approximately 6 pg/L in Lake Ontario surface waters in 1999, with hexaBDE congeners BDE153 and BDE154 each contributing approximately 5 to 8% of the total.

C-OctaBDE was not detected in 1987 in 75 surface water samples taken in Japan at a detection limit of 0.1µg/L or in 1988 in 147 water samples at a detection limit of 0.07 µg/L (Environment Agency Japan 1991). According to European Communities (2003), the concentrations are considered to be representative of industrial, urban and rural areas of Japan, but it is not known whether any of the sampling sites were in the vicinity of a polybrominated diphenyl ether production site or a polymer processing site.

3.3.3 Sediments

Concentrations of c-OctaBDE in UK sediments ranged from <0.44 to 3030 µg/kg dw (Allchin et al. 1999; Law et al. 1996). The highest levels were in sediments downstream from a warehouse where c-DecaBDE was stored (Environment Agency 1997). C-OctaBDE was detected in 3 of 51 sediment samples from Japan in 1987 at concentrations from 8 to 21 µg/kg (detection limit 7 µg/kg; ww or dw not specified), and in 3 of 135 samples collected in 1988 at concentrations of 15 to 22 µg/kg (detection limit 5 µg/kg; ww or dw not specified) (Environment Agency Japan 1991).

Kolic et al. (2004) presented levels of PBDEs in sediments from tributaries flowing to Lake Ontario, and area biosolids in southern Ontario. Total hexa- and heptaBDEs (i.e., BDE 138, 153, 154 and 183) measured in sediment samples taken from fourteen tributary sites (only 6 sites were reported) ranged from approximately 0.5 to 4.0 µg/kg dw.

3.3.4 Soil

Hassanin et al. (2004) determined PBDEs in undisturbed surface soils (0-5 cm) and subsurface soils from remote/rural woodland and grassland sites on a latitudinal transect through the United Kingdom and Norway. In total, 66 surface soils were analyzed for 22 tri- to heptaBDEs. Concentrations of total PBDEs in the surface soils ranged from 0.065 to 12.0 µg/kg dw. Median PBDE concentrations in the surface soils ranged from 0.61 to 2.5 µg/kg dw, with BDEs 47, 99, 100, 153 and 154 dominating the total concentrations. The median concentration of the sum of these five congeners ranged from 0.44 to 1.8 µg/kg dw. The researchers noted that the congener patterns in the European background soils closely matched that reported for the c-pentaBDE mixture. Northward along the latitudinal transect, there was an increasing relative contribution of BDE 47 and other lighter PBDEs in comparison to the heavier PBDEs measured in the samples.

3.3.5 Waste Effluent and Biosolids

Kolic et al. (2004) presented levels of PBDEs in sediments from tributaries flowing to Lake Ontario, and of biosolids from nearby wastewater treatment facilities in southern Ontario. Total hexa- and
heptaBDEs (i.e., BDEs 138, 153, 154 and 183) measured in biosolids ranged from approximately 111 to 178 µg/kg dw.

La Guardia (2001) analyzed 11 sewage sludge samples before land application from Canada and the United States and found that total hexa- to OctaBDE congener concentrations ranged from 40 to 2080 µg/kg dw. Kolic et al. (2003) investigated PBDE levels in sewage sludge from 12 sites in southern Ontario and found hexa- to OctaBDE congener concentrations totaled 124 to 705 µg/kg dw. Hexa- to OctaBDE congeners were not detected in manure samples, and were at very low levels in pulp mill biosolids (up to approximately 3 µg/kg dw).

Martinez et al. (2006) have recently reported concentrations of sum of hexa to nonaBDE in the range of 15.5 to 160 µg/kg dw in sludge from municipal wastewater treatment facilities in Spain, and up to 268 µg/kg dw in industrial facilities.

Gevao et al. (2006) measured PBDEs in coastal sediments receiving industrial and municipal effluents in Kuwait. Total concentrations varied from 80 to 3800 pg/g dw with heptaBDE183 dominating the congener distribution which resembled the commercial formulation, Bromkal 79-8DE. Wastewater discharge from industrial activities appeared to be the primary source of the compounds.

3.3.6 Biota

Concentrations of components found in c-OctaBDEs in biota were reviewed in Law et al. (2003). The concentration of c-OctaBDE (reported as the commercial mixture DE-79) in various biota found in aquatic environments in the UK ranged up to 325 µg/kg ww in the liver of dab (Allchin et al. 1999). Concentrations of OctaBDE in muscle tissue from UK fish ranged from <1 to 12 µg/kg ww (Allchin et al. 1999). In Japan, OctaBDE was not detected in 75 fish samples taken in 1987 (detection limit 5 µg/kg ww), nor was it detected in 144 fish samples taken from 48 locations in 1988-89 (detection limit 4 µg/kg; ww or dw not specified) (Environment Agency Japan 1991). HeptaBDE, along with other PBDE congeners, was detected in eggs of peregrine falcons, Falco peregrinus, from Sweden, at concentrations from 56 to 1300 µg/kg lipid (Lindberg et al. 2004).

Alaee et al. (1999) sampled lake trout from Lakes Superior, Huron and Ontario and found that the total of hexaBDE and heptaBDE congeners ranged from an estimated 11 to 53 µg/kg lipid.

Rice et al. (2002) compared PBDE levels and congener patterns in carp and bass sampled from two industrialized regions in the eastern U.S. The fish were collected from the Detroit River, MI. and the Des Plaines River, IL. in May and June of 1999, and analyzed for the presence of BDEs 47, 99, 100, 153, 154, 181, 183 and 190. Both river systems are considered to receive high contributions from municipal and industrial effluents. BDE47 dominated in fish taken from the Detroit River, comprising an average of 53 to 56% of the total PBDEs by wet weight. BDEs 99, 100, 153 and 154 each contributed between 8 and 9%, and BDEs 181 and 183 each comprised about 5% of the total PBDEs. BDE190 was not detected in either fish species. Only carp were sampled from the Des Plaines River, and these exhibited a markedly different PBDE profile from that seen in the Detroit River fish. HeptaBDEs 181 and 183 were predominant, contributing about 21% and 19%, respectively. BDE47 was third in prevalence, comprising about 17% of the total PBDEs. Levels of the two hexaBDE congeners, BDEs 153 and 154 were 8 to 13%, compared with about 5% for each of the penta-congeners, BDEs 99 and 100. BDE190, not detected in the Detroit River fish, was present at about 12% of total PBDE.

The congener profile identified in the Detroit River fish, with predominance of the tetra- and pentaBDE congeners, was consistent with patterns reported in biota from other parts of North America and the world. The greater prevalence of higher brominated congeners evident in the Des Plaines River fish, however, was atypical. The authors postulated that the unusual congener pattern displayed in fish from the Des Plaines River may relate to the nature of wastewater discharges from manufacturing or waste facilities in the region. The higher quantities of hexa- and heptaBDEs evident in the fish samples may reflect higher discharge volumes of commercial OctaBDE products, so that levels measured in fish represent a combination of commercial pentaBDE and OctaBDE sources. The researchers note that the possible sources for the other heptaBDEs, BDE 181 and BDE 190 that were found in the carp in Des Plaines River are not obvious especially since no commercial products have been documented as containing major quantities of these congeners. The authors speculate that the
significant presence of heptaBDE in the Des Plaines River fish may result from active metabolism of BDE209 present in the river sediment; however river sediment concentrations were not obtained in this study. They also suggest that differing contributions to the two river systems from municipal wastewater treatment facilities potentially played a role, but that congener determinations in effluent sources were also not determined in this study.

Norstrom et al. (2002) evaluated the geographical distribution and temporal trends (during the 1981 to 2000 period) of PBDEs in herring gull (Larus argentatus) eggs from a network of colonies scattered throughout the Great Lakes and their connecting channels in 2000 (see Section 2.1.6.6 and Appendix D). Although samples were analyzed for octa- to DecaBDE, these were not found at their respective limits of detection (0.01-0.05 µg/kg ww). However, total concentrations of hexa- and heptaBDE congeners (i.e., BDEs 153,154 and 183) increased 6 to 30 fold over the 1981 to 2000 period at the Lake Michigan (from 6.7 to 195.6 µg/kg ww), Lake Huron (from 13.8 to 87.6 µg/kg ww) and Lake Ontario (3.8 to 112.1 µg/kg ww) sites. This increase was not as dramatic as that found for the tetra- and pentaBDE congeners.

Wakeford et al. (2002) conducted sampling of wild bird eggs in western and northern Canada between 1983 and 2000. They determined that the total of hexa- and heptaBDE congeners ranged from 0.148 to 52.9 µg/kg ww in Great Blue Heron (Ardea herodias) eggs (on Canada’s west coast), 0.03 to 0.68 µg/kg ww in Northern Fulmer (Fulmarus glacialis) eggs (in the Canadian arctic) and 0.009 to 0.499 µg/kg ww in Thick Billed Murre (Uria lomvia) eggs (in the Canadian arctic). OctaBDE, nonaBDE and DecaBDE congeners were subject to analysis by the researchers, but were not detected (detection limit was not specified) in the any of the samples.

3.3.7 Humans

European Communities (2003) presents some information on the levels of components of c-octaBDE measured in human samples. Large variations among individuals were generally observed, but significant differences between the control population and occupationally exposed groups were also reported.

In a recent study (Toms et al., 2007) the concentrations of PBDEs found in Australian human milk were lower than those reported from North America but higher than those reported from Europe and Asia.

- Summary of relevant information concerning exposure as a result of LRET

Measured levels of components of c-OctaBDE in biota from remote areas seem to be the best available information for estimating exposure as result of LRET for these chemicals. Knudsen et al (2005; Norway info) have recently review temporal trends of PBDE in eggs from three bird species, three locations and three sampling times (from 1983 to 2003) from Northern Norway. Spatial differences were only observed for hexaBDE 153, and increases in the measured concentration from 1983 to 2003 were observed for the hexaBDE 153 and 154 and the heptaBDE 183. Mean values were around 1 µg/kg ww for each isomer and maximum values above 10 µg/kg ww were observed for BDE 154 and 183. Inter species differences could be associated to feeding behavior and migration. In general the concentrations were lower than those reported for similar species in industrialized areas and those observed in terrestrial predatory birds.

Exposure to components of c-OctaBDE in remote areas is confirmed and based on the available information should be attributed to a combination of releases and transport of c-OctaBDE, c-pentaBDE (for hexaBDE) and c-DecaBDE (for nonaBDE), and to the debromination of DecaBDE in the environment and biota. There is no sufficient information for assessing these processes in quantified terms. The exposure route is mainly via food, and even for water column animals water exposure is of low, if any, relevance; therefore the BCFs are not suitable parameters for estimating the bioaccumulation potential.

In addition to the feeding strategy, several additional confounding factors are associated to the species to specific differences observed in the isomer distribution pattern of PBDE in wildlife. These factors include, among others, species-specific differences in assimilation, metabolism and depuration of different isomers, even with the same level of bromination.
The presence of hexa and heptaBDE in fish from remote alpine lakes in Switzerland (Switzerland info2) reported to be related to atmospheric deposition confirms the potential for atmospheric long-range transport.

- **Information on bioavailability**

Despite its large molecular size, the evidence demonstrates the capability of c-OctaBDE components to cross the cellular membranes and to accumulate in biota. Exposure from water is not relevant and significant assimilation rates have been observed for oral and sediment exposures. Although the information is limited, the assimilation and metabolisms of each isomer may vary significantly among species, but also in relation to the administered dose. As a consequence, it is essential to understand the toxicokinetics of these chemicals at environmentally relevant concentrations.

These differences would justify the disparities observed in the assessment of biomagnification potential for different trophic chains.

Like for other chemicals with similar properties, aging processes are expected to reduce the bioavailability, and the experiments conducted on sediment dwelling organisms comparing the bioaccumulation in spiked sediments and from contaminated biosolids offer and indirect support for this hypothesis.

### 3.4 Hazard assessment for endpoints of concern

#### Experimental studies

**Aquatic Organisms**

The EU Risk Assessment report (European Communities 2003), presents a set of studies on the commercial mixture and concludes that for water it seems sensible to assume that no adverse effects on aquatic organisms are likely to occur at concentrations up to the substance’s water solubility. However it must be noted, first, that aquatic organisms are also exposed from food and/or sediment; and second, that setting this strong conclusion on chemicals such as PBDEs requires multigenerational or at least full life-cycle assays on the three taxonomic groups covering a large list of sublethal effects, information which is unavailable at this time.

**Benthic Organisms**

There are two available 28 day spiked sediment studies on *Lumbriculus variegatus* using the c-OctaBDE product (Great Lakes Chemical Corporation 2001a, b). These studies found no statistically significant effects relevant to survival, reproduction or growth at the highest tested concentration (1272 mg/kg dw and 1340 mg/kg dw measured for sediments with 2.4% and 5.9% OC, respectively). Kinetic data from Ciparis and Hale (2005) confirms the expected exposure and bioaccumulation under these conditions.

**Soil Organisms**

Survival and growth of earthworms, *Eisenia fetida*, were not affected by a 56 day exposure to a commercial OctaBDE formulation in an artificial soil at concentrations up to 1470 mg/kg dw (measured concentration in sediments with 4.7% OC) (Great Lakes Chemical Corporation 2001c).

The toxicity of c-OctaBDE to corn (*Zea mays*), onion (*Allium cepa*), ryegrass (*Lolium perenne*), cucumber (*Cucumis sativa*), soybean (*Glycine max*), and tomato (*Lycopersicon esculentum*) was evaluated in a 21-day emergence and growth study using an artificial sandy loam soil (Great Lakes Chemical Corporation 2001d). No statistically significant effects were observed for any plant species between the controls and the treatments for emergence, survival or growth at any of the tested concentrations (up to 1190 mg/kg dw, measured concentration).

**Mammals and Birds**

The lowest reported NOAEL for traditional endpoints is a NOAEL of 2 mg/kg/d based on slight fetotoxicity at 5 mg/kg/d (considered relevant in the EU report) or 5 mg/kg bw/d based on increased liver weights and decreased body weight gain among the maternal treatment group and delayed fetal skeletal ossification at 15 mg/kg bw/d (for those reviewers that do not consider relevant the slight
fetotoxicity effects) described by Breslin et al. (1989) in a developmental toxicity study with Saytex 111 on New Zealand White rabbits exposed orally via gavage over days 7 to 19 of gestation.

Effects on other endpoints have been described at lower concentrations, including:

- A significant increase in EPN detoxification and p-nitroanEROD and isole demethylation in male Sprague-Dawley rats at an oral dose of 0.60 mg/kg bw/day OBDE formulation for 14-days.
- dose-dependent depletion of serum total thyroxine T4 and induced pentoxyresorufin O-deethylase (PROD) activities in rats receiving 10 or more mg/kg bw/day of commercial octabromo (Zhou et al. 2001)
- Delayed neurotoxic effects. Neonatal mice exposed to a single dose of 0.45 mg BDE153/kg bw on postnatal day 10 showed when tested at 2, 4, and 6 months of age altered motor behavior. Spatial learning ability and memory function in the adult mice were also affected (Viberg et al. 2001)
- Eriksson et al. 2002a confirmed neurotoxic effects (aberrant behavioral responses) on developing male mice exposed to 0.45 to 9.0 mg/kg bw of BDE153 on day 10 of development. The effects were comparable to those observed for PCB153 leading the authors to speculate that interactive neurotoxic action may be possible between the two compounds.
- These neurotoxic effects have also been observed after a single oral dose of nonaBDE 206 or OctaBDE 203 administered on postnatal day 3 or 10 to, or PBDE 183; with disturbances in spontaneous behavior, leading to disrupted habituation and a hyperactive condition in adults at the age of 2 months. (Viberg et al., 2006).
- Immunomodulation effects in captive nestling American kestrels (*Falco sparverius*) have been reported by Fernie et al. (2005). Eggs within each clutch, divided by laying sequence, were injected with safflower oil or penta-BDE congeners-47, -99, -100, and -153 dissolved in safflower oil (18.7 microg PBDEs/egg). For 29 days, nestlings consumed the same PBDE mixture (15.6+/−0.3 ng/g body weight per day), reaching PBDE body burden concentrations that were 120x higher in the treatment birds (86.1+/−29.1 ng/g ww) than controls (0.73+/−0.5 ng/g ww). PBDE-exposed birds had a greater PHA response (T-cell-mediated immunity), which was negatively associated with increasing BDE-47 concentrations, but a reduced antibody-mediated response that was positively associated with increasing BDE-183 concentrations. There were also structural changes in the spleen (fewer germinal centers), bursa (reduced apoptosis) and thymus (increased macrophages), and negative associations between the spleen somatic index and PBDEs, and the bursa somatic index and BDE-47. Immunomodulation from PBDE exposure may be exacerbated in wild birds experiencing greater environmental stresses.
- Fernie et al., 2006 also reported for the same species and test conditions that exposure did not affect hatching or fledging success. PBDE-exposed nestlings were larger (weight, bones, feathers) as they gained weight more quickly and ate more food, the latter in association with their PBDE body burdens. BDE-100 was most influential on nestling growth, being positively associated with size, weight gain, and food consumption. Increasing concentrations of BDE-183 and -153 were related to longer bones and BDE-99 to longer feathers. The larger size of the PBDE-exposed birds may be detrimental to their bone structure and have excessive energetic costs.
- In vitro studies indicates that BDE (including the hexaBDE 153) affected protein kinase C (PKC) and calcium homeostasis in cerebellar granule neuronal cultures in a similar way to those of a structurally-related polychlorinated biphenyl (PCB) (Kodavanti et al., 2005)

**Monitoring data on effects**

There are several scientific papers comparing population effects observed in the field with measured concentrations of POP like chemicals, including hexa to nonaBDE in individuals from different species.

Unfortunately, wild populations are co-exposed to a mixture of PBDEs as well as to other related brominated and chlorinated persistent pollutants, and with the current level of knowledge epidemiological investigations can just present associations but no cause-effect relationships between
the exposure/accumulation of the components of the commercial OctaBDE mixtures and potential adverse effects observed in wildlife.

A similar situation is observed regarding human health data, and no studies offering conclusive evidence on the hazards of hexa to nonaBDE for humans at environmentally relevant exposure levels have been found.

4. SYNTHESIS OF THE INFORMATION
The evaluation of the specific risks of commercial OctaBDE is complex and uncertain as the consequence of:

- the presence of its components in commercial penta- and DecaBDE,

- the additional transformation in the environment through debromination, in particular the evidence suggesting a significant role of debromination of Octa to DecaBDE to other PBDE congeners in biota is highly relevant as food is expected to be the main exposure route for these chemicals, although increases the difficulties for quantitative assessments, and

- mostly, because the lack of a solid body of toxicological and ecotoxicological information for the mixture and its components covering the long-term low level exposure conditions and the sublethal endpoints considered relevant for assessing the risk of a POP candidate.

In this risk profile, hexa to nonaBDE have been considered the relevant components in c-OctaBDE. It should be noted that other BDE are also found in commercial mixtures, but those are expected to be covered by the c-penta and c-deca entries. It should be noted that theoretically this represent 80 different congeners, while the available information focuses on less than ten congeners and some mixtures.

The persistence of these PBDE in the environment is well documented. The only relevant degradation pathways identified until now are photolysis, anaerobic degradation and metabolism in biota, acting through debromination and producing other BDE which may have higher toxicity and bioaccumulation potential.

The bioaccumulation potential of these highly brominated BDEs cannot be described by the BCF as waterborne exposure is of low if any relevance. As bioaccumulation is the result of assimilation efficiency, metabolism and elimination rate, a reduced assimilation does not necessarily result in low bioaccumulation potential. In fact, if metabolisms and elimination rates are very low, the reduced assimilation will result in longer times for achieving the steady state conditions, but for species with long life spans a significant bioaccumulation may be expected.

The bioaccumulation potential for sediment exposure and particularly for exposure via food is well documented for some hexa to nonaBDE. There is also enough toxicokinetic information demonstrating that elimination rates in some vertebrate and invertebrate species are equivalent to those observed for other POPs, with values in the range of 0.01 to 0.5 days⁻¹, equivalent to a half life of about 14 to 70 days assuming first order kinetic; and therefore, low enough for supporting a justified concern on the bioaccumulation potential of Hexa to NonaBDE. Monitoring programs measuring PBDE concentrations in biota conducted in industrialized and remote areas confirm this potential for bioaccumulation.

It should be noted that differences in both assimilation and metabolism, and therefore in the overall bioaccumulation potential, have been observed not only among homologues, but also between isomers with the same level of bromination. Inter-species differences have also been reported. These differences may explain the discrepancies in terms of food chain biomagnification potential described in the literature.

In summary, a bioaccumulation potential is confirmed at least for some isomers, as well as biomagnification in some food chains. As debromination into other POP-like chemicals is expected to be a relevant contribution to the dissipation of hexa to nonaBDE, the absence of food-chain biomagnification for a specific congener on a specific taxonomic group does not necessarily decrease the overall concern.
In fact, biota monitoring data in remote areas offer the best demonstration on the potential for long range transport of c-OctaBDE components, in particular for Hexa and HeptaBDE. Theoretically this presence could also be explained by the transport of DecaBDE and its subsequent debromination. However, the comparative analysis of the available information on the physical-chemical properties of the different PBDE homologues indicates that debromination from DecaBDE might contribute to the process but it is not realistic to assume that this explains the process without additional transport from other congeners. Thus, based on the available information a long-range transport is expected for the c-OctaBDE components, and the role of atmospheric transport is confirmed at least for Hexa and HeptaBDE based on its detection in alpine lakes.

Unfortunately, the available information on the toxicity and ecotoxicity of hexa to nonaBDE is very limited and does not offer enough information for presenting sound toxicological and ecotoxicological profiles for each isomer, mixtures of isomers and commercial mixtures.

No relevant effects have been observed in aquatic, sediment and soil laboratory studies. The uptake of BDEs at least for the sediment exposure is expected within the time-frame of the experiment, based on toxicokinetic studies. However, this information cannot be used to conclude that Hexa to NonaBDE are not toxic for these organisms. In fact, the measured endpoints and the exposure conditions, employed in these assays are clearly insufficient for a proper assessment of chemicals such as hexa to nonaBDE. Ecotoxicity tests on these types of chemicals should cover if possible several generations or at least a full life cycle, and the measured endpoints must include sublethal effects associated to the accumulation and re-mobilization of the PBDEs during critical periods of development and reproduction, as well as the ecologically relevant consequences of metabolic changes. In addition, all environmentally relevant exposure routes must be addressed. The available tests do not fulfill these conditions, thus, although no toxicity was reported, no concluding statements on the toxicity of hexa to OctaBDE congeners for aquatic, sediment and soil dwelling organisms can be presented.

The available information on mammals and birds offer relevant information. The lowest reported NOAEL for traditional endpoints is 2-5 mg/kg bw/d based on slight fetotoxicity or increased liver weights and decreased body weight gain among the maternal treatment group and delayed fetal skeletal ossification. These effects are relevant for the health and the ecological assessment and therefore useful for assessing risks for humans and wildlife. Nevertheless, the additional available information also creates concerns on the capability of these traditional endpoints for assessing the toxicological profile of hexa to nonaBDE in mammals and other vertebrates.

The metabolic effects, their relevance and their level of coverage by the endpoints measured in the experimental toxicity test should be considered.

However, the immuno-toxicological effects and particularly the delayed neurotoxic effects observed after a single dose require specific attention. Although a quantitative evaluation of these effects in terms of its potential risk for human health and ecosystem is not possible based on the current level of information, the reported observations must be analyzed with care. Certainly, the doses at which the effect have been observed are well above exposure levels in remote areas estimated from current monitoring data for a single congener. However, the effects have been observed for different congeners, and realistic environmental exposure occurs for a mixture of PBDEs. There is not enough information for considering if these effects may be additive or even more than additive in synergistic exposures. The margins between effects observed in the lab and estimated oral exposure levels in the field (based on monitoring data) are not so high when the different isomers/homologues are sum. McDonald (2005) estimated a critical body burden for hexa BDE 153 of 2000 µg/kg lipid based on the NOEL of 0.45 mg/kg reported by Viberg et al 2003 and gives a margin of safety of 7 between this level and the 95 percentile of total PBDE levels in US human populations. It should be noted that hexa BDE 153 concentrations close to these value have been found in several species and geographic sites (see Canada info 2 for a review) and total PBDE concentrations frequently exceed largely this threshold.

Three additional concerns must be mentioned in the risk profile of PBDEs:
• First, the reported debromination of highly brominated BDEs in the environment and biota suggests a potential for the formation of congeners with demonstrated POP properties such as those included in the risk profile of c-pentaBDE.

• Second, there is an increasing evidence suggesting similar toxicological profiles and therefore, equivalent hazards and concerns, between PBDEs and PCBs, although the mode of action seems to be better categorized by AhR-independent mechanisms, as PBDEs do bind but not activate the AhR-AhR nuclear translocator protein-XRE complex (Peters et al., 2006) and appear capable of up-regulating CYP2B and CYP3A in rats at doses similar to that for non-dioxin-like PCB153 (Sanders et al., 2005). As the persistence, bioaccumulation potential and long range transport of the c-octaBDE components are well documented, the confirmation of an equivalent level of hazard for these two groups should be sufficient for confirming a long-range transport associated risk.

• Third, the potential for formation of polybrominated dibenzo-p-dioxins and furans (See European Communities, 2003 for a general discussion), although not considered in this risk profile, should also be taken into account.

5. CONCLUDING STATEMENT

The evaluation of the human and environmental risk of commercial OctaBDE associated to its potential for long range transport is not an easy task as the commercial product is a mixture of components with different properties and profiles, which may also be released to the environment due to its presence as components of other PBDE commercial products and also produced in the environment by debromination of commercial decaBDE.

Although the production of c-OctaBDE has ceased in developed countries and there is no information suggesting that the chemical is produced elsewhere; it must be noticed that the product is still present and released from articles in use and during their disposal. Model estimations and measured levels in sewage sludge suggest that current emissions are still significant.

The persistence of the hexa to nonaBDE is well documented and the main route of degradation is debromination forming other BDEs, also of concern. The potential for certain components in c-OctaBDE to bioaccumulate and also for biomagnification in some trophic chains is also sufficiently documented and confirmed by the good agreement between field observations in monitoring programmes and toxicokinetic studies. Monitoring data in remote areas confirm the potential for long-range transport and at least for some congeners the relevance of atmospheric distribution in this process.

The highest difficulty appears for the estimation of the potential hazard of the commercial mixture and its components. There are traditional ecotoxicological and toxicological studies where no effects have been observed even at unrealistically high concentrations. However, an in-depth assessment of these studies considering in particular the properties and toxicokinetic of PBDE indicates that the test design, exposure conditions and measured endpoints are not appropriate for a sound assessment of these types of chemicals. Thus, the lack of effects reported in those tests should be considered with care.

In addition, specific studies have reported particular hazards such as delayed neurotoxicity and immunotoxicity which may be particularly relevant in the assessment of both human health and ecosystem risks.

Based on the existing evidence, additional concerns related to the debromination into toxic BDEs, the increasing evidence relating these chemicals with other POPs (similarities between PBDEs and PCBs; relationships with dioxins and furans), and that under Article 8, paragraph 7(a) of the Convention the lack of full scientific certainty shall not prevent a proposal from proceeding, it is concluded that the components of c-OctaBDE, Hexa to NonaBDE, are likely, as a result of LRET, to lead to significant adverse human health and/or environmental effects, such that global action is warranted.
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Viberg et al, 2003 reported in Mc Donalds, 2005.


B.8. Pentachlorobenzene – SUMMARY

<table>
<thead>
<tr>
<th><strong>Composition</strong></th>
<th>Pentachlorobenzene belongs to the group of chlorobenzenes, which are characterized by a benzene ring in which the hydrogen atoms are substituted by one or more chlorines.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Uses</strong></td>
<td>No current intentional use believed though PeCB was a component of a chlorobenzenes mixture used to reduce the viscosity of PCB products employed for heat transfer. Formerly, PeCB and TeCB could be found in dyestuff carriers. PeCB can be found as an impurity in several herbicides, pesticides and fungicides currently in use in Canada (Environment Canada, 2005). Pentachlorobenzene was identified in pentachloronitrobenzene (quintozene), endosulfan, chlorpyrifos-methyl, atrazine, and clopyrillid, but not in simazine, chlorothalonil, picloram and dacthal (US EPA, 1998). Technical grade hexachlorobenzene (HCB) contains about 98 % HCB, 1.8 % pentachlorobenzene and 0.2 % 1,2,4,5-tetrachlorobenzene (WHO-IPCS, 1997). The available data suggest a decrease in pentachlorobenzene use for the preparation of quintozene. However, this conclusion is based on data for Europe and North America only. PeCB may have been used in the past as a fungicide and as a flame retardant (Van de Plassche et al., 2002).</td>
</tr>
<tr>
<td><strong>Releases</strong></td>
<td>Currently, PeCB is believed to come primarily from unintentional production from sources that include: PCBs, chlorinated solvents, pesticides, chemical manufacturing, aluminum casting, waste combustion including barrel burning, ore treatment for metal production of magnesium, copper, niobium, tantalum, titanium dioxide production, wood treatment plants, and hazardous waste incineration.</td>
</tr>
<tr>
<td><strong>Fate</strong></td>
<td>PeCB is spread widely in the environment on a global scale. Levels of PeCB in abiotic and biotic media in remote regions such as the (ant) arctic environment are available, as well as monitoring data on PeCB in abiotic and biotic media of temperate zones. Pentachlorobenzene has a high bioaccumulation potential. Due to the fact that biotransformation of PeCB will be insignificant and the substance is very hydrophobic, the compound may also have a high biomagnification potential.</td>
</tr>
<tr>
<td><strong>Effects</strong></td>
<td>PeCB is moderately toxic to humans. Animal studies reveal effects including decreased thyroxin, abnormal sperm, and histopathological effects on the kidneys. Pentachlorobenzene is very toxic to aquatic organisms and may cause long-term adverse effects in the aquatic environment.</td>
</tr>
<tr>
<td><strong>Exposure</strong></td>
<td>PeCB has been detected in breast milk and found to accumulate in human placenta (Shen et al., 2007). PeCB has also been measured in abdominal, mammary, and perirenal fat tissue from 27 adult Finnish males and females (Smeds and Saukko, 2001). Workers with occupational exposure to PeCB were found to have higher levels of the substance in blood than control groups (Lunde and Bjorseth, 1977). PeCB has been found in Alaskan grayling, lake trout, cod, and halibut and in Greenland krill, cod, and arctic char. It is also found in seals from northern Russia, Canada, and Greenland and in whale blubber from Canada and musk ox blubber from Greenland. PeCB is found in polar bears from the arctic Svalbard islands, Alaska, Canada, and East</td>
</tr>
</tbody>
</table>
Greenland. PeCB is present in arctic fox and Canadian snow crabs.

| Status | The European Commission has submitted a proposal to include pentachlorobenzene to the Protocol to the 1979 Convention on Long Range Transboundary Air Pollution (LRTAP) on Persistent Organic Pollutants to the Executive Secretariat of the United Nations Economic Commission for Europe in 2006. PeCB is identified as a priority substance within the European Water Framework Directive (2000/60/EC). Within the list of these priority substances so-called priority hazardous substances are identified which are of particular concern for the freshwater, coastal and marine environment. These substances will be subject to cessation or phasing out of discharges, emissions and losses within 20 years after adoption of the Directive. The European Commission has proposed to include pentachlorobenzene as a priority hazardous substance. [COM(2006) 397 final]. PeCB is listed on the OSPAR 1998 List of Candidate Substances (OSPAR, 1998). |
| Alternatives | Will be discussed in Annex F evaluation if PeCB advances. |
Candidate for POPs List

Pentachlorobenzene
Introduction

Pentachlorobenzene belongs to the group of chlorobenzenes. This substance has been used in the past as a pesticide, flame retardant, and in combination with PCBs in dielectric fluids. It is not clear whether it is still used as a pesticide or flame retardant on its own; however, it can be found as an impurity of pentachloronitrobenzene (quintozene) and other pesticides such as Clopyralid, Atrazine, Chlorothalonil, Dacthal, Lindane, pentachlorophenol, Picloram and Simazine. It may be emitted to the environment indirectly: as a result of waste incineration and barrel burning of household waste; in waste streams from pulp and paper mills, iron and steel mills, and petroleum refineries; and in activated sludge from waste water treatment facilities. Pentachlorobenzene is not produced commercially in the UNECE Europe any more (Belfroid et al. 2005).

1. Identification of the chemical

1.1 Names and registry numbers

CAS\textsuperscript{63}/IUPAC\textsuperscript{64} chemical name: Pentachlorobenzene

Synonyms: 1,2,3,4,5-pentachlorobenzene; benzene, pentachloro-; quintochlorobenzene; PeCB

Trade names: None

CAS registry number: 608-93-5

EINECS\textsuperscript{65} Number: 210-172-0

1.2 Structure

![Molecular Structure of Pentachlorobenzene]

Molecular formula: $\text{C}_6\text{HCl}_5$

Molecular weight: 250.32 g/mol

2 Persistence

According to CEPA (1993) pentachlorobenzene (PeCB) can be photo-oxidized in the atmosphere, largely through reactions with hydroxyl (OH) radicals. There are no experimental data on atmospheric degradation, but the estimated half-life of PeCB is 45 to 467 days. Vulykh et al. (2005) estimate a half life in air of 65 days based on modelling data.

Although photodegradation in surface water is fast under sunlight irradiation (41% loss after 24 h), under field conditions the strong adsorption to solids may counteract this process (HSDB, February 2000). The half-life of PeCB in surface water was estimated to range from 194 to 1 250 days, while the estimated half-life for the anaerobic biodegradation in deeper water ranged from 776 to 1 380 days (CEPA, 1993).

Although anaerobic degradation occurs, half-lives for anaerobic degradation are still high. In sediment cores at Ketelmeer (the Netherlands), PeCB is apparently persistent, i.e., several years in the

\textsuperscript{63} Chemical Abstracts Service.

\textsuperscript{64} International Union of Pure and Applied Chemistry.

\textsuperscript{65} European Inventory of Existing Chemical Substances.
presence of native anaerobic microflora. A special mixed culture of anaerobic species showed a half-life of several days (Beurskens et al., 1994). Beck and Hansen (1974) observed a half-life of 194-345 days in soils.

In sediments and soils oxygen is generally scarce, which favours reductive dechlorination. Information on PeCB's degradation pathways is scarce. Most work for the higher chlorinated benzenes has been done on hexachlorobenzene, for which the first step in the proposed pathway is dechlorination to PeCB. Further dechlorination leads to monochlorobenzene (Van Agteren et al., 1998).

Min-Jian Wang et al. (1994, 1995) have done research on the behaviour and fate of chlorobenzenes (CB) in spiked and sewage sludge-amended soil from a long-term (from 1942 to 1961) agricultural experiment. Their conclusion is that about 10% of the applied total CBs became recalcitrant and that the main loss of CBs is by volatilisation. Half-lives of 219 and 103 days were reported for PeCB. It is expected that if PeCB is released to soil, it will absorb strongly to the soil and will not leach to the groundwater. It will not be expected to be subject to significant hydrolysis or biodegradation.

PeCB appear to be very persistent in soils, water and the atmosphere, based on actual measurements and experimental estimates.

3 Bioaccumulation

Measured and calculated log K\textsubscript{ow} values for PeCB vary between 4.8 and 5.18. The measured BCF values based on whole body wet weight vary between 3 400 and 13 000. In many cases the BCF value exceeds the limit of 5 000, indicating a high potential for accumulation. Van de Plassche (1994) reviewed the information on bioconcentration in fish and molluscs for PeCB and derived a geometric mean BCF for fish of 5 300. CEPA (2002) reports BAFs of 810 in mussels (Mytilus edulis), 20 000 in rainbow trout (Oncorynchus mykiss) and 401 000 for earthworms (Eisenia andrei).

4 Potential for long-range environmental transport

The vapour pressure of PeCB is 2.2 Pa at 25 °C and the calculated half-life in air is 277 days, ranging from 45 to 467 days (Van de Plassche et al. 2002). These two properties seem to indicate that PeCB is very likely to undergo long range environmental transport (LRET). In table 1 the values for water solubility, vapour pressure and Henry's Law Constant for PeCB can be compared with the maximum and the minimum for currently listed POPs. Henry's Law Constant, a key property to determine if there is risk of long range environmental transport for a substance, is well inside the range set by the other POPs, which also supports the potential for long-range transport of PeCB.

Table 1: Water solubility (WS), vapour pressure (VP) and Henry's Law Constant (HLC) for pentachlorobenzene and currently listed POPs.

<table>
<thead>
<tr>
<th>Substance</th>
<th>WS mg/L</th>
<th>VP Pa</th>
<th>HLC Pa m\textsuperscript{3}/mol</th>
</tr>
</thead>
<tbody>
<tr>
<td>PeCB</td>
<td>0.56 *</td>
<td>2.2 *</td>
<td>983.4 **</td>
</tr>
<tr>
<td>POP-min</td>
<td>1.2 x 10\textsuperscript{-3} (DDT)</td>
<td>2.5 x 10\textsuperscript{-5} (DDT)</td>
<td>4 x 10\textsuperscript{-2} (endrin)</td>
</tr>
<tr>
<td>POP-max</td>
<td>3.0 (toxaphene)</td>
<td>27 (toxaphene)</td>
<td>3 726 (toxaphene)</td>
</tr>
<tr>
<td>POP-2\textsuperscript{nd} max</td>
<td>0.5 (dieldrin)</td>
<td>0.04 (heptachlor)</td>
<td>267 (heptachlor)</td>
</tr>
</tbody>
</table>

* Van de Plassche et al. 2002
** Calculated from VP and WS

There is also evidence based on modelling data. Mantseva et al. (2004) developed a multi-compartment transport model for the evaluation of long-range atmospheric transport and deposition of POPs. Based on this model assessment a transport distance in Europe of over 8 000
km has been calculated for PeCB. The modelling performed by Vulykh et al. (2005) gives a similar value of 8,256 km.

PeCB has been detected in air samples collected at 40 sampling stations in North America (Canada, USA, Mexico, Belize and Costa Rica), including 5 Arctic stations (Shen et al. 2005). The air concentrations were almost uniform across North America with an average concentration of 0.045 ng/m³ and a range 0.017 – 0.138 ng/ m³. According to the authors, this small spatial variability across the northern hemisphere indicates that PeCB has a very long atmospheric residence time, which allows it to become widely distributed in the global atmosphere.

In Sweden, PeCB was also detected in all 8 analyzed air samples (median 0.033 ng/m³) and in two atmospheric deposition samples (max 0.16 ng/m²/day) collected in the Stockholm area (Kaj and Palm, 2004).

In all six bottom sediments from harbours of northern Norway and the Russian Kola Peninsula in the Arctic, PeCB ranged from 2-5 µg/kg dry weight (AMAP 2004). These concentrations are similar to those detected in 3 of the 20 freshwater sediment samples collected in 2002 in the Stockholm area (Sternbeck et al. 2003). The maximum concentration was 6 µg/kg dry weight. In another study, PeCB was detected in Swedish sediment samples (4 out of 6 samples, median 1 µg/kg dry weight) (Kaj and Palm 2004).

PeCB was detected in fish muscle collected in 2002 at Swedish marine and freshwater sites regarded as uncontaminated. Kaj and Dusan (2004) measured 2.2 ng PeCB/g lipid weight in herring from one location and a maximum 16 ng PeCB/g lipid weight in perch from two other locations.

In the Netherlands, PeCB was detected in all 10 flounder liver samples collected in 1996, including flounder from two relatively unpoluted reference locations (De Boer et al., 2001). The highest concentration was 1100 µg/kg lipid weight (280 µg/kg wet weight), and at the reference location 3 ng/g lipid weight (0.64 ng/g wet weight). Also in 2003, PeCB was detected in 50% of the freshwater fish samples (eel and pike perch) in concentrations ranging between 1-10 ng/g wet weight (Van Leeuwen et al. 2004).

PeCB has been detected in different arctic species. Vorkamp et al. (2004) analysed PeCB in biota from Greenland and measured the following concentrations in lipid (lw) and wet weight (ww):
(a) Ptarmigan liver approximately 23 ng/g lw (1.5 ng/g ww);
(b) Kittiwake muscle approximately 8 ng/g lw (1.1 ng/g ww);
(c) Musk ox blubber approximately 0.32 ng/g lw (0.29 ng/g ww);
(d) Arctic char approximately 3.9 ng/g lw (0.07 ng/g ww).

A study by Verreault et al. (2005) showed that PeCB was present in the adipose tissue of polar bears in a variety of Arctic populations (Alaska, Canada, East Greenland and Svalbard Islands). PeCB was also detected in all 15 plasma and fat samples of polar bears from the arctic Svalbard Islands with an average concentration of 7.9 and a maximum of 13.3 ng/g wet weight (Gabrielsen et al. 2004). PeCB was detected in soils and mosses from coastal areas of Victoria Land (Antarctica) (Borghini et al. 2005). Concentrations in the six mosses samples varied between 1-2.4 ng/g dry weight and in the four soil samples between 0.4 and 1.3 ng/g dry weight.

The modelling and monitoring data, as well as PeCB's chemical properties, indicate that this substance has a considerable potential for long-range environmental transport.

5 Adverse effects

PeCB is classified within the EU as "Harmful if swallowed" and "Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment". A review of the different adverse effects of PeCB can be found in the report by Van de Plassche et al. (2002). PeCB has been tested on rats and mice. Acute toxicity tests were available after oral and dermal exposure. The lowest LD50 value after oral exposure was for rats, i.e. 250 mg/kg body weight. In a study in which the rats were orally exposed to 250 mg/kg body weight/day during 3 days, some liver functions were increased. To determine a dermal LD50 one concentration (i.e.2500 mg/kg body weight) was tested on rats, but no toxic effects were seen at this dose. In a subchronic toxicity study oral uptake of 25 mg/kg body
weight and more resulted in effects on the liver and kidneys (increased weight and histopathological changes). A concentration of 12.5 mg/kg body weight has been determined as the no-observed-effect-concentration (NOEC). In a 15 day study by McDonald for the National Toxicology Program (1991) the no-observed effect levels (NOELs) for histologic lesions were 33 mg/kg for male rats and 330 mg/kg body weight for female rats. The NOEL for histologic lesions in female mice was 100 mg/kg body weight. A NOEL was not reached for male mice.

PeCB was classified in Group V (inadequate data for evaluation) of the classification scheme for carcinogenicity (CEPA, 1993). Regarding teratogenic effects, suckling pups from mothers fed a dose of 12.5 mg/kg body weight developed tremors 4 - 14 days after birth. At a maternal dose of 6.3 mg/kg body weight this effect did not occur in pups. In another study, pregnant female rats were administered PeCB at levels of 50, 100 and 200 mg/kg body weight daily on day 6 through 15 of gestation. The number of live foetuses was not affected; however, the mean foetal weight was decreased in the highest dose group (Sloof et al., 1991).

Acute toxicity data for freshwater organisms is available on algae, crustaceans and fish. Chronic toxicity data is only available on crustaceans and fish. For marine organisms, only acute toxicity data is available for fish. Based on the data available, marine and freshwater organisms do not seem to differ significantly in sensitivity to PeCB. The lowest LC50 value for fresh water organisms is 250 µg/L for fish. The lowest NOEC reported is 10 µg/L for crustaceans.

Based upon acute and subchronic animal tests, PeCB is probably moderately toxic to humans and based on experiments with aquatic spieces, toxic for a range of aquatic organisms.

**Reasons for concern**

"PeCB is persistent in soil, water and the atmosphere. It has been shown to bioconcentrate in different species and to be toxic to aquatic organisms. It is also widely found in humans and biota in the environment as a result of its long range transport.

Although its production seems to have ceased in Europe and North America, it is still present as an impurity in commercial pesticides which are still used, and it is unclear whether it may be used as a pesticide or flame retardant in other parts of the world. As PeCB can move in the atmosphere far from its sources, single countries or groups of countries alone cannot abate the pollution caused by it. Due to its harmful POP properties and risks related to its possible continuing production, use and releases to the environment, international action is warranted to control this pollution."

Data sources:


RISK PROFILE

EXECUTIVE SUMMARY

The European Community and its Member States being Parties to the Stockholm Convention have proposed pentachlorobenzene (PeCB) to be listed to the Convention. The Persistent Organic Pollutants Review Committee concluded that pentachlorobenzene fulfilled the screening criteria set and decided to establish an ad hoc working group to review the proposal further.

Most of the countries who submitted information to the UNEP secretariat reported no production or use of PeCB (Czech Republic, Germany, Lithuania, Mauritius, Turkey, Canada), which is in agreement with the information in the dossier submitted. Past uses of PeCB are PeCB as a component in PCB products, in dyestuff carriers, as a fungicide and a flame retardant and as a chemical intermediate e.g. for the production of quintozene. Major U.S. and European manufacturers of quintozene have changed their manufacturing process to eliminate this use of PeCB. PeCB is also present at low levels as an impurity in several herbicides, pesticides and fungicides. In the United States, some pesticide manufacturers have changed their manufacturing processes to reduce the concentration of HCB impurities in their products, and these changes may have reduced concentrations of PeCB contaminants also, PeCB is also a low level degradation product of some pesticides. Literature sources show that pentachlorobenzene is of no economic significance. No trade or stockpiles have been reported.

Nowadays pentachlorobenzene enters the environment through various sources of which PeCB as a byproduct of incomplete combustion is the most important. However, there is considerable uncertainty on the release of PeCB by various sources and available data are limited to the United States and Canada. The limited data available makes it difficult to provide a proper global estimate on amounts and trends. Total estimated annual global emissions of PeCBs based on the US-TRI database were 85,000 kg/yr.

PeCB should be considered as persistent given the estimated and experimental half lives in atmosphere, soils, sediments, and water. According to the available data pentachlorobenzene has a high bioaccumulation potential. Log \( K_{ow} \) values vary between 4.88 and 6.12, with recommended values of 5.17-5.18. BCF values range from 1085 - 23000 L/kg for fish, 833 – 4300 L/kg for mollusca, and 577 – 2258 L/kg for crustacea. Due to the fact that biotransformation of PeCB will be insignificant and the substance is very hydrophobic, the compound may also have a high biomagnification potential.

PeCB is moderately toxic to humans and is not classified as a carcinogen. Within the European Union PeCB is classified as a substance which is very toxic to aquatic organisms (LC50 for fish, daphnia or algae ≤ 1 mg/L). Limited data are available on terrestrial ecotoxicity and data for toxicity to birds are lacking.

Physical-chemical characteristics, such as water solubility, vapour pressure and Henry’s Law Constant, are within the range of the other POPs. PeCB can be photo-oxidized in the atmosphere, largely through reactions with hydroxyl (OH) radicals. However, estimated half-lives of PeCB in air of 45 to 467 days were reported. Considering its physical-chemical characteristics and persistence in air, PeCB has a potential for long range transport through the atmosphere. This is supported by the presence of PeCB in environmental compartments, including biota, from remote regions.

PeCB is spread widely in the environment on a global scale. Levels of PeCB in abiotic and biotic media in remote regions such as the (ant) arctic environment are available, as well as monitoring data on PeCB in abiotic and biotic media of temperate zones. In general, data from developed countries indicates that concentrations of PeCB in the temperate zones of the world seem to decrease. For the (ant)arctic area, only recent data are available which do not allow to derive a trend.

Based on the available evidence, pentachlorobenzene is likely, as result of its long range environmental transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted.
INTRODUCTION

The European Community and its Member States being Parties to the Stockholm Convention have proposed pentachlorobenzene to be listed in Annex A, B and/or C to the Convention pursuant to paragraph 1 of Article 8 of the Convention.

The acceptance of the original proposal for further consideration by the Persistent Organic Pollutants Review Committee implies that the properties of the substance fulfilled the screening criteria set out in Annex D of the Convention.

All data in this document are presented according to the International System of Units (SI) and, therefore, many have been recalculated from other units in the data sources. Furthermore, all concentrations are presented based on kg or L (e.g. µg/kg or mL/L).

1.1 Chemical Identity of the proposed substance

1.1.1 Names and registry numbers

Pentachlorobenzene belongs to the group of chlorobenzenes, which are characterised by a benzene ring in which the hydrogen atoms are substituted by one or more chlorines. The chlorobenzenes are neutral, thermally stable compounds with increasing stability and higher melting and boiling points with increasing chlorine substitution. Pentachlorobenzene has a very low solubility in water (Rossberg et al., 2006).

IUPAC Name: benzene, pentachloro-

CAS Chemical Name: 1,2,3,4,5-pentachlorobenzene; Pentachlorobenzene; PCB; PeCB; QCB; quintochlorobenzene

CAS Registry Number: 608-93-5
EINECS Number: 210-172-0
Trade names: -

1.1.2 Structure

1,2,3,4,5-Pentachlorobenzene

1.1.3 Physical chemical properties

The physical and chemical properties of pentachlorobenzene are listed in Table 1.1. Vapour pressure increases with temperature. Mackay et al (2006) provided a recommended value of 0.11 Pa at 20 °C. Water solubility at 25 °C varied between 0.135 and 3.46 mg/L, whereas the recommended value in various sources was around 0.55 mg/L. The log Kow values in Mackay et al (2006) varied between 4.88 and 6.12. This source and the PHYSPROP and CHEMFATE databases recommend values of 5.17-5.18 as most reliable.

Table 1.1 Physical and chemical properties of pentachlorobenzene
<table>
<thead>
<tr>
<th>Property</th>
<th>Unit</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular formula</td>
<td></td>
<td>( C_6HCl_5 )</td>
<td></td>
</tr>
<tr>
<td>Molecular weight</td>
<td>g/mol</td>
<td>250.34</td>
<td>US NIST 2005, Rossberg et al., 2006</td>
</tr>
<tr>
<td>Appearance at normal temperature and pressure</td>
<td></td>
<td>colorless needles white crystalline solid</td>
<td>Rossberg et al., 2006 Government of Canada, 1993</td>
</tr>
<tr>
<td>Vapour pressure (^{66})</td>
<td>Pa</td>
<td>133 (at 98.6 °C)</td>
<td>Stull, 1947 in Mackay et al., 2006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.212 (at 25 °C)</td>
<td>Polednicek et al., 1996 in Mackay et al., 2006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.11 (at 20 °C)</td>
<td>Rohac et al., 1999 in Mackay et al., 2006, recommended</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.22 Pa at 25 °C (est.)</td>
<td>Weast, 1972 -1973 in Government of Canada, 1993</td>
</tr>
<tr>
<td>Water solubility (at 25 °C)</td>
<td>mg/L</td>
<td>0.551 mg/L</td>
<td>Yalkowsky et al., 1979 in IUPAC-NIST solubility database recommended value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.562 mg/L</td>
<td>Horvath, 1982 in Mackay et al., 2006 recommended value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.552 mg/L</td>
<td>Horvath &amp; Getzen, 1985 in Mackay et al., 2006 recommended value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>86</td>
<td>Weast et al., 1985 in SRC Chemfate database</td>
</tr>
<tr>
<td>Boiling point</td>
<td>°C</td>
<td>277</td>
<td>SRC PhysProp database</td>
</tr>
<tr>
<td></td>
<td></td>
<td>277</td>
<td>Weast et al., 1985 in SRC Chemfate database</td>
</tr>
<tr>
<td>Log ( K_{ow} )</td>
<td></td>
<td>5.17</td>
<td>Sangster, 1993 in Mackay et al., 2006 recommended value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.18</td>
<td>Hansch et al., 1995 in Mackay et al., 2006 recommended value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.17 (exp.)</td>
<td>Hansch et al., 1995 in SRC PhysProp database</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.17</td>
<td>Hansch &amp; Leo, 1985 in SRC Chemfate database</td>
</tr>
</tbody>
</table>

\(^{66}\) Relationship between vapour pressure and temperature provided in Mackay et al., 2006
<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log $K_{oc}$</td>
<td>4.77</td>
<td>Karickhoff et al., 1979 in WHO-IPCS, 1991</td>
</tr>
<tr>
<td></td>
<td>4.24 (calculated)</td>
<td>US EPA superfund (n.d.)</td>
</tr>
<tr>
<td></td>
<td>4.51 (measured)</td>
<td>US EPA superfund (n.d.)</td>
</tr>
<tr>
<td>Henry’s Law constant</td>
<td>Pa m$^3$/mol</td>
<td>Ten Hulscher et al., 1992 in Mackay et al., 2006</td>
</tr>
<tr>
<td></td>
<td>59.0 at 20 °C</td>
<td>Staudinger &amp; Roberts, 1996, 2001 in Mackay et al., 2006</td>
</tr>
<tr>
<td></td>
<td>(measured range</td>
<td>Oliver, 1985 in Mackay et al., 2006</td>
</tr>
<tr>
<td></td>
<td>14.8-50.5 °C)</td>
<td>Ten Hulscher et al., 1992 in SRC PhysProp databas</td>
</tr>
<tr>
<td></td>
<td>52.6 at 20 °C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>71.9 at 25 °C</td>
<td></td>
</tr>
<tr>
<td></td>
<td>71.23 at 25 °C (exp.)</td>
<td></td>
</tr>
</tbody>
</table>

1) WHO-IPCS mentions abusively Karlockoff et al., 1979
2) Mackay et al. (2006) provide a value of 59.0 at 25 °C, but from the data at the end of the chapter on PeCB it appears that this refers to the measured value at 20 °C.

n.d. = no date

1.3 Data sources

- CHEMFATE Database: Environmental Fate Data Base (EFDB) at Syracuse Research Centre. Available at: [http://www.syres.com/esc/efdb.htm](http://www.syres.com/esc/efdb.htm)

Where the reviews mentioned above have been cited, the text quoted (or quoted with modifications) includes the references cited in the original review. These references are not shown individually in the reference list. The following parties and observers have answered the request for information specified in Annex E of the Convention: Canada, Czech Republic, Germany, Japan, Lithuania, Mauritius, Switzerland, Turkey, United States of America, International POPs Elimination Network (IPEN), and the International Council of Chemical Associations/World Chlorine Council (ICCA-WCC).

Additional information was retrieved through a search using Current Contents and through a search on the Internet using various public databases. Search terms used were pentachlorobenzene, PeCB, 608-93-5, bioaccumulat*, bioconc*, biomagnificat*, BSAF*, BCF*, BMF*, BAF*, elimination, uptake, persist*, degradation, resistance, half-life, toxic*, release and inventory. The databases...

1.4 Status of the chemical under international conventions

Pentachlorobenzene is not included in any international convention. The European Commission has submitted a proposal to include pentachlorobenzene to the Protocol to the 1979 Convention on Long Range Transboundary Air Pollution (LRTAP) on Persistent Organic Pollutants to the Executive Secretariat of the United Nations Economic Commission for Europe in 2006 (European Commission, 2007). The provisions of the Protocol oblige Parties to phase out all production and uses of pentachlorobenzene.

PeCB is identified as a priority substance within the European Water Framework Directive (2000/60/EC). Within the list of these priority substances so-called priority hazardous substances are identified which are of particular concern for the freshwater, coastal and marine environment. These substances will be subject to cessation or phasing out of discharges, emissions and losses within 20 years after adoption of the Directive. The European Commission has proposed to include pentachlorobenzene as a priority hazardous substance. [COM(2006) 397 final]. PeCB is listed on the OSPAR 1998 List of Candidate Substances (OSPAR, 1998).

2 SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

2.1 Sources

Production, use and sources of release have been described extensively in the two documents submitted by Canada (Government of Canada, 1993, 2002), the proposed risk management strategy for Pentachlorobenzene by Canada (Environment Canada, 2005) and the document submitted by the ICCA/WCC (2007). Additional information was retrieved from the documents submitted by other Parties and Observers and from the open literature.

2.1.1. Production, trade, stockpiles

The submission document for PeCB reported that PeCB was not produced anymore within Europe and North America (Van de Plassche et al., 2002). PeCB has not been reported by EU Industry as an HPVC or LPVC (http://ecb.jrc.it/esis/). Most of the countries who submitted information to the UNEP secretariat reported no production (Czech Republic, Germany, Lithuania, Mauritius, Turkey, and Canada). WHO-IPCS (1991) reported no manufacturing within the USA in 1985. The USA reported that PeCB is subject to a Toxic Substances Control Act (TSCA) Significant New Use Rule requiring notification to EPA prior to manufacture, import or processing of 10,000 pounds (4,536 kg) or more of PeCB per year per facility for any use. No such notification has been received. No intentional production was mentioned in the document submitted by the ICCA/WCC and according to Ullmann’s Encyclopedia of Industrial Chemistry pentachlorobenzene is of no economic significance (Rossberg et al., 2006). No trade or stockpiles have been reported.

2.1.2. Uses
Canada and the USA reported that there is no current domestic commercial demand for pentachlorobenzene and that PeCB is not used as an end product. Ullmann's Encyclopedia of Industrial Chemistry does not mention any present use of PeCB (Rossberg et al., 2006). However, various past uses or unintentional uses of PeCB are mentioned in the literature:

1. PeCB was a component of a chlorobenzenes mixture used to reduce the viscosity of PCB products employed for heat transfer (Environment Canada, 2005). However, after regulations prohibiting new uses of PCB-containing dielectric fluids were introduced in 1980, the amount of pentachlorobenzene used for this purpose declined considerably in Canada. Based on the results of a survey small amounts of pentachlorobenzene (40 kg during the first 6 months of 1992) were still imported into Canada in dielectric fluids for use in the maintenance of transformers (Government of Canada, 1993). PCBs are still in use in some old electrical equipment in North America and Europe so that there is a small potential for release of PeCB from this source (Environment Canada, 2005). It can be presumed that some PCBs are also still in use elsewhere in the world and some fraction of them contain PeCB. PCBs are being taken out of service in many countries of the world so that any related PeCB emissions are expected to decrease with time.

2. Formerly, PeCB and TeCB could be found in dyestuff carriers. The applications in dye carriers have been discontinued (Environment Canada, 2005). It is not clear from the Canadian document if PeCB, TeCB or both have been used in dyestuff carriers.

3. PeCB can be found as an impurity in several herbicides, pesticides and fungicides currently in use in Canada (Environment Canada, 2005). The US EPA carried out a study to assess the dietary cancer risk of hexachlorobenzene and pentachlorobenzene as impurities in chlorothalonil, PCNB, picloram, and several other pesticides. Pentachlorobenzene was identified in pentachloronitrobenzene (quintozene), endosulfan, chlorpyrifos-methyl, atrazine, and clopyralid, but not in simazine, chlorothalonil, picloram and dacthal (US EPA, 1998). Technical grade hexachlorobenzene (HCB) contains about 98 % HCB, 1.8 % pentachlorobenzene and 0.2 % 1,2,4,5-tetrachlorobenzene (WHO-IPCS, 1997). HCB is already listed in annex A and C of the Stockholm convention and it may thus be expected that HCB is of minor importance as a source for PeCB. The present situation for the other pesticides is unknown.

4. The use of PeCB as chemical intermediate is mentioned in WHO-IPCS (1991). So far, only the use as an intermediate in the manufacture of pentachloronitrobenzene (quintozene) has been found in the literature. PeCB is present as an impurity in this fungicide. Quintozene has been commercially produced since the 1930s (US EPA TRI 2001). In 2000 there were at least two producers of quintozene in the EU and several more suppliers. Quintozene was only authorized for use in the UK, Ireland, France, Spain and Greece (RPA, 2000). In 2000 authorisations for plant protection products in the EU containing quintozene were withdrawn (European Commission, 2000). Quintozene is currently used, but not produced, in Canada (Environment Canada, 2005). HSDB mentions one manufacturer in the US citing a reference from 1989. Production in the US in 1979 was at least 4.54x10^5 kg (HSDB, 2003). Van de Plassche et al. (2002) report on the production and use of quintozene in various countries and indicated that the use outside the UNECE region is unknown. Van de Plassche et al. (2002) stated: ‘Nowadays, quintozene is manufactured using another production process without PeCB. Amvac does not know of any current quintozene producer using PeCB as feedstock. They conclude that it is unlikely that there are any stockpiles of quintozene containing appreciable quantities of PeCB.’ Feiler (2001) in ICCA/WCC (2007) reported that quintozene is now being made by chlorination of nitrobenzene instead of using PeCB as an intermediate. The available data suggest a decrease in pentachlorobenzene use for the preparation of quintozene. However, this conclusion is based on data for Europe and North America only.

5. PeCB may have been used in the past as a fungicide and as a flame retardant (Van de Plassche et al., 2002). WHO-IPCS (1991) mentions that PeCB was formerly used in a pesticide to combat oyster drills. No further sources of these applications have been found.

6. Less than 0.1 kg per year of pure pentachlorobenzene was imported into Canada from the United States for use as a laboratory reagent (Government of Canada, 1993). The use as laboratory reagent, based on data applicable to 1995, is also mentioned in Government of Canada (2002). The present situation is unknown.
From the data submitted and data in the literature it is obvious that production and use of PeCB in Europe and North America are negligible. The situation in other parts of the world is less clear.

2.1.3. Releases to the environment

The proposed risk management strategy for pentachlorobenzene prepared by Environment Canada in 2005 mentions various routes through which PeCB can be released into the Canadian environment (Environment Canada, 2005). The main sources of release in Canada are given in Table 2.1.

<table>
<thead>
<tr>
<th>Sources</th>
<th>Releases in kg/yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Air</td>
</tr>
<tr>
<td>Barrel burning of household waste</td>
<td>1.814</td>
</tr>
<tr>
<td>Wood treatment plants and in service utility poles</td>
<td>2.24</td>
</tr>
<tr>
<td>Pesticide use</td>
<td>6.2</td>
</tr>
<tr>
<td>Dielectric fluid spill and cleanup</td>
<td>0.004</td>
</tr>
<tr>
<td>Municipal solid waste incineration</td>
<td>0.364</td>
</tr>
<tr>
<td>Hazardous waste incineration</td>
<td>1.835</td>
</tr>
<tr>
<td>Magnesium production</td>
<td>1.449</td>
</tr>
<tr>
<td>Solvent use</td>
<td>0.037</td>
</tr>
<tr>
<td>Long-range transport</td>
<td>n.a.</td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
</tbody>
</table>

As potential sources of release are mentioned:
1. Magnesium production (less than 2% of total annual releases)
2. Chlorinated solvents (negligible)
3. Secondary copper and aluminium processing (no data)
4. Chemical manufacturing (unlikely)
5. Iron and steel mills (scarcity of data)
6. Petroleum Refineries (unlikely)
7. Wastewater treatment plants (unlikely)
8. Textile mills (unlikely)
9. Long range transport (amount not known, expected to decrease)

The sources of release and potential sources are described more extensively in Environment Canada (2005).

The total release provided by Environment Canada in the risk management strategy of PeCB (Environment Canada, 2005), 41.8 kg/yr, is a factor of 10 lower than the release of >580 kg/yr provided in the Priority substances list assessment report for pentachlorobenzene (Government of Canada, 1993), submitted by Canada for the drafting of this Risk profile. Main differences can be related to the phasing out of pentachlorobenzene in dielectric fluids which changed the release from 180 kg/yr to 5.6 kg/yr, PeCB in pesticides, which changed from 17 to 6.2 kg/yr, PeCB release through solvent use, which decreased from 4 to 0.04 kg/yr and long range transport and deposition which accounted for 286 kg/yr in the 1993 report and which was reported as n.a. in 2005. The 2005 document indicates that the amount of 286 kg/yr should be regarded as an overestimation, and that the other emissions are expected to be reduced through a combination of different national and international measures (Environment Canada, 2005). The most important sources in the Canadian risk management report (Environment Canada, 2005), barrel burning of household waste (21.93 kg/yr), municipal solid waste incineration (2.36 kg/yr), hazardous waste incineration (1.84 kg/yr) and magnesium production (1.53 kg/yr), were not identified as sources in 1993.
Data on releases of PeCB in the USA can be found in the U. S. EPA Toxics Release Inventory (TRI). The TRI contain release data for 2000 – 2004, which are summarized in Table 2.2 (US EPA 2007a, http://www.epa.gov/tri/tridata/index.htm#pdr).

Pentachlorobenzene was not included in the decision of the European Commission on the implementation of a European Pollutant Emission Register (EPER) (2000/479/EC), but it is in the regulation concerning the establishment of a European Pollutant Release and Transfer Register (E-PRTR) (EC/166/2006). No data on PeCB are available yet.

Table 2.2. Releases inventory for PeCB in the U.S between 2000 and 2004 in kg/year.

<table>
<thead>
<tr>
<th>Year</th>
<th>Air emissions</th>
<th>Surface water discharges</th>
<th>Underground injection</th>
<th>On-site releases to land</th>
<th>Transfers off-site to disposal</th>
<th>Total releases on- and off-site</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>74</td>
<td>79</td>
<td>5</td>
<td>1192</td>
<td>161</td>
<td>1512</td>
</tr>
<tr>
<td>2001</td>
<td>34</td>
<td>60</td>
<td>0</td>
<td>1069</td>
<td>105</td>
<td>1270</td>
</tr>
<tr>
<td>2002</td>
<td>37</td>
<td>61</td>
<td>1</td>
<td>875</td>
<td>391</td>
<td>1365</td>
</tr>
<tr>
<td>2003</td>
<td>40</td>
<td>5</td>
<td>0</td>
<td>606</td>
<td>111</td>
<td>763</td>
</tr>
<tr>
<td>2004</td>
<td>100</td>
<td>8</td>
<td>0</td>
<td>1006</td>
<td>38</td>
<td>1151</td>
</tr>
</tbody>
</table>

The ICCA/WCC provided a document with an estimation of the annual global emissions of PeCB based on the U. S. Toxics Release Inventory (TRI) (ICCA/WCC, 2007). A total of 20 industrial facilities are listed as emitting or transferring 2533 pounds (1151 kg) of PeCB in 2004. The industries represented are chemical, waste treatment, and coal burning electric power. Total reported air emissions were 220 pounds (100 kg) and water emissions of 17 pounds (7.8 kg). In industrial chlorination reactions it is possible to produce PeCB as a byproduct and it probably accounts for some of the emissions reported in the TRI. PeCB formation has been observed during combustion of PVC, a common component of municipal solid waste (Kim et al., 2004; Aracil et al., 2005; Muller et al., 1997). There are other processes which produce a variety of chlorinated aromatics that may contribute to PeCB even if PeCB has not been explicitly detected and reported yet. TRI includes only industrial facilities handling relatively large amounts of chemicals so that additional emissions are expected.

Total estimated annual global emissions of PeCBs by ICCA/WCC (2007) were 85,000 kg/yr, about 2000 times the amount estimated for Canada and 850 times the total release of the United States. Most of the emission sources are similar with those provided in the Canadian risk management document (Environment Canada, 2005), but some are different. Hazardous waste incineration and wood treatment plants are lacking in the ICCA/WCC study, whereas combustion of coal and combustion of biomass, which amounts half of the total global emissions, are lacking in the Canadian study. Although chemical manufacturing was thought to be unlikely as a source, the highest reported chlorobenzene concentrations in Canadian sediment have been observed near industrial sites (Government of Canada, 2003).

In conclusion, pentachlorobenzene can enter the environment through various sources of which PeCB as a byproduct of incomplete combustion is the most important. Nearly all fuels contain some chlorine, especially biomass and waste. In industrial chlorination reactions it is possible that PeCB is produced as a byproduct and it probably accounts for some of the emissions reported. For a number of potential sources, such as copper and aluminium processing plants and steel mills no or limited data are available. From the data provided in the various documents one may expect a decrease of releases through past intentional use, due to phasing out of PeCB. In the case of unintentional releases a decrease can be expected in those cases where measures were taken to reduce the releases of other compounds or materials. The global estimate should be considered taking into account these uncertainties and the variation in industrial and waste handling processes among the various countries.
2.2 Environmental fate
2.2.1 Persistence

Pentachlorobenzene (PeCB) can be photo-oxidized in the atmosphere, largely through reactions with hydroxyl (OH) radicals (CEPA, 1993). There are no experimental data on atmospheric degradation, but the estimated half-life of PeCB is 45 to 467 days. For PeCB, the calculated half-life in air based on reaction with OH-radicals is 277 days (EPISUITE, US EPA, 2007b). Vulykh et al. (2005) estimate a half-life in air of 65 days based on modelling data. This estimate is the result of degradation as well as dry and wet deposition and gaseous exchange with various surfaces. The atmospheric half-life of PeCB due to the degradation process only is estimated to be 155 days.

In the OECD TG 301C test PeCB was non-biodegradable (NITE, 2007). Photodegradation of PeCB is fast in surface water under sunlight irradiation: 41% loss after 24 hours (HSDB, February 2000). The half-life of PeCB in surface water was estimated to range from 194 to 1250 days, the estimated half-life for the anaerobic biodegradation in deeper water ranged from 776 to 1380 days (CEPA, 1993).

Wang et al. (1994) studied PeCB in spiked (4.5 µg/kg) and sewage sludge-amended soil (3 µg/kg) at 20-30 °C. Half of the dosage of PeCB is rapid loss by volatilization, followed by degradation with half-lives of 187 days (spiked soil; 1.4 o.m.) to 1550 days (amended soil, 4.5% o.m.). Formation of bound residues is a relatively minor route of dissipation on soil. Scheunert et al. (1985) recovered 1% of a 2 mg/kg dosage as bound residue after 126 days. Under aerobic conditions PeCB is persistent in soil. Beck and Hansen (1974) found disappearance half-lives based on duplicate samples, of 194 – 345 days in an aerobic loamy sand soil (1.9% o.m.); 18-20°C treated at 7 mg/kg. Standard deviations were 20 to 25%. The 95% confidence limits are thus 112-726 and 289-3176 days. Since the values were based on duplicates, the total range of 112-3176 days represents the experimental results. Soils were kept in 10L buckets covered with two plastic sheets. During the experiment that lasted 600 days, water losses were compensated; apparently the total water content of the soil evaporated from the soils every 100 days (Bro-Rasmussen et al., 1970). The reported disappearance values are based on log(2)/k; instead of ln(2)/k. Correct half lives thus span the range of 260 – 7300 days. The contribution of volatilization of PeCB to these half lives is unknown.

Susarla et al. (1997) investigated the degradation of HCB in a methanogenic slurry of sandy sediment (<1% o.m.) with lake water (1:3 v/v), spiked at 1.14 mg/L. After 75% of the HCB had degraded after 150 days, the degradation of the primary metabolite PeCB followed first order kinetics with a half life of approximately 50 days at 25 °C. Masunaga et al. (1996) investigated the degradation of PeCB in sulfidogenic estuarine sediments that had been pre-exposed to various chemicals from local industries. Sediment slurries contained 272 g/kg solids; of which 12% can be lost by ignition, and were kept at 25°C. PeCB half-life was 18 days. In autoclaved samples the half-life was 990 days.

In sediment cores of Ketelmeer in The Netherlands, that had been selectively enriched with HCB to get a dechlorinating anaerobic community, PeCB is not persistent: the adapted anaerobic microflora gives half-lives of about 6 days at 25 °C when spiked at 50 µg/L (Beurskens et al., 1994). A mixture of clay loam soil (5.38% o.m.) and a sterile medium (50 g soil and 70 ml medium) was incubated anaerobically at room temperature after inoculation with a 10% slurry of an adapted microbial culture. The soil was spiked with 14.2 mg/L HCB, 25 mg/L PeCB, and 254.1 mg/L 1,2,4-TCB. Concentrations PeCB decreased with a half-life of approximately 23 days. Chlorobenzene accumulated as the major metabolite after 80 and 142 days to 1 mmol/L (Ramanand et al., 1993). So far, only one bacterial strain which reductively dechlorinates chlorobenzenes has been isolated (Adrian and Görisch, 2002).

Comparison of PeCB concentrations in Ketelmeer sediment (The Netherlands) sampled and measured in 1972 to concentrations in samples taken in 1988 from sediment layers deposited around 1970, showed a small but statistically significant decline of 35%. HCB had decreased by 80%. Lower chlorinated benzenes like di and tetrachlorinated benzenes had increased up to 80% (Beurskens et al., 1993). Lake Ketelmeer sediment contains 9-13% o.m. (Aarnoutse et al., 1996; Cornelissen and Gustafsson, 2004). In a UK soil (Woburn) that had received 25 separate sewage sludge applications in 20 years time (until 1961), approximately 21% of the added PeCB was still in the soil 30 years after application had stopped (Wang et al., 1995). This soil received about 25% of its dry weight in sludge. Assuming that sludge contained 80% organic matter and a 2% organic matter breakdown per year, the mean o.m. content was 15%. Input of HCB during these years was about 4 times higher than the PeCB input; and HCB residues also declined to 22% in these 30 years.
Experimental data on degradation of PeCB in water are lacking. PeCB is expected to dissipate from the water phase to the sediment or into the air. PeCB is persistent in soils and sediments under aerobic conditions. In anaerobic sediment-water slurries PeCB is considered persistent, except at temperatures above 10°C in combination with low organic matter contents. Higher organic matter contents seem to drastically increase the persistency. Actual field measurements of PeCB may overestimate persistency as a result of formation of PeCB from HCB. The true field half life of PeCB is estimated around 6 years in organic soil and sediment in the temperate zone.

PeCB should be considered as persistent given the magnitude of estimated and experimental half-lives in atmosphere, soils, sediments, and water. Persistence in the environment depends on the rate of photo-oxidation, the presence of oxygen and organic matter.

2.2.2 Bioaccumulation

Pentachlorobenzene is highly hydrophobic. Mackay et al. (2006) report log K\textsubscript{ow} values between 4.88 and 6.12, with recommended values of 5.17-5.18. Therefore, it can be assumed that the compound has a high bioaccumulation potential. This is confirmed by the data shown in Table 2.3. In this table, BCFs are summarized which were evaluated and which were considered reliable according to the Klimisch criteria (Klimisch, 1997).

The results in Table 2.3 show that BCFs range from 1085 - 23000 L/kg for fish; 833 – 4300 L/kg for mollusca, and 577 – 2258 L/kg for crustacea. It should be noted that for the lowest BCF data for fish it is not explicitly clear if exposure concentrations have been measured (Schuler et al., 2007). If these BCFs are based on nominal instead of measured exposure concentrations, then they are probably lower than the ‘real’ BCFs based on measured concentrations.

Table 2.3. BCF values for pentachlorobenzene

<table>
<thead>
<tr>
<th>Species</th>
<th>Exp. time</th>
<th>Exp. concn. [mg/L]</th>
<th>BCF [L/kg\textsubscript{ww}]</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td><strong>Algae</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Chlorella fusca</td>
<td>24h</td>
<td>0.05</td>
<td>4000</td>
<td>Geyer et al., 1984</td>
</tr>
<tr>
<td><strong>Macrophytes</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Myriophyllum spicatum</td>
<td>25d</td>
<td>0.0059</td>
<td>1375</td>
<td>Gobas et al., 1991</td>
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<tr>
<td><strong>Crustacea</strong></td>
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<tr>
<td>Hyalella azteca</td>
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<td>0.0048</td>
<td>1913</td>
<td>Landrum et al., 2004</td>
</tr>
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<td>Hyalella azteca</td>
<td>28d</td>
<td>0.009</td>
<td>1452</td>
<td>Landrum et al., 2004</td>
</tr>
<tr>
<td>Hyalella azteca</td>
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<td>0.02</td>
<td>1874</td>
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</tr>
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<td>Hyalella azteca</td>
<td>28d</td>
<td>0.03</td>
<td>2164</td>
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<tr>
<td>Hyalella azteca</td>
<td>28d</td>
<td>0.05</td>
<td>2258</td>
<td>Landrum et al., 2004</td>
</tr>
<tr>
<td>Hyalella azteca</td>
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<td>0.08</td>
<td>1139</td>
<td>Landrum et al., 2004</td>
</tr>
<tr>
<td>Hyalella azteca</td>
<td>28d</td>
<td>0.105</td>
<td>2143</td>
<td>Landrum et al., 2004</td>
</tr>
<tr>
<td>Hyalella azteca</td>
<td>28d</td>
<td>0.118</td>
<td>871</td>
<td>Landrum et al., 2004</td>
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<tr>
<td>Hyalella azteca</td>
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<td>0.14</td>
<td>666</td>
<td>Landrum et al., 2004</td>
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<td>Hyalella azteca</td>
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<td>0.14</td>
<td>780</td>
<td>Landrum et al., 2004</td>
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<td>Hyalella azteca</td>
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<td>0.2</td>
<td>577</td>
<td>Landrum et al., 2004</td>
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<tr>
<td>Portunus pelagicus</td>
<td>24h</td>
<td>0.025; 0.05</td>
<td>678</td>
<td>Mortimer and Connell, 1993</td>
</tr>
<tr>
<td><strong>Molluscs</strong></td>
<td></td>
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</tr>
<tr>
<td>Lymnaea stagnalis</td>
<td>10d</td>
<td>0.012</td>
<td>833</td>
<td>Legierse et al., 1998</td>
</tr>
<tr>
<td>Lymnaea stagnalis</td>
<td>10d</td>
<td>0.008</td>
<td>1563</td>
<td>Legierse et al., 1998</td>
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<tr>
<td>Mytilus edulis</td>
<td>21d</td>
<td>0.001</td>
<td>4300</td>
<td>Renberg et al., 1985</td>
</tr>
<tr>
<td><strong>Fish</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cyprinus carpio</td>
<td>56d</td>
<td>0.0003</td>
<td>5656</td>
<td>Yakata et al., 2006</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>0.0157; 0.0062;</td>
</tr>
<tr>
<td>Gambusia affinis</td>
<td>96h</td>
<td>0.0033</td>
<td>4000</td>
<td>Chaisuksant et al., 1997</td>
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### Table 1: Bioconcentration Factors (BCF) and Bioconcentration Coefficients (BCF) for Various Species

<table>
<thead>
<tr>
<th>Species</th>
<th>Exp. time</th>
<th>Exp. concn. [mg/L]</th>
<th>BCF [L/kg ww]</th>
<th>Reference</th>
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</thead>
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<tr>
<td><em>Lepomis macrochirus</em></td>
<td>48h</td>
<td></td>
<td>5100</td>
<td>Banerjee et al., 1984</td>
</tr>
<tr>
<td><em>Lepomis macrochirus</em></td>
<td>28d</td>
<td>0.0052</td>
<td>3400</td>
<td>Barrows et al., 1980</td>
</tr>
<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>48h</td>
<td>0.0006</td>
<td>4000</td>
<td>Banerjee et al., 1984</td>
</tr>
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<td><em>Oncorhynchus mykiss</em></td>
<td>48h</td>
<td>0.0076</td>
<td>4300</td>
<td>Banerjee et al., 1984</td>
</tr>
<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>48h</td>
<td>0.0328</td>
<td>6900</td>
<td>Banerjee et al., 1984</td>
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<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>48h</td>
<td>0.0708</td>
<td>5800</td>
<td>Banerjee et al., 1984</td>
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<td><em>Oncorhynchus mykiss</em></td>
<td>48h</td>
<td>0.106</td>
<td>5500</td>
<td>Banerjee et al., 1984</td>
</tr>
<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>119d</td>
<td>3E-07</td>
<td>14000</td>
<td>Oliver and Niimi, 1983</td>
</tr>
<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>105d</td>
<td>9E-06</td>
<td>23000</td>
<td>Oliver and Niimi, 1983</td>
</tr>
<tr>
<td><em>Pimephales promelas</em></td>
<td>31d</td>
<td>0.028</td>
<td>9600</td>
<td>Carlson and Kosian, 1987</td>
</tr>
<tr>
<td><em>Pimephales promelas</em></td>
<td>31d</td>
<td>0.055</td>
<td>6900</td>
<td>Carlson and Kosian, 1987</td>
</tr>
<tr>
<td><em>Pimephales promelas</em></td>
<td>10d</td>
<td>0.1</td>
<td>1582</td>
<td>Schuler et al., 2007</td>
</tr>
<tr>
<td><em>Pimephales promelas</em></td>
<td>10d</td>
<td>0.15</td>
<td>1346</td>
<td>Schuler et al., 2007</td>
</tr>
<tr>
<td><em>Pimephales promelas</em></td>
<td>10d</td>
<td>0.2</td>
<td>1300</td>
<td>Schuler et al., 2007</td>
</tr>
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<td><em>Pimephales promelas</em></td>
<td>10d</td>
<td>0.25</td>
<td>1773</td>
<td>Schuler et al., 2007</td>
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<tr>
<td><em>Pimephales promelas</em></td>
<td>10d</td>
<td>0.3</td>
<td>1085</td>
<td>Schuler et al., 2007</td>
</tr>
<tr>
<td><em>Poecilia reticulata</em></td>
<td>5d</td>
<td>±0.54 μmol/L</td>
<td>4700</td>
<td>Opperhuizen, 1988</td>
</tr>
<tr>
<td><em>Poecilia reticulata</em></td>
<td>48h</td>
<td></td>
<td>7300</td>
<td>Banerjee et al., 1984</td>
</tr>
</tbody>
</table>

In conclusion, these values show that pentachlorobenzene can be considered to have a high bioaccumulation potential. Due to the high logKow and the fact that biotransformation may be insignificant (Schuler et al., 2006, 2007), the compound may also have a biomagnification potential. However, data on the biomagnification of pentachlorobenzene are lacking.

2.2.3 Potential for Long range environmental transport

The potential for long-range environmental transport can be estimated from monitoring data in remote regions, through physical-chemical properties of the molecule which are promoting such transport and/or through model results. The most well known mechanism of long-range transport is atmospheric transport of substances in the vapour phase. A prerequisite for long-range atmospheric transport is its persistence to degradation.

Overall persistence and long-range transport potential were estimated for five new POP candidates (including PeCB) with the OECD Pov & LRTP Screening Tool using the input properties in the POPRC proposal documents (Wegmann et al, 2007). The tool does not provide absolute levels in the environment, but facilitates comparison with earlier identified POP substances. The authors conclude that, although there are considerable uncertainties in the chemical characteristics of the five chemicals investigated, the POP candidates (including PeCB) have Pov and LRTP properties similar to those of several earlier identified POPs.

There is also evidence based on calculations of the transport distance of PeCB through the atmosphere. Mantseva et al. (2004) developed a multi-compartment transport model for the evaluation of long-range atmospheric transport and deposition of POPs. Based on this model assessment a transport distance in Europe of over 8 000 km is calculated for PeCB. The model is described in detail by Vulykh et al. (2005) who assessed a transport distance of 8 256 km. Based on measured concentrations in air samples of North America an empirical estimation of 13 338 km was made for the long range transport of PeCB through air (Shen et al., 2005). This distance is larger than...
that of the other organochlorine pesticides that were part of this study including the currently listed POPs dieldrin, DDT and heptachlor.

Monitoring data also indicate that PeCB is subject to long range transport. PeCB was detected in air and precipitation at various locations in the world, many of those far from its sources.

In a survey of ambient air at Windsor and Walpole Island, Ontario, mean concentrations in samples taken between August 1988 and October 1989 were 0.12 ng/m$^3$ in Windsor (detected in 31 of 32 samples) and 0.07 ng/m$^3$ for Walpole Island (detected in 27 of 30 samples), respectively. Maximum concentrations at the 2 sites were 0.28 and 0.22 ng/m$^3$ at Windsor and Walpole Island, respectively (Government of Canada, 1993). These concentrations are in the same range, notwithstanding the fact that Windsor is located 6 km from a municipal waste incinerator plant in Detroit, Michigan, while Walpole Island is a rural location located 55 km from the same plant.

According to the Integrated Atmospheric Deposition Network (IADN), the average atmospheric concentration of PeCB measured above the North American Great Lakes in 2000 is about 0.072 ng/m$^3$ (ICCA/WCC 2007 citing Buehler et al., 2004). In all air samples collected in 2000-2001 at the 40 sampling stations in North America (including 5 arctic stations), PeCB was detected. The measured concentrations were relatively constant across the continent, averaging 0.045 ng/m$^3$ with a range of 0.017 to 0.136 ng/m$^3$ (Shen et al., 2005). According to the authors, the small spatial variability across the Northern Hemisphere indicates that PeCB has a very long atmospheric residence time, which allows it to become widely distributed in the global hemisphere.

At the European arctic locations Bear Island, Lillestroem and Spitzbergen PeCB concentrations in air collected in the period 1980/1981 ranged from 0.0033 to 0.078 ng/m$^3$ (Vulykh, 2005 citing Oeme and Manø, 1984). At the arctic station Alert (Northwest Territories, Canada) PeCB concentrations in air collected between February and April 1988 ranged from 0.0031 to 0.135 ng/m$^3$ (Government of Canada, 1993).

The presence of PeCB has been reported in several abiotic (air, rainwater, water, sediment and soil) and biotic (fishes, birds, mammals) matrices at remote regions including the arctic region and Antarctica. These are described in detail in the section Exposure.

In conclusion, modeling, monitoring data of PeCB in air, as well as PeCB's chemical properties indicate that this substance has a considerable potential for long range environmental transport. The presence of PeCB in matrices from remote regions, some that can only have received PeCB after transport via air, supports the conclusion that PeCB is subject to long range transport.

### 2.3 Exposure

PeCB is spread widely in the global environment. The first two sections will focus on the levels of PeCB in abiotic and biotic media in remote regions such as the (ant)arctic environment. The third section will focus on monitoring data on PeCB in abiotic and biotic media of temperate zones, as well as observed trends. The last section discusses human exposure.

#### 2.3.1 Levels in abiotic environmental matrices of remote regions

Atmospheric concentrations of PeCB have been measured at various locations around the world. Concentrations in air collected at Alert (Northwest Territories, Canada) ranged from 0.0031 to 0.135 ng/m$^3$ (Government of Canada, 1993). PeCB was also detected in all air samples collected in 2000-2001 at the 40 sampling stations in North America (including 5 arctic stations). The measured concentrations were relatively constant across the continent, averaging 0.045 ng/m$^3$ with a range of 0.017 to 0.136 ng/m$^3$ (Shen et al., 2005). Shen et al., 2005, also observed that atmospheric levels of organochlorine compounds including PeCB increased with increasing elevation in the Canadian Rocky Mountains. This proposed cold-trapping of PeCB is also reflected by increasing concentrations of PeCB in mosses in the Andean mountains and the occurrence of PeCB in the arctic and Antarctic regions in several biotic and abiotic matrices.
A study of the influence of emission sources on atmospheric PeCB concentrations in Germany showed that concentrations were higher at industrial or urban locations (ranging from 0.057 to 0.286 ng/m³) than at a rural reference site (0.031 ng/m³) (ICCA/WCC 2007 citing Wenzel et al., 2006). Concentrations at the rural site are comparable to the average atmospheric concentration measured by the Integrated Atmospheric Deposition Network (IADN) above the North American Great Lakes in 2000, i.e., about 0.072 ng/m³ (ICCA/WCC 2007 citing Buehler et al., 2004).

Samples of rain collected in Canada from 1987 to 1991 on average contained 0.02 ng/L PeCB, with a range of <0.01 to 0.09 ng/L (Government of Canada, 1993 citing Muir, 1993 and Strachan, 1993). A study from the river Ob region in the Russian arctic reported traces of PeCB in snow and ice (Melnikov et al., 2003). This was not further quantified.

PeCB was found in all water samples collected during a study of the distribution of chlorinated organics in the North Pacific Ocean, the Bering and Chukchi streets (ICCA/WCC 2007 citing Strachan et al., 2001). Concentrations of PeCB in the dissolved phase averaged 0.016 ng/L, while suspended solids represented only a small fraction of the total amount of PeCB. Bottom sediment samples taken from harbours in northern Norway and the Kola Peninsula in the arctic contained PeCB in concentrations ranging from 2 to 5 µg/kg dry weight. PeCB concentrations in four Alaskan arctic lakes sampled from 1991 to 1993 averaged 0.10 ±0.10 µg/kg dry weight (ICCA/WCC, 2007 citing Allen-Gil et al., 1997). Concentrations in soil samples from the coastal areas of Victoria Land (Antarctica) varied between 0.4 and 1.3 µg/kg dry weight (Borghini et al., 2005). In these soil samples PeCB was the dominant organic compound. Muir et al. (1995 as cited by ICCA/WCC, 2007) reported PeCB in sediment of a series of remote lakes in northern Canada. Sediment surface layer concentrations (representing a period of time estimated between 1979-1988) of PeCB in these northern lake ranged from less than 0.01 to 0.73 µg/kg sediment.

2.3.2. Levels in biota of remote regions

Contamination of the environment and biota in remote regions can be a threat to vulnerable species and ecosystems. PeCB is detected in mosses, fish, penguin eggs, seals and predatory mammals in the arctic and antarctic regions.

PeCB concentrations in mosses from coastal areas of Victoria Land (Antarctica) varied between 1 and 2.4 µg/kg dry weight (Borghini et al., 2005). The mosses do not have a root system and their supply is largely dependent on atmospheric deposition. Also Antarctic soil has received PeCB through contact with the atmosphere. The measured PeCB concentrations in both mosses and soils were higher than those of the currently listed POPs HCB and DDT that were also included in this study. PeCB concentrations in mosses growing in the Andean Mountains at elevations between 700-4500 m ranged from 0.2 – 2.4 µg/kg dw (Grimalt et al., 2004). This study shows that PeCB is likely subject to cold-trapping. An inverse relationship was established with higher PeCB concentrations at lower temperatures. A similar relationship was established for mountain soils in Tenerife (Ribes et al., 2002).

Concentrations (µg/kg wet weight) of PeCB in fish from four Alaskan lakes averaged at 1.42 ± 1.82 in grayling liver, 0.06 ±0.08 in grayling muscle, 0.48 ±0.35 in lake trout liver and 1.21 ± 3.66 in lake trout muscle (ICCA/WCC, 2007 citing Allen-Gil et al., 1997). Navaga fish from the White Sea in Northwestern Russia contained 5.06 µg/kg wet weight PeCB (ICCA/WCC, 2007 citing Muir et al., 2003). Fish liver samples from near Adak Island (Alaska) contained 0.8 and 1.4 µg/kg of PeCB in cod and 1.4 µg/kg of PeCB in halibut (ICCA/WCC, 2007 citing Arend et al., 2001). Furthermore PeCB has been found in other artic biota, such as krill (0.05 µg/kg ww), emerald rock cod muscle (0.08 µg/kg ww), emerald rock cod whole body (0.09 µg/kg ww) and Greenland arctic char (0.07 µg/kg ww which is equivalent to 3.9 µg/kg lipid weight) (Vorkamp et al., 2004; Corsolini et al., 2006).

In Greenland PeCB was observed at levels of 23 µg/kg lipid weight in ptarmigan liver (1.5 µg/kg wet weight) and 8 µg/kg lipid weight in kittiwake muscle (1.1 µg/kg wet weight) (Vorkamp et al., 2004). Adelie penguin eggs (Antarctic) contained 0.68 µg/kg ww PeCB (Corsolini et al., 2006).

Inuit hunter collected tissue samples of ringed seals from the east and west sides of the Northwater Polyna between Canada and Greenland during the spring of 1998 (ICCA/WCC, 2007 citing Fisk et al.,
The concentration (wet weight) of PeCB in these sampled ranged from 7.3 ±1.9 µg/kg in male ringed seals to 8.4 ±1.1 µg/kg in females from the west side. Seals from the east side (Quebec) contained 5.0 ±0.5 µg/kg (males) and 7.0 ±1.5 µg/kg (females). Seals from the White Sea in Northwestern Russia collected in the period 1992-1998 contained PeCB at concentrations ranging from 0.9 (bearded seal) to 12.0 µg/kg lipid weight (harp seal) in their blubber (ICCA/WCC, 2007 citing Muir et al., 2003). The mean concentration (± standard deviation of the 10 samples) of PeCB in 1992 was 11±2.0 ng/g lipid weight whereas the concentration of PeCB in 1998 was 5.0±1.8 ng/g lipid weight. PeCB concentrations in bowhead whales collected between 1994 and 1998 averaged at 0.3 ± 0.1 and 0.8 ± 0.1 µg/kg wet weight in liver and blubber, respectively (ICCA/WCC, 2007 citing Hoekstra et al., 2002). St. Lawrence Bay (Canada) Beluga Whale blubber was found to contain 24.5 (1.56 – 1510) µg/kg (lipid weight) PeCB for females and 144.5 (1.5 - 1500) µg/kg for males (ICCA/WCC, 2007 citing Hobbs et al., 2003). In Greenland, blubber of musk ox (captured between 1998 and 2001) was reported to contain 0.32 µg/kg lipid weight PeCB (equivalent to 0.29 µg/kg ww) (Vorkamp et al., 2004).

PeCB has also been detected in polar bears. The compound was present in all 15 fat and plasma samples taken from polar bears from the arctic Svalbard islands (Gabrielsen et al., 2004) at an average concentration of 7.9 and a maximum of 13.9 µg/kg (wet weight). Similar concentrations are observed in polar bears from Alaska, Canada and East-Greenland, according to the authors. Concentrations and body burdens of chlorobenzenes (including PeCB) in polar bears of different ages have been studied before and after their seasonal fasts (ICCA/WCC, 2007 citing Polischuk et al., 2002). The authors conclude that no PeCB is metabolized or excreted during the fast, leading to increasing concentrations of the compound in fat tissue. Amounts of PeCB in cubs is reported to be greater than in adults due to the fact that nursing bear cubs receive an increased amount of PeCB.

The accumulation of PeCB has also been measured in the arctic fox during 1999-2001 (ICCA/WCC 2007, citing Hoekstra et al., 2003). The animals were collected some distance from human habitation to minimize effects of garbage scavenging. About 20 animals were collected at each site. PeCB concentrations (µg/kg) found in arctic foxes were: 61 ± 0.12 in muscle (Arivat), 0.29 ± 0.06 in muscle (Holman), 0.57 ± 0.11 in liver (Holman), 0.55 ± 0.20 in muscle (Barrow) and 0.73 ± 0.17 in liver (Barrow).

King et al (2003) studied the chlorobenzenes spilled after an accident in the Gulf of St Lawrence. There was a rapid decline in tri- to peCB concentrations in snow crabs from sampling location 1 [near the spill] between 1996 and 1998. From 1998 to 2000 the chlorobenzenes concentrations in snow crabs persisted at low levels. In 1996, chlorobenzenes concentrations at locations 2 to 11 were much lower than at location 1, but showed no consistent decrease with time.

### 2.3.3. Levels at temperate regions including trends

A large quantity of monitoring data exists on PeCB detected in abiotic matrices as well as in biota in temperate zones, mainly originating from developed countries. In general, concentrations of PeCB in the temperate zones of the world seem to decrease. This pattern is representative for that of most POPs. For the (ant)arctic area, only recent data are available which do not allow to derive a trend.

A clear trend of the presence of PeCB in the environment can be derived from its presence in sediment cores. Sediment cores from the industrially impacted area from Lake Ontario near the mouth of the Niagara River (Canada) show an increase in PeCB concentration from early 1900 until the period 1960-1970 (peak concentration of over 100 µg/kg) after which concentrations declined to about 10% of the peak concentration by 1980 (ICCA/WCC, 2007 citing Durham and Oliver, 1983 and NYDEC, 1998). Also PeCB concentrations in the Niagara river water dropped from 0.351 to 0.093 ng/L during the period 1987-1997 (ICCA/WCC 2007, citing Williams et al., 2000). However, data in the mussel watch programme for the Niagara river do not show a decrease in PeCB concentrations between 1997 and 2000 on several locations (Ministry of the Environment Ontario, 1999, 2003 ). Concentrations of PeCB in sediment of the Ketelmeer in The Netherlands dropped by 37% in the period 1972-1988 (Beurskens et al., 1993). PeCB concentrations in Herring gull eggs from Muggs Island / Leslie spit (Canada) have dropped from 50 µg/kg in 1970 to non-detected at 1 µg/kg in the mid 1990s (ICCA/WCC 2007, citing Bishop et al., 1992; Petit et al., 1994; Pekarik et al., 1998; Jermyn-Gee et al., 2005; Havelka, 2006).
Calambokis et al (1999) studied persistent pollutants in Harbor seals (*Phoca vitulina*) in Puget harbor (US) during the period 1984-1997. They concluded that total TEQ showed a near significant decline by year (*p*=0.07) and that other pesticides also showed general declining trends. Only for HCB, total chlorobenzenes, and chlordane was the decline statistically significant.

Because concentrations of PeCB have decreased, only recent data (last 15 years) are summarized in Table 2.5 for abiotic and Table 2.6 for biota matrices.

**Table 2.5** Concentrations of pentachlorobenzene in abiotic matrices from temperate regions. Data of the last 15 years.

<table>
<thead>
<tr>
<th>Year</th>
<th>Location</th>
<th>Compartment</th>
<th>Concentration</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004</td>
<td>Sweden</td>
<td>Air</td>
<td>0.033 ng/m²</td>
<td>Kaj and Dusan, 2004</td>
</tr>
<tr>
<td>2004</td>
<td>Sweden</td>
<td>Atmospheric deposition</td>
<td>0.16 ng/m² day</td>
<td>Kaj and Dusan, 2004</td>
</tr>
<tr>
<td>1997-1998</td>
<td>Lake Malawi, Africa</td>
<td>Precipitation</td>
<td>0.01 ± 0.014 ng/L</td>
<td>Karlsson et al., 2000*</td>
</tr>
<tr>
<td>2002</td>
<td>Sweden</td>
<td>Sediment, lakes</td>
<td>&lt;6 µg/kg dry weight</td>
<td>Sternbeck et al., 2003</td>
</tr>
<tr>
<td>2002</td>
<td>Sweden</td>
<td>Sediment, C. Stockholm</td>
<td>&lt;4 µg/kg dry weight</td>
<td>Sternbeck et al., 2003</td>
</tr>
<tr>
<td>1996-1997</td>
<td>Niagara river, US</td>
<td>Water</td>
<td>0.093 ng/L</td>
<td>Williams et al., 2000*</td>
</tr>
<tr>
<td>2002</td>
<td>Danube, Central Europe</td>
<td>Sediment</td>
<td>0.0001 – 3.5 mg/kg</td>
<td>Slobodniki, &amp; Dogterom, 2003</td>
</tr>
<tr>
<td>1998</td>
<td>Yangtse river, China</td>
<td>Water; sediment</td>
<td>0.4 ng/l; 3 µg/kg</td>
<td>Jiang et al., 2000*</td>
</tr>
<tr>
<td>?</td>
<td>Elbe river, Germany</td>
<td>Sediment</td>
<td>&lt;1 - 71 µg/kg</td>
<td>Witter et al., 1998*</td>
</tr>
<tr>
<td>1993-1994</td>
<td>Spree river; Havel river, Germany</td>
<td>Sediment</td>
<td>&lt;10 - 17; &lt;10 - 76 µg/kg</td>
<td>Schwarzbauer et al., 2001*</td>
</tr>
<tr>
<td>1999-2001</td>
<td>Lippe river, Germany</td>
<td>Sediment</td>
<td>1 - 6 µg/kg</td>
<td>Kronimus et al., 2004*</td>
</tr>
<tr>
<td>2003</td>
<td>Ebro river, Spain</td>
<td>Sediment</td>
<td>1.17 µg/kg</td>
<td>Lacorte et al., 2006*</td>
</tr>
<tr>
<td>2004</td>
<td>Sweden</td>
<td>Sediment</td>
<td>1 µg/kg</td>
<td>Kaj and Dusan, 2004</td>
</tr>
<tr>
<td>?</td>
<td>Mulde reservoir, Germany</td>
<td>Sediment</td>
<td>0.4 µg/kg</td>
<td>Zoumis et al., 2001*</td>
</tr>
<tr>
<td>1996</td>
<td>Taiwan</td>
<td>Sediment</td>
<td>Up to 15.7 µg/kg</td>
<td>Lee et al., 2000*</td>
</tr>
<tr>
<td>?</td>
<td>Kishon river, Israel</td>
<td>Sediment</td>
<td>0.01 - 0.06 µg/kg</td>
<td>Oren et al., 2006°</td>
</tr>
<tr>
<td>1997</td>
<td>Masan bay, Korea</td>
<td>Sediment</td>
<td>Up to 0.28 µg/kg</td>
<td>Hong et al., 2003*</td>
</tr>
</tbody>
</table>

*Data cited by ICCA/WCC (2007)

? = Year of sampling unknown.

**Table 2.6** Concentrations of pentachlorobenzene in biotic matrices from temperate regions. Data of the last 15 years.

<table>
<thead>
<tr>
<th>Year</th>
<th>Location</th>
<th>Species</th>
<th>Tissue</th>
<th>Concentration (ng/kg)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1996</td>
<td>The Netherlands</td>
<td>Flounder (Platichthys flesus)</td>
<td>Liver</td>
<td>Reference site: 3 (0.64); polluted site: 1100000 (280000), lipid weight (wet weight)</td>
<td>De Boer et al., 2001</td>
</tr>
<tr>
<td>2002</td>
<td>Sweden</td>
<td>Herring (Clupea harengus); Perch (Perca fluviatilis)</td>
<td>Muscle</td>
<td>2.2; 16 (lipid weight)</td>
<td>Kaj and Dusan, 2004</td>
</tr>
</tbody>
</table>
### 2003 The Netherlands

**Eel (Anguilla anguilla); Pike perch (Sander lucioperca)**

**Whole body**

- 1 - 10 (wet weight)

*Van Leeuwen et al., 2004*

### 2006 Ebro river, Spain

**Fish**

**Whole body**

- 1100 (320 - 3310)

*Lacorte et al., 2006*

### 1994 Rhine; Meuse; Ysselmeer, The Netherlands

**Zebra Mussel (Dreissena polymorpha)**

**Whole body**

- 490; 270; 500 (wet weight)

*Hendriks et al., 1998*

*Data cited by ICCA/WCC (2007)*

During a survey within the Danube Regional Project for the European Water Framework Directive, pentachlorobenzene was detected in almost all sediment samples at concentration levels of 0.0001 – 3.5 mg/kg and in most of the suspended solid samples at concentration levels of 0.001 – 0.028 mg/kg (Slobodník and Dogterom, 2003).

The ATSDR database from the US Government contains 41 records of polluted sites with pentachlorobenzene. Maximum concentrations of pentachlorobenzenes at these sites vary between 147 and 5100 mg/kg in sediments and between 0.43 and 2040 mg/kg in soil. Concentrations in fish vary between 0.00019 and 2.4 \( \text{g/kg} \). The data does not mention if these concentrations are based on wet or dry weight basis (ATSDR, 2007).

#### 2.3.4. Human exposure

PeCB has been detected in breast milk and found to accumulate in human placenta (Shen et al., 2007). The mean concentration of PeCB in the breast milk of Canadian women taken 3 to 4 weeks after parturition was < 1 \( \mu \text{g/kg} \) (trace) with a maximum value of 1 \( \mu \text{g/kg} \). In this survey, the compound was detected in 97% of the 210 samples analyzed (detection limit and sampling period unspecified) (Government of Canada, 1993 citing Mes et al., 1986). In the breast milk of women of Canadian indigenous population, "trace" (< 1 \( \mu \text{g/kg} \) ) amounts of PeCB were observed in 17% of the 18 samples (detection limit not specified) (Government of Canada, 1993 citing Davies and Mes, 1987). Two other studies investigating PeCB in human milk reported concentrations in the range of 1 to 5 \( \mu \text{g/kg} \) (WHO-IPCS, 1991).

PeCB has also been measured in abdominal, mammary, and perirenal fat tissue from 27 adult Finnish males and females (Smeds and Saukko, 2001). Workers with occupational exposure to PeCB were found to have higher levels of the substance in blood than control groups (Lunde and Bjorseth, 1977).

#### 2.3.5. Bioavailability

The Environmental Health Criteria on chlorobenzenes (WHO/IPCS, 1991) concluded that limited evidence was available showing that sediment-bound residues of chlorobenzenes are bioavailable to organisms; i.e., aquatic invertebrates can take up residues from sediment, and plants, from soil. Since then, more information on the bioavailability of hydrophobic substances became available.

Bioavailability of chlorobenzenes is inversely proportional to the organic carbon content of the soil or sediment (Government of Canada (2003) citing e.g. van Gestel and Ma, 1988; Hulzebos et al., 1993). It was furthermore stated in the Canadian Follow-up Report that persistent substances can remain bioavailable for long periods of time, thereby increasing the probability and duration of potential exposure relative to compounds that do not persist in the environment.
It is generally accepted that not all fractions of organic pollutants bound on sediments or soils are equally toxic due to their various resistances to desorption. The resistant and sequestered fractions of pentachlorobenzene are environmentally less harmful than the more readily desorbing, labile, or available fractions. The large fraction of water soluble organic matter in the sediments is potentially highly mobile and could be easily resuspended or leached to the overlying water column. If the soluble organic matter carries the major amount of pentachlorobenzenes as expected, continuous contamination of the water body from the sediments is very likely. Qiao & Farrell (1996) carried out experiments with PeCB in rainbow trout and concluded that mass balance analysis suggests that the appearance of HCBP and PeCB in the fish after 6 days could not be accounted for solely by the amount of chemical dissolved in the water at the time when the fish were introduced. The chemical uptake in fish with the pharynx plugged, to eliminate the gut uptake route, was similar to that in control fish. Because direct access to bottom sediments did not alter chemical uptake, they concluded that hydrophobic chemicals such as PeCB and HCBP associated with suspended sediments from the Fraser River can readily desorb and be taken up across the gill. Åkerblom (2007) concluded that pesticide sorption to organic particles in standardized toxicity tests is fast and efficient and that substances bound to the sediment may act as a reservoir, continuously supplying the pore water with low pesticide concentrations.

As sediment or organic matter bound organic pollutants may still become available, an evaluation should focus on sorption and desorption kinetics of pentachlorobenzene and modifying circumstances rather than on statements on bioavailability. Such data are however scarce.

### 2.4 Hazard assessment for endpoints of concern

#### 2.4.1. Toxicity

**Toxicokinetics**

Toxicokinetic studies with rats show that after an oral dose, the substance is distributed to the blood and tissues (Umegaki et al., 1993; WCC, 2007 citing Thomas and coauthors). Linder et al., (1980) observed that rats fed with PeCB accumulated approximately 1.5 – 2.2 times the dietary concentration in their adipose tissues. Umegaki et al., (1993) studied the kinetics of PeCB in blood and tissues of rats given a single oral dose by gavage of either 15 mg or 20 mg. PeCB was observed in the blood, liver, kidney, brain, and fat tissue as well as in the feces (4.8% of the dose). In the blood, also the major metabolite pentachlorophenol was observed.

Using the blood concentrations after a single gavage of 15 mg PeCB (results of the study of Umegaki et al., 1993), WCC (2007) calculated that after exposure to repeated oral daily doses of 15 mg (or 77 mg/kg) the steady-state blood concentration of PeCB would be 2.6 μg/ml in rats. Assuming linear kinetics in this dose range, WCC (2007) extrapolated this value from the 77 mg/kg-day exposure to the lowest observed adverse effect level (LOAEL) for PeCB of 8.3 mg/kg-day (value derived from the study of Linder et al., 1980, see section Subchronic Toxicity). According to the calculations of WCC, this resulted in a predicted steady-state blood concentration of PeCB of 0.28 μg/ml. Since PeCB partitions predominantly in the lipid portion of blood and adipose tissue, the concentration of PeCB in blood lipid fraction would be predicted to be approximately 46 μg/g lipid.

Den Besten et al (1994) studied the urinary metabolite profile of PeCB in the rat after dietary exposure for 13 weeks. PeCB was metabolized to the major metabolites pentachlorophenol (PCP), 2,3,4,5-tetrachlorophenol (TCP), mercaptotetrachloro-phenol (MTCP), the glucuronide derivative of pentachlorothiophenol (PCTP), and the minor metabolites tetrachlorohydroquinone (TCHQ), methylthiotetrachlorophenol (MeTTP), hydroxytetrachlorophenyl sulphoxide (HTCPS), and bis(methylthio)-trichlorophenol (bis-MeTRiCP). The study also revealed that oxidation of PeCB to 2,3,4,5-TCP was not mediated by cytochrome P450IIIα. In the urine of rabbits exposed to a single oral dose of PeCB, also pentachlorophenol and 2,3,4,5-tetrachlorophenol was observed (Slooff et al., 1991, citing Kohli et al., 1976).

A study with coyotes showed that PeCB is secreted in the faeces (Johnston et al., 1997). Coyotes were dosed with PeCB (single dose of 130, 260 or 520 mg). In both studied matrices, faeces and adipose tissue, residues of PeCB were determined. PeCB was detectable in faeces for six months post-dosing. In the faeces, also the metabolites pentachlorophenol and 2,3,4,5-tetrachlorophenol were detected.
Acute toxicity
Pentachlorobenzene has been tested on rats and mice. Results of acute toxicity tests are available for oral and dermal exposure, see Table 2.7.

LD\textsubscript{50}s for PeCB (by gavage in peanut oil) are 940 to 1125 mg/kg bw in adult and weanling rats and 1175 and 1370 mg/kg bw in Swiss Webster mice (Linder et al., 1980 cited in Government of Canada, 1993). Decreased activity and tremors were observed in both species at sublethal doses; the kidneys, liver and adrenal glands of rats were also enlarged. In some rats, the gastric mucosa was hyperaemic, and a slight reddish fluorescence of the gastrointestinal tract was observed in both rats and mice under ultraviolet light, suggesting porphyria (Government of Canada, 1993). In the study of Allen et al., (1979, cited in Slooff, 1991), a LD\textsubscript{50} of 250 mg/kg bw was observed in rats.

Ariyoshi et al., (1975, cited in Slooff, 1991) observed an increase of cytochrome P450 content in rats as well as an increase in the activity of two hepatic enzymes after oral administration of 250 mg/kg bw once daily during 3 days.

To determine a dermal LD\textsubscript{50} one concentration (i.e., 2500 mg/kg bw) was tested on rats, but no toxic effects were seen at this dose (Linder et al., 1980 cited in Slooff, 1991). Based on this study, a NOEC of > 2500 mg/kg bw can be established for dermal exposure.

(Sub)chronic toxicity
In female Sherman rats ingesting diets containing 500 ppm (mg/kg) and greater (> 37.5 mg/kg bw/day) PeCB for 100 days, there was an increase in liver weight and hypertrophy of hepatic cells (Linder et al., 1980). There was also an increase in kidney weights and renal hyaline droplet formation in males at exposure levels \( \geq 125 \) ppm (mg/kg) (equivalent to \( \geq 8.3 \) mg/kg bw/day). In addition, at 1 000 ppm (equivalent to 81.1 mg/kg bw/day for males and 78.7 mg/kg bw/day for females), the effects observed were: an increase in adrenal weight and focal areas of renal tubular atrophy and interstitial lymphocytic infiltration in males; an increase in kidney weight in females; a decrease in haemoglobin and an increase in white blood cells in both sexes; and decreases in red blood cells and haematocrit in males. The no-observed-effect-level (NOEL) in female rats, derived on the basis of the results of this study, was 250 ppm (equivalent to 18.2 mg/kg bw/day); the lowest-observed-effect-level (LOEL) in males was 125 ppm (equivalent to 8.3 mg/kg bw/day) (calculations by Government of Canada, 1993).

In a study of NTP (1991) rats and mice were exposed to PeCB through their diet. Observed effects were among others: decreases in the mean body weights of male rats at exposure levels \( \geq 1 \) 000 ppm (mg/kg diet) and in females at all concentrations (\( \geq 33 \) ppm), increase in absolute and relative liver weights (33 ppm in males), centrilobular hepatocellular hypertrophy (as low as 330 ppm for males), increases in kidney weights and renal histopathological effects at concentrations as low as 100 ppm, nephrotoxic effects in females (\( \geq 1 \) 000 ppm), increase of the concentration of protein in the urine in male and female rats at \( \geq 1 \) 000 ppm, decrease of free thyroxin and total thyroxin concentrations in male and female rats indicating moderate hypothyroxinemia and abnormalities were observed at concentrations of \( \geq 330 \) ppm in females and \( \geq 1 \) 000 ppm in males. The incidence of abnormal sperm in males was also increased at both dietary concentrations at which it was examined (330 and 2 000 ppm). On the basis of histopathological lesions, the authors considered the NOELs to be 33 ppm in male rats and 330 ppm in females (approximately 2.4 and 24 mg/kg bw/day, respectively) (calculations by Government of Canada, 1993).

In PeCB exposed mice in the same study NTP (1991), observed effects were among others: ventral swelling and ruffled fur (2 000 ppm [mg/kg]), increase of kidney weights (\( \geq 330 \) ppm in males), functional effects on the thyroid at all concentrations in both sexes (\( \geq 33 \) ppm), increase in liver weights (at 100 ppm in males). The only exposure-related histological lesion in mice of either sex was centrilobular hepatocellular hypertrophy and minimal necrosis, observed at all concentrations in males and at \( \geq 330 \) ppm (equivalent to 68 mg/kg bw/day) in females. On the basis of the histopathological lesions, the authors considered the NOEL in female mice to be 100 ppm (approximately 22 mg/kg bw/day). No NOEL for males could be established (LOEL = 33 ppm or approximately 5.2 mg/kg bw/day) (calculations by Government of Canada, 1993).
Mutagenicity and carcinogenicity

Epidemiological studies of exposed populations are not available and information on carcinogenicity in experimental animals has not been identified. PeCB showed no genotoxicity in a small number of in vitro and in vivo studies of a limited range of investigated genetic endpoints.

PeCB has been tested negative in the Ames test (Table 2.7). Based on limited available data, mutagenicity in S. typhimurium with and without metabolic activation, effects on chromosomes in Chinese Hamster ovary cells in vitro, and micronuclei in peripheral blood smears in animals from the NTP sub-chronic study, PeCB has been assessed as not genotoxic (Haworth et al., 1983 and NTP, 1991 cited in Government of Canada, 1993).

Several studies (Thomas et al., 1998 and Gustafson et al., 2000; Ying et al., 2001) investigated the tumor-promoting activity in medium term carcinogenicity assays of various chlorobenzene isomers including PeCB. The results suggest that PeCB promotes glutathione S-transferase (GSTP1-1) positive preneoplastic foci formation in rat liver, following diethylnitrosamine (DEN) initiation.

Pentachlorobenzene has been classified, therefore, in Group V (inadequate data for evaluation) of the classification scheme for carcinogenicity developed for use in the derivation of the Guidelines for Canadian Drinking Water Quality (Environmental Health Directorate, 1989 cited in Government of Canada, 1993).

The only risk phrase for pentachlorobenzene in the European ESIS database is R22, harmful if swallowed (European Chemicals Bureau, 2007). According to HSDB (2003) pentachlorobenzene is not classifiable as to human carcinogenicity because there are no human data and no animal data available. WHO-ICPS (1991) concludes: ‘Available data are inadequate for the assessment of the carcinogenicity of the higher chlorinated benzenes (tri- to penta-).’ On mutagenity WHO-ICPS (1991) concludes that although the available data from in vitro and in vivo assays for isomers other than 1,4-DCB are limited, chlorobenzenes do not appear to be mutagenic.

Reproductive and developmental toxicity

Available studies concerning the embryotoxicity, foetotoxicity and teratogenicity of PeCB include one study in rats (Villeneuve and Khera, 1975, cited in Government of Canada, 1993) and one in mice (Courtney et al., 1977, cited in Government of Canada, 1993). Results of the study of Villeneuve and Khera (1975, cited in Government of Canada, 1993) indicated that PeCB is foetotoxic (an increased incidence of extra ribs and sternal defects was observed in the offspring) at maternal exposure doses of 50 mg/kg bw/day,. The exposure concentration was below the concentration that induced toxic effects in the mothers. In mice, no embryotoxic, foetotoxic or teratogenic effects were observed in the offspring at doses which were maternally toxic (50 mg/kg bw/day and above)(Courtney et al., 1977, cited in Government of Canada, 1993). In the only identified study on reproductive toxicity of PeCB, Linder et al. (1980) reported that suckling pups of PeCB treated mothers fed ≥ 250 ppm developed tremors (LOAEL = 18.2 mg/kg/day). At 1000 ppm, most sucklings died before weaning.
Table 2.7: Toxicity of PeCB

<table>
<thead>
<tr>
<th>Test result</th>
<th>Test details</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Acute oral toxicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouse, Swiss Webster</td>
<td>LD50 = 1,175 - 1,370 mg/kg bw</td>
<td>Decreased activity and tremors; a slight reddish fluorescence of the gastrointestinal tract was observed under ultraviolet light, suggesting porphyria Linder et al 1980, cited in CEPA (1993)</td>
</tr>
<tr>
<td>Rat, adult and weanling</td>
<td>LD50 = 940 - 1,125 mg/kg bw</td>
<td>Decreased activity and tremors; kidneys, liver and adrenal glands enlarged. In some rats, hyperaemic gastric mucosa and indications for porphyria Linder et al 1980, cited in Government of Canada (1993)</td>
</tr>
<tr>
<td>Rat</td>
<td>3 days 250 mg/kg bw/day</td>
<td>Content of Cyt P-450 and enzymatic activities in liver increased Ariyoshi et al 1975 cited in Slooff (1991).</td>
</tr>
<tr>
<td><strong>Acute dermal toxicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>(sub)Chronic oral toxicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rat, Sherman female</td>
<td>NOEL = 250 mg/kg diet Equiv. to a NOEC of 18.2 mg/kg bw 100 days 0 - 1000 ppm</td>
<td>Increase in liver weights and hypertrophy of hepatic cells Linder et al. 1980 cited in Government of Canada 1993</td>
</tr>
<tr>
<td>Rat, male</td>
<td>LOEL = 125 mg/kg diet Equiv. to a NOEC 8.3 mg/kg bw 100 days 0 - 1000 ppm</td>
<td>Increase in liver and kidney weights, hypertrophy of hepatic cells and renal hyaline droplet formation Linder et al. 1980 cited in Government of Canada 1993</td>
</tr>
<tr>
<td>Rat F344/N, male</td>
<td>NOEL = 33 mg/kg 91 days 0-2000</td>
<td>Histopathological lesions NTP 1991</td>
</tr>
<tr>
<td>Test result</td>
<td>Test details</td>
<td>Reference</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td><strong>Exposure</strong></td>
<td><strong>Observation</strong></td>
<td><strong>Dose</strong></td>
</tr>
<tr>
<td>period</td>
<td>period</td>
<td></td>
</tr>
<tr>
<td>diet</td>
<td>Equiv. to a NOEL</td>
<td>ppm</td>
</tr>
<tr>
<td>2.4 mg/kg bw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rat F344/N, female</td>
<td>NOEL = 330 mg/kg diet</td>
<td>91 days</td>
</tr>
<tr>
<td>Equiv. to a NOEL 22 mg/kg bw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouse B6C3F1, female</td>
<td>NOEL = 100 mg/kg diet</td>
<td>91 days</td>
</tr>
<tr>
<td>Equiv. to a NOEL 22 mg/kg bw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mouse, male</td>
<td>LOEL = 33 mg/kg diet</td>
<td>91 days</td>
</tr>
<tr>
<td>Equiv. to a LOEL 5.2 mg/kg bw</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Genetic toxicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Developmental toxicity – oral</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rat</td>
<td>NOAEL (developmental) = 6.3 mg/kg bw (Maternal dose)</td>
<td>180 days</td>
</tr>
<tr>
<td>LOAEL (developmental) = 18.2 mg/kg bw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOAEL (maternal toxicity) = 37.5 mg/kg bw/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test result</td>
<td>Test details</td>
<td>Reference</td>
</tr>
<tr>
<td>-------------</td>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>Mouse</td>
<td>NOEL (developmental) = 50 mg/kg bw/day Maternal toxicity ≥ 50 mg/kg bw/day</td>
<td>No effects observed</td>
</tr>
<tr>
<td>Rat</td>
<td>LOAL = 50 mg/kg bw/day Maternal toxicity &gt; 50 mg/kg bw/day</td>
<td>increased incidence of extra ribs and sternal defects in the offspring</td>
</tr>
</tbody>
</table>
Human studies
Occupational exposure to PeCB may be through inhalation and dermal contact with this compound at workplaces where PeCB is produced or used. Examples are wood treatment plants, dielectric fluid spill and cleanup, municipal solid waste incinerators, hazardous waste incinerators, and magnesium production plants. Exposure may also arise in occupational settings where the pesticide quintozene is produced and used. The general population may be exposed to PeCB via inhalation of ambient air, ingestion of food and drinking water. Case reports of adverse effects in individuals, or epidemiological studies of populations exposed to PeCB have not been identified (Government of Canada, 1993).

2.4.2. Ecotoxicity

Aquatic toxicity
Acute and chronic toxicity data are available for both freshwater (Table 2.8) and marine organisms (Table 2.9).

The lowest acute toxicity values are 100 µg/L for freshwater fish species (EC50) and 87 µg/L for a marine crustacean (LC50). The lowest chronic values (NOECs) are 2 µg/L for a freshwater fish and 14 µg/L for a marine crustacean. According to these findings, species sensitive to PeCB can be found in both the freshwater and the marine environment.

Within the European Union PeCB is classified as a substance which is very toxic to aquatic organisms and which may cause long-term adverse effects in the aquatic environment (Risk phrases N; R50 and R53) (European Chemicals Bureau, 2007). This classification is based on the fact that the substance is very toxic to fish, daphnia or algae (LC50 ≤1 mg/L) and the substance is not readily degradable or bioaccumulative.

Soil and sediment toxicity
Limited data are available for soil and sediment. Tests with various chlorobenzenes were carried out by Van Gestel et al (1991). Two earthworm species were raised on a natural sandy soil (KOBG) and an artificial OECD standard soil. Average LC50 values varied between 115 and 238 mg/kg dry weight, whereas LC50 values in pore water varied between 55.1- 117.7 µg/L. Van Gestel et al (1991) concluded that based on pore water concentrations earthworms are more sensitive to PeCB than fish, but that this may be due to differences in test design.

Only one study on the toxicity of pentachlorobenzene in plants was identified. Duplicate tests were carried out in which *Lactuca sativa* seedlings were grown on OECD soil contaminated with pentachlorobenzene. The seedlings were harvested after 7 and 14 days. EC50 values varied between 56 and 862 mg/kg dw (Hulzebos et al. 1993). Experiments in solution resulted in an EC50 value of ±1.0 mg/L. Details of the tests are provided in Table 2.10.

Toxicity to birds
No toxicity data on birds are available for PeCB.
Table 2.8. Freshwater species: selection of acute and chronic aquatic toxicity data.

<table>
<thead>
<tr>
<th>Species</th>
<th>Exp. time</th>
<th>Criterion</th>
<th>Test endpoint</th>
<th>Value (mg/L)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Algae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Ankistrodesmus falcatus (acicularis)</em></td>
<td>4 h</td>
<td>Primary production ($^{14}$C uptake)</td>
<td>EC50</td>
<td>1.25&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Wong et al., 1984 in Hesse et al., 1991</td>
</tr>
<tr>
<td><strong>Crustacea</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>48 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>0.300, 1.25&lt;sup&gt;a&lt;/sup&gt;, 5.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Abernethy et al., 1986; Bobra et al., 1983; Leblanc, 1980 in Hesse et al., 1991</td>
</tr>
<tr>
<td><em>Ceriodaphnia dubia</em></td>
<td>7 d</td>
<td>Reproduction</td>
<td>IC50</td>
<td>0.520&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Oris et al., 1991 in Priority Substance No. 26. Pentachlorobenzene. Substance Data Sheet., 2005</td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>16 - 21 d</td>
<td>Reproduction</td>
<td>NOEC</td>
<td>0.010, 0.031, 0.100</td>
<td>Hermens et al., 1984, De Wolf et al., 1988, Van Leeuwen 1987 in Hesse et al., 1991</td>
</tr>
<tr>
<td><strong>Insects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Chironomus thummi</em></td>
<td>48 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>0.230</td>
<td>Roghair et al., 1994 in Priority Substance No. 26. Pentachlorobenzene. Substance Data Sheet., 2005</td>
</tr>
<tr>
<td><em>Chironomus tentans</em></td>
<td>2 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>168 (10 °C)</td>
<td>Lydy et al., 1999</td>
</tr>
<tr>
<td><em>Chironomus tentans</em></td>
<td>2 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>150 (20 °C)</td>
<td>Lydy et al., 1999</td>
</tr>
<tr>
<td><em>Chironomus tentans</em></td>
<td>2 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>137 (30 °C)</td>
<td>Lydy et al., 1999</td>
</tr>
<tr>
<td><strong>Fish</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Oncorhynchus mykiss</em></td>
<td>48 h</td>
<td>Behaviour</td>
<td>EC50</td>
<td>0.100</td>
<td>Ahamd et al., 1984 in Priority Substance No. 26. Pentachlorobenzene. Substance Data Sheet., 2005</td>
</tr>
<tr>
<td><em>Lepomis macrochirus</em></td>
<td>96 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>0.250</td>
<td>Buccafusco et al., 1981 in Hesse et al., 1991</td>
</tr>
<tr>
<td><em>Gambusia affinis</em></td>
<td>96 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>3.2</td>
<td>Chaisuksant et al., 1998</td>
</tr>
<tr>
<td><em>Gambusia affinis</em></td>
<td>42 d</td>
<td>Growth rate</td>
<td>EC50</td>
<td>0.15</td>
<td>Chaisuksant et al., 1998</td>
</tr>
<tr>
<td><em>Gambusia affinis</em></td>
<td>42 d</td>
<td>Growth rate</td>
<td>EC10</td>
<td>0.002</td>
<td>Chaisuksant et al., 1998</td>
</tr>
</tbody>
</table>
a Value above water solubility (0.56 mg/L at 25 °C).
Table 2.9. Marine species: selection of acute and chronic aquatic toxicity data.

<table>
<thead>
<tr>
<th>Species</th>
<th>Exp. time</th>
<th>Criterion</th>
<th>Test endpoint</th>
<th>Value (mg/L)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Algae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Crustacea</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portunus pelagicus</td>
<td>96 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>0.087</td>
<td>Mortimer and Connell, 1995</td>
</tr>
<tr>
<td>Portunus pelagicus</td>
<td>40 d</td>
<td>Growth</td>
<td>EC50</td>
<td>0.041</td>
<td>Mortimer and Connell, 1995</td>
</tr>
<tr>
<td>Portunus pelagicus</td>
<td>40 d</td>
<td>Growth</td>
<td>EC10</td>
<td>0.014</td>
<td>Mortimer and Connell, 1995</td>
</tr>
<tr>
<td><strong>Fish</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyprinodon variegatus</td>
<td>96 h</td>
<td>Mortality</td>
<td>LC50</td>
<td>0.8&lt;sup&gt;a&lt;/sup&gt;, 0.46</td>
<td>Heitmuller, 1981 in Hesse et al., 1991; Mayer, 1987 in Van der Plassche et al., 1993</td>
</tr>
</tbody>
</table>

Table 2.10. Soil species: selection of acute and chronic aquatic toxicity data.

<table>
<thead>
<tr>
<th>Species</th>
<th>Exp. time</th>
<th>Criterion</th>
<th>Test endpoint</th>
<th>Value (mg/kg dw)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Macrophyta</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactuca sativa</td>
<td>7 d</td>
<td>Growth</td>
<td>EC50</td>
<td>228</td>
<td>Hulzebos et al., 1993</td>
</tr>
<tr>
<td>Lactuca sativa</td>
<td>7 d</td>
<td>Growth</td>
<td>EC50</td>
<td>862</td>
<td>Hulzebos et al., 1993</td>
</tr>
<tr>
<td>Lactuca sativa</td>
<td>14 d</td>
<td>Growth</td>
<td>EC50</td>
<td>56</td>
<td>Hulzebos et al., 1993</td>
</tr>
<tr>
<td>Lactuca sativa</td>
<td>14 d</td>
<td>Growth</td>
<td>EC50</td>
<td>±320</td>
<td>Hulzebos et al., 1993</td>
</tr>
<tr>
<td><strong>Annelida</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eisenia Andrei</td>
<td>14 d</td>
<td>Mortality</td>
<td>LC50</td>
<td>134</td>
<td>Van Gestel et al, 1991</td>
</tr>
<tr>
<td>Eisenia Andrei</td>
<td>14 d</td>
<td>Mortality</td>
<td>LC50</td>
<td>238</td>
<td>Van Gestel et al, 1991</td>
</tr>
<tr>
<td>Lumbricus rubellus</td>
<td>14 d</td>
<td>Mortality</td>
<td>LC50</td>
<td>201</td>
<td>Van Gestel et al, 1991</td>
</tr>
</tbody>
</table>

In conclusion, based on the fact that the lowest acute aquatic toxicity value (LC50) of PeCB is < 1 mg/L and the lowest NOEC is 10 µg/L, PeCB is considered very toxic to aquatic organisms.

**Multiple chemicals and toxicological interactions**

Annex E request information on toxicological interactions involving multiple chemicals (Annex E, b). Limited information is available on this subject. Yoo et al (2003) report on their studies on the kinetics of PeCB: “The kinetics and toxicity of pentachlorobenzene were assessed using a freshwater (*Hyalella azteca*) and marine amphipod (*Leptocheirus plumulosus*). The
results of these studies demonstrated the additive toxicity of PeCB with other organic chemicals (pyrene).

Comparison of exposure and effect data
ICCA/WCC (2007) followed several approaches for relating exposure to adverse effects of pentachlorobenzene, one of which is presented here. Environment Canada calculated an estimated no effect value (ENEVsed) for freshwater benthic organisms exposed to PeCB of 25 µg/g (25,000 ng/g) organic carbon (Environment Canada, 2003 cited by ICCA/WCC, 2007). This PeCB ng/g-OC concentration was exceeded in 1994 in Canada only at a location near a single site of industrial contamination, which has since undergone remediation activities. Rural and remote sites which have been studied for long range transport have PeCB ng/g-OC concentrations typically more than three orders of magnitude less than, or 1/1000, of the ENEVsed. For example, Muir et al. (1995) report sediment surface layer concentrations of PeCB in northern Canada lake sediments of less than 0.01 to 0.73 ng/g sediment between 1979 and 1988 (ICCA/WCC, 2007). The other two approaches also showed considerable difference between exposure and effect concentrations.

Several methods, exposure routes and species with very different feeding strategies were used by ICCA/WCC to determine the lethal and critical body burden of PeCB. Based on the estimations a Lethal Body Burden of 1 to 2.5 mmol/kg (250 to 626 mg PeCB/kg) would be expected. Based on the general knowledge on substances with a narcotic mode of action and the available data on PeCB, such as the Hyalella growth/mortality study and other information discussed, an estimation of 0.1 mmol PeCB/kg (25 mg/kg) was tentatively proposed by ICCA/WCC as a Critical Body Burden for chronic effects.

Hoydal and Dam (2003) measured concentrations of <0.1 – 37 ng/g wet weight in biota captured in the environment of the Faroe Islands. The highest amount is a factor of 5000-20000 lower than the critical tissue residues of 165-861 µg/g reported by ICCA/WCC (2007). The highest concentrations reported in chapter 2.3.3 are a factor of 50 lower than these critical tissue residue concentrations. We may conclude that in most cases environmental concentrations do not reach the estimated effect concentrations.

A very recent publication of Schuler et al (2007b) have reported critical whole body residues of pentachlorobenzene of 58 µg/g and 5 µg/g for Hyalella azteca and Chironomus tentans respectively. In contrast to most other studies chronic toxicity data was used to establish these critical whole body residues instead of acute toxicity data. These critical whole body residues values are 2-25 times higher than the highest concentrations reported in chapter 2.3.3 and 150-1500 times higher than the highest value reported for the Faroe Islands.

3 SYNTHESIS OF THE INFORMATION

Pentachlorobenzene is a chlorinated organic compound. According to available data, pentachlorobenzene should be considered as persistent given the considerable number of estimated and experimental half-lives in atmosphere, soils, sediments, and water. Persistence in the environment depends on the rate of photo-oxidation, the presence of oxygen and organic matter. Pentachlorobenzene meets the criterion on bioaccumulation. BCF values for pentachlorobenzene range from 1085 – 23 000 L/kg for fish, 833 – 4 300 L/kg for mollusca, and 577 – 2258 L/kg for crustacean. Biomagnification may be expected due to the high logKow and the fact that biotransformation is insignificant. However, data on the biomagnification of pentachlorobenzene are lacking.

The available data support the potential for long range transport of pentachlorobenzene. The physical-chemical characteristics are within the range of the other POPs. Model estimations on the transport distance resulted in distances of 8 000, while estimates based on air measurements suggested 13 338 km. Monitoring data also indicate that PeCB is subject to long range transport. PeCB was detected in air and precipitation at various locations in the world, many of those far from its sources. The small spatial variability across the Northern Hemisphere observed in some studies also indicate that PeCB has a very long atmospheric residence time, which allows it to become widely distributed in the global hemisphere.
A large quantity of monitoring data exists on PeCB detected in abiotic matrices as well as in biota in temperate zones, mainly originating from developed countries. In general, concentrations of PeCB in the temperate zones of the world seem to decrease. This pattern is representative for most POPs. For the (ant)arctic area, only recent data are available which do not allow to derive a trend.

Case reports of adverse effects in individuals, or epidemiological studies of populations exposed to PeCB have not been identified. The only risk phrase for pentachlorobenzene in the European ESIS database is R22, harmful if swallowed. Lowest LD50 observed for acute exposure was 250 mg/kg bw. According to the American Hazardous Substances Data Bank pentachlorobenzene is not classifiable as to human carcinogenicity because there are no human data and no animal data available. PeCB is moderately toxic to humans. Pentachlorobenzene is very toxic to aquatic organisms and may cause long-term adverse effects in the aquatic environment. Data on soil and sediment organisms are limited or lacking.

Bioavailability of pentachlorobenzene is inversely proportional to the organic carbon content of the soil or sediment. However, experiments suggest that hydrophobic chemicals bound to the sediment or suspended sediment may act as a reservoir and result in continuous uptake. There are limited quantitative data on this process for pentachlorobenzene.

Generally, the levels that are found in environmental compartments, including biota, are well below known and established critical effect levels based on acute toxicity data. Recently established critical body residues based on chronic toxicity data are 2-25 times higher than the highest concentrations reported in chapter 2.3.3 and 150-1500 times higher than the highest value reported for the Faroe Islands.

The data from Europe and North America show that production and use of pentachlorobenzene has ceased over the last decades, but it cannot be excluded that PeCB is produced or used elsewhere. Unintentional release of pentachlorobenzene as a byproduct of incomplete combustion appears to be the most important source. However, this conclusion is based on data for Europe and North America only.

PeCB meets all screening criteria on long range transport, persistence, bioaccumulation and toxicity. Data on biomagnification and bioavailability do not permit a conclusive statement on these topics as data are lacking, not quantitative or controversial. Environmental concentrations in remote areas are well below the established critical effect levels, but approach these effect levels at highly polluted sites. Generally, environmental concentrations seem to decrease. Production and use have ceased in Europe and North America, but data from other parts of the world are limited. Unintentional release as a byproduct of incomplete combustion appears to be the most important source of pentachlorobenzene in the environment.

4 CONCLUDING STATEMENT

It has been demonstrated that pentachlorobenzene meets all the criteria laid down in Annex D of the Stockholm Convention. Its physical-chemical characteristics are within the range of the other POPs in the Convention.

The substance is persistent in the environment and is bioaccumulative. The small spatial variability across the Northern Hemisphere indicates that pentachlorobenzene has a very long atmospheric residence time, which allows it to become widely distributed in the global hemisphere. There are monitoring data from remote areas, backed up by modelling results that suggest that pentachlorobenzene can be transported over great distances. Pentachlorobenzene is moderately toxic to humans, but is very toxic to aquatic organisms. Present concentrations in remote areas are well below estimated critical body burdens.
Because of its long range transport, neither a single country nor a group of countries alone can abate the pollution caused by this substance. Unintentional release of pentachlorobenzene as a byproduct of incomplete combustion appears to be the most important source. Measures to reduce these releases can only be taken at a global scale. Although the production and use of pentachlorobenzene seems to have ceased in most countries, its reintroduction remains possible. This could lead to increased releases and levels in the environment. Based on the available evidence, it is thus likely that pentachlorobenzene can, as a result of long range environmental transport, cause significant adverse effects on human health and/or the environment, such that global action is warranted.


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B.9. Pentabromodiphenyl ether – SUMMARY

Summary
9. PentaBDE

Draft Risk Management Evaluation May 2007
http://www.pops.int/documents/meetings/poprc/drprofile/drme/DraftRME_PeBDE.pdf

Risk Profile UNEP/POPS/POPRC.2/17/Add1

| Composition | Commercial pentabromodiphenyl ether (C-PentaBDE) refers to mixtures of bromodiphenyl ether congeners in which the main components are 2,2', 4,4'-tetrabromodiphenyl ether (BDE-47 CAS No. 40088-47-9) and 2,2',4,4',5-pentabromodiphenyl ether (BDE-99 CAS No. 32534-81-9), which have the highest concentration by weight with respect to the other components of the mixture. |
| Uses | Commercial pentabromodiphenyl ether mixtures (C-PentaBDE) are used for flame retardant purposes as additives in consumer products. The commercial mixtures contain brominated diphenyl ether congeners with three to seven bromines in the molecule, but molecules with four and five bromines predominate. The proportion of the different polybromodiphenyl ether (PBDE) congeners in C-PentaBDE varies in different regions of the world. The main source in North America and Western Europe has been the C-PentaBDE incorporated in polyurethane foam, used in domestic and public furniture. This use is now mainly phased out. The information is too limited to draw conclusions on the importance of other uses, like textiles, electrical and electronic products, building materials, vehicles, trains and aeroplanes, packaging, drilling oil fluid and rubber products. While some representative examples are covered, detailed information on use is lacking for many regions of the world. Emission sources include production filter waste, foam production, release from products, wastes, landfills, incineration, and waste recycling. |
| Fate | Due to its high persistency in air, the main route for long-range transport of PentaBDE - as with so many substances that are sufficiently volatile, persistent and bioaccumulative - is through the atmosphere. Modelling and environmental studies indicate that the transport is through a series of deposition/volatilization hops towards the poles but particulate transport is known to be important, too. Long-range transport through water and emigrating animals is also likely. Several studies show that PentaBDE in soil and sediments is bioavailable, enters the food chain and that it bioaccumulates and biomagnifies in the food webs, ending up in high levels in top predators. |
| Effects | Toxicological studies have demonstrated reproductive toxicity, neurodevelopmental toxicity and effects on thyroid hormones in aquatic organisms and in mammals. The potential for the toxic effects in wildlife, including mammals, is evident. A Canadian assessment of risk quotients suggests that the highest risks accrue to species high in the food chain. Information is lacking on the effects in humans of short-term and long-term exposure, although it is to be expected that vulnerable groups can be pregnant women, embryos and infants. |
| Exposure | PentaBDE is widespread in the global environment. Levels of components |
of C-PentaBDE have been found in humans in all UN regions. Most trend analyses show a rapid increase in concentrations of PentaBDE in the environment and in humans from the early 1970s to the middle or end of the 1990s, reaching plateau levels in some regions in the late 1990s, but continuing to increase in others. The levels in North America and the Arctic are still rising. Vulnerable ecosystems and species are affected, among them several endangered species. Some individuals of endangered species show levels high enough to be of concern. Potential exposure to humans is through food, and through use of products and contact with indoor air and dust. PentaBDE transfers from mothers to embryos and lactating infants. Considerably higher levels are found in humans from North America in general. About 5% of general populations have been found to be subjected to elevated exposure. This, together with the estimates of the long half-life of PentaBDE congeners in humans, raises concern for long-term effects on human health.

| Status       | An OSPAR Commission background document on PBDEs was reviewed by Sweden in 2001. The next full review of this document is not planned before 2008. At the 4th North Sea Conference, it was decided to phase out the use of brominated flame retardants by 2020. C-PentaBDE was nominated as a new POP to the UNECE Convention on Long-range Transboundary Air Pollution in 2004 by Norway. In December 2005 it was considered by the Executive Body of the Convention to meet the screening criteria for POPs, set out in EB decision 1998/2. They requested that the UNECE Task Force on POPs continue with the review and further explore management strategies. The EU notified PentaBDE to the Rotterdam Convention in 2003. For it to become a candidate, bans of the substance must be notified by two parties under the Convention. |

| Alternatives | There are three ways to provide flame retardancy in products without using BFRs: 1) substitute them with another flame retardant in a given material (i.e. plastic or foam); 2) substitute them with another flame retardant in a different type of plastic or foam; or 3) redesign the product so that there is no need for using flame retardants. Some manufacturers have already replaced C-PeBDE with cost competitive non-POPs alternatives in all uses, including flexible polyurethane and electronics. |
Pentabromodiphenyl ether
Introduction

Commercial pentabromodiphenyl ether is a highly viscous liquid mixture of tri-, tetra- and pentabromodiphenyl ethers, hexabromodiphenyl ethers and heptabromodiphenyl ethers. The major components of pentabromodiphenyl ether products are 2,2',4,4'–tetrabromodiphenyl ether (BDE-47) and 2,2',4,4',5–pentabromodiphenyl ether (BDE-99). Commercial pentabromodiphenyl ether is used mainly in rigid and flexible polyurethane foams and polyurethane elastomers. Most of this polyurethane is used in turn in upholstery and furnishing. Global market demand for pentabromodiphenyl ether has more than doubled in the last decade to the present $8.5 \times 10^6$ kg per year. Simultaneously, use in Europe has decreased to approximately $2.1 \times 10^5$ kg per year.

All Nordic countries have committed themselves to stop using this flame retardant. The European Commission has already made a proposal for banning the use and placing on the market of pentabromodiphenyl ether or products and articles treated with it. Alternative chemicals and techniques for avoiding the use of pentabromodiphenyl ether are available for most of its uses.

Data Source


1. Identification of the chemical

1.1 Names and registry numbers

CAS chemical name:

Commercial product is a mixture. Major components are BDE-99 (2,2',4,4',5–pentabromodiphenyl ether) and BDE-47 (2,2',4,4'–tetrabromodiphenyl ether)

Synonyms/abbreviations:

Pentabromodiphenyl ether (PeBDPE and PentaBDPE),
Benzene, 1,1'-oxybis-, pentabromo derivative,
Pentabromophenoxybenzene,
Pentabromodi(s)phenyl ether; biphenyl ether, pentabromo derivative = PeBBE,
Pentabromodi(s)phenyl oxide = PeBBO,
Pentabromodiphenyl oxide = PeBDPO = PentaBDPO

Trade names:
Bromkal 70, Bromkal 70 DE, Bromkal 70 5DE, Bromkal G1, Great Lakes DE 71, Great Lakes DE-60 F (85% PeBDE), FR 1205/1215, Pentabromprop, Saytex 115, Tardex 50.

**CAS registry number:**
The commercial mixture, while sold as a technical grade under the Chemical Abstracts Service (CAS) Registry number for the penta isomer, is more accurately identified by the CAS Registry numbers of the individual components::
(a) Pentabromodiphenyl ether (CAS No. 32534-81-9) 50–62% w/w;
(b) Tetrabromodiphenyl ether (CAS No. 40088-47-9) 24–38% w/w;
(c) Tribromodiphenyl ether (CAS No. 49690-94-0) 0–1% w/w;
(d) Hexabromodiphenyl ether (CAS No. 36483-60-0) 4–12% w/w;
(e) Heptabromodiphenyl ether (CAS No. 68928-80-3) trace.

1.2 Structure

![Structure of 2,2',4,4',5-pentabromodiphenyl ether (BDE-99)](image)

2,2',4,4',5-pentabromodiphenyl ether (BDE-99)

Molecular formula: $\text{C}_{12}\text{H}_{5}\text{Br}_5\text{O}$
Molecular weight: 564.7

2. Persistence

According to a standard Organisation for Economic Cooperation and Development test with aerobic activated sludge, pentabromodiphenyl ether is not readily biodegradable. No experimental studies have been reported on its abiotic degradation. Some photolysis resulting in reductive debromination may occur and be a possible pathway for abiotic degradation. Abiotic and biotic degradation of pentabromodiphenyl ether in sediment, water and soil have not been reported in experimental studies but the half-lives for BDE-99 and BDE-47 have been estimated at 600 days (aerobic sediment) and 150 days (water and soil) for both congeners.

3. Bioaccumulation

Commercial pentabromodiphenyl ether and all its components have logKow values greater than 5. All the components of commercial pentabromodiphenyl ether bioconcentrated in carp (*Cyprinus carpio*). The bioconcentration factor for commercial pentabromodiphenyl ether in carp was estimated to be ca. 27,400. BDE-99 and BDE-47 are taken up efficiently in pike (*Esox lucius*) and to similar or higher levels than many PCBs. The bioaccumulation potentials of BDE-47 and BDE-99 in blue mussels (*Mytilus edulis*) have been shown to be one order of
magnitude higher than the bioconcentration potentials for several PCBs. BDE-47, BDE-99 and commercial pentabromodiphenyl ether are taken up efficiently and excreted slowly by rats and mice.

Concentrations of the major pentabromodiphenyl ether congeners increase in successive trophic levels. Tetrabrominated and pentabrominated diphenyl ethers show the highest biomagnification potential of all polybrominated diphenyl ethers studied. Increasing levels of pentabromodiphenyl ether congeners have been reported in high trophic level biota from around the world.

4. Potential for long-range environmental transport

Commercial pentabromodiphenyl ether components have very low volatility (vapour pressures between $9.6 \times 10^{-8}$–$4.7 \times 10^{-5}$ Pa) and water solubility (between 2 and 13 µg/l). The estimated Henry’s Law constants suggest that the less brominated components can be volatilized in significant amounts from aqueous solutions. Vapour pressure and water solubility decrease with increasing bromination. According to the atmospheric half-life estimates from structure-activity relationship (SAR) modelling, pentabromodiphenyl ether has long-range transport potential in the atmosphere (10–20 days for BDE-99; 11 days for BDE-47). Both BDE-47 and BDE-99 have been found in the Arctic air in Canada and Sweden.

Data from remote areas are still scarce but indicate clearly increasing contamination by pentabromodiphenyl ether. Concentrations of the two major congeners in whales have been reported in the range of ca. 66 to 864 ng/g lipid (BDE-47) and 24 to 169 ng/g lipid (BDE-99).

5. Adverse effects

Rat studies indicate that the liver is the main target organ affected by pentabromodiphenyl ether. Other in vivo studies have found developmental neurotoxicity and behavioural effects in young mice. Immunotoxic effects have been reported in mice but not in rats. Several pentabromodiphenyl ether congeners appear to be anti-oestrogenic.

BDE-47 was shown to be acutely toxic for the copepod *Acartia tonsa* in a standard 48-hour test and caused disturbances in larval development at much lower levels. The EC$_{50}$ in a five-day study was 13 µg/l.

6. Statement of the reasons for concern

According to the available data, pentabromodiphenyl ether resists abiotic and biotic degradation and thus persists in the environment for long times. It has a great potential for
bioaccumulation and in addition there is monitoring evidence of its biomagnification. Due to its physical and chemical properties and considerably long atmospheric half-life it can be assumed that pentabromodiphenyl ether can be transported long distances in air. There is a solid data base on the toxic and ecotoxic properties of pentabromodiphenyl ether showing that it or its metabolites cause, inter alia, adverse developmental effects in offspring, liver effects, growth disturbance, dioxin-like effects and endocrine disruption depending on the target organism studied.

These data on the harmful properties of pentabromodiphenyl ether are supported by data from the environment. The available data from remote areas show clearly contamination of biota and air by pentabromodiphenyl ether. A few observations of temporally increasing contamination are also available from remote areas. It must be underscored that biological effects in remote area marine mammals cannot be excluded. An upward trend has been observed also in the general human population in blood and milk.

Pentabromodiphenyl ether is widely used as a flame retardant in various articles throughout the world, for the most part in connection with polyurethane applications. The releases of pentabromodiphenyl ether are coming mainly from diffuse sources. The ability of pentabromodiphenyl ether to move in the atmosphere far from its sources further widens the area contaminated by it. No single country, nor group of countries, alone can abate the pollution caused by the production, use and releases of pentabromodiphenyl ether. Therefore regional and global actions are needed to eliminate this pollution.”
RISK PROFILE

Executive summary

A substantial range of studies on PeBDE has been identified. New findings support the conclusion that PeBDE’s properties fulfil the screening criteria in Annex E of the Stockholm Convention. Due to combination of known toxic effects and widespread exposure, it poses significant risks to human health and the environment.

PeBDE is released into the environment during the manufacture of the commercial PeBDE mixture, the manufacture of products, during their use and after they have been discarded as waste. The main source in North America and Western Europe has been products with polyurethane foam, but is now mainly phased out. The information is too limited to draw conclusions on the importance of other uses, like textiles, electric and electronic products, drilling oil fluid and rubber products. Dismantling and reuse of electric and electronic consumer goods can be an extensive source for releases of PeBDE. In addition detailed information on use is lacking for many regions of the world.

The releases are to air, water and soil. The major part of the releases ends up in soil. The distribution between the environmental compartments is: soil>>>water>air. The main part of PeBDE in the environment is bound to particles; only a small amount is transported in its gaseous phase or diluted in water but such transport over long periods can be effective in distributing the PeBDE widely in the environment, especially into Arctic regions.

Due to their high persistency in air, the main route for long-range transport of PeBDE, as with so many volatile, persistent and bioaccumulative substances, is through the atmosphere. Modelling and environmental studies indicate that the transport is through a series of deposition/volatilisation hops towards the poles. Long-range transport through water and emigrating animals is also likely. Several studies show that PeBDE in soil and sediments is bioavailable, enters the food chain and that it bioaccumulates and biomagnifies in the food webs, ending up in high levels in top predators.

PeBDE is widespread in the global environment and in humans. Vulnerable ecosystems and species are affected, among them several endangered species. Some individuals of endangered species show high levels of concern. The potential for the toxic effects in wild life and mammals is evident.

The exposure to humans is through food, use of products and indoor air and dust. PeBDE transfers from mothers to embryos and lactating infants. The detected levels are considerably lower than observed NOELs in laboratory mammals. But knowledge is too scarce to conclude on the effects of long-term exposure. Vulnerable groups can be pregnant women, embryos and infants.

Most countries have ceased their production and uses of PeBDE have been phased-out in several countries, but the substances are still on the market in big regions of the world.

Data sources

This risk profile is elaborated using Annex E information submitted by countries and non-governmental organizations, national reports from web sites for EPAs in different countries, contact and submissions from Norwegian research institutes, the bromine industry, EMEP and AMAP. Eleven countries have submitted information (Australia, Brazil, Canada, Japan, Norway, Mexico, Poland, Republic of Lebanon, Spain, Switzerland and United States of America). Of them four countries did not submit information on production and use (Canada, Mexico, Spain and Switzerland). One did not submit information on use (United States of America). Only one country submitted information on releases. One country reported that they did not have release data. All except one country provided monitoring data. There was no information on stock-piles from submitting countries and only a few have submitted information on trade.
Two observers submitted information - World Wide Fund for Nature (WWF) and the International POPs Elimination Network (IPEN).

INFORMATION RELEVANT TO THE RISK PROFILE

1. SOURCES

1.1. Production and use

Based on the last information on total market demand of PeBDE presented at the Bromine Science and Environmental Forum (BSEF), the total market demand has decreased from 8,500 tons in 1999 to 7,500 tons in 2001. The estimated cumulative use of PeBDE since 1970 was 100,000 t (BSEF, 2001).

Table 1.1. PBDE volume estimates: Total market demand by region in 2001 in metric tons (and by percent) (BSEF, 2001).

<table>
<thead>
<tr>
<th></th>
<th>America</th>
<th>Europe</th>
<th>Asia</th>
<th>Rest of the world</th>
<th>Total</th>
<th>% of total world usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penta-mix PBDE formulation</td>
<td>7,100</td>
<td>150</td>
<td>150</td>
<td>100</td>
<td>7,500</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>53,900</td>
<td>117,950</td>
<td>117,950</td>
<td>2,430</td>
<td>203,790</td>
<td></td>
</tr>
</tbody>
</table>

PeBDE has been produced in Israel, Japan, U.S. and EU (Peltola et al. 2001 and van der Goon et al. 2005). Since 2001 actions to regulate or voluntarily phase-out PeBDE have been conducted in several countries.

There is today no production in Japan and the use of PeBDE was voluntarily withdrawn from the Japanese market in 1990 (Kajiwara et al. 2004). Some developing countries around the East China Sea are potentially “hot spots” releasing PBDE into the marine environment (Ueno et al. 2004). Many industrial manufacturers of computers, televisions and other electric household equipment are situated in the coastal areas of Asian developing countries (Ueno et al. 2004). There are indications on a phase-out of PeBDE in manufacture of new electric and electronic products in the Asian region. The extent of this is uncertain. Waste electric products used in developed countries have been exported to Asian developing countries, such as China, India and Pakistan. This waste material has been recycled for valuable metals (Ueno et al. 2004) and can therefore still be a source to PeBDE releases. No restrictions have so far been implemented in developing countries in the Asia Pacific and the southern hemisphere. In China a commercial product of PeBDE exists that is different with another ratio of its constituents than the mixture used in Europe and North America.

Production in EU ceased in the former EU (15) in 1997 (EU 2000). Usage in the EU (15) has been declining during the second half of the 1990s and is estimated to be 300 metric tonnes in 2000 (used solely for polyurethane production) (EU 2000). The use of PeBDE was banned in the EU (25) in 2004.

In the U.S. the producers voluntarily ended their production of PeBDE in 2004. In 2001 alone, almost 70,000 metric tons of polybrominated diphenyl ethers (PBDEs) were produced globally, almost half of which was used in products sold in the U.S. and Canada. Before the phase-out in U.S. the majority of PeBDE formulation produced globally was used in North America (>97 %). At the end of 2004 in U.S., approximately 7.5% of the more than 2.1 billion pounds of flexible polyurethane foam produced each year in the U.S. contained the commercial PeBDE formulation (Washington State 2005).

In 2004, Australia, through the National Industrial Chemicals Notification and Assessment Scheme (NICNAS), was advised that all importers were phasing out imports of PeBDE by the end of 2005, and this was reconfirmed by the major importers in mid 2005.

The most common use (95-98%) of PeBDE has been in polyurethane foam since 1999. This foam contains between 10 and 18% of the commercial PeBDE formulation. This PUR is in turn used mainly as polyurethane foam for furniture and upholstery in domestic furnishing,
automotive and aviation industry. Other uses are in rigid polyurethane elastomers in instrument casings, in epoxy resins and phenol resins in electric and electronic appliances, and construction materials. PeBDE can also be incorporated in minor amounts in textiles, paints, lacquers, in rubber goods (conveyer belt, coating and floor panels) and in oil drilling fluids. Levels range from 5-30% by weight. Up to the early 1990s PeBDE was used in printed circuit boards, which was usual for FR2 laminates (phenol resins) in Asia. FR2 laminates is used in household electronics (television, radio, video), vehicle electronics, white goods (washing machines, kitchen appliances, for example). In the beginning of 1990s the amount PeBDE used in textile treatment was 60 % of total use in EU. This application of PeBDE is now banned in EU. According to information obtained from the bromine industry the use of PeBDPE as hydraulic fluid (in the form of a mixture) in petroleum borings and mining was discontinued 10-20 years ago. Australia has reported uses in manufacture of polyurethane foams for refrigerators and packaging, and in epoxy resin formulations supplied into aerospace market for use as potting agents, laminating systems and adhesive systems. US have reported use of PeBDE in the air craft industry. There is no use of PeBDE in newer aircraft, and thus exposure of the public, but PeBDE is still used in military aircraft.

1.2 Releases to the environment

Brominated flame retardants are released into the environment during the manufacture of PeBDE, the manufacture of products, during their use and after they have been discarded as waste. A manufacturing process that does not cause emissions is not sufficient. It is also necessary to avoid emissions from products during use as well as after they have been discarded. Most of the PeBDE is released as diffuse pollution during and after the service life of articles incorporating PeBDE and as small-scale point source pollution from the waste management chain of the end products.

PeBDE is synthesised from diphenyl ether by brominating it with elemental bromine in the presence of a powdered iron Friedel-Craft catalyst. The producers of PeBDE have reported that the major routes of PeBDE from this process to the environment are filter waste and rejected material, both of which are disposed of in landfills. Waste water releases of PeBDE may also occur from spent scrubber solutions (Peltola et al. 2001).

According to the EU risk assessment of PeBDE, the emissions in polyurethane production are assumed to occur prior to the foaming process, when handling the additives (discharges to water) and during the curing (emissions to air).

Approximately 3.9 % of the PeBDE present in articles was estimated to be released through volatilisation during their assumed service life of 10 years in the EU risk assessment. PeBDE is used solely as an additive chemical. Thus it can volatilise from the products during their whole life-cycle.

[Note: There is inadequate treatment in this risk profile of the house dust exposure route. It is mentioned, but many studies have been published and these should be discussed. “Volatilise” usually implies going from liquid to gas phase and escaping from the PUR matrix. This does not take into account another viable pathway -- PeBDE adsorbed onto PUR particulates that are released during the life cycle (use and disposal) of the flame retarded article. The next paragraph hints at this approach but still refers to volatilisation during those erosion and grinding processes. See several paragraphs in 2.2.3.1 especially ter Schure analysis for additional support of this concept of particulate transport versus volatilisation.] The considerations raised above were received too late for incorporation in this draft. Attention will be given to them in a subsequent draft.

There are also releases from products due to weathering, wearing, leaching and volatilisation at the end of their service life during disposal or recycling operations (dismantling, grinding or other handling of waste, transport, storage, etc.). The annual releases in the EU region from the product life-cycle of polyurethane products were estimated to be distributed among the different compartments as follows: 75 % to soil, 1 % to air and 24 % to surface water.
Releases from landfills by leaching, and emissions from incineration are considered negligible. In a Dutch project, the emissions of PeBDE in the EMEP region were estimated and distribution between sources was as follows: 0.33 tonnes/year from industrial combustion and processes, 9.45 tonnes/year from solvent and product use and 0.05 tonnes/year from waste incineration (van der Gon et al. 2005).

2. ENVIRONMENTAL FATE

2.1 Persistence

[Note: This section refers to the half life of PeBDE but there is no mention of any data on the product of this "degradation" at the half life, such as lower brominated homolog PBDE formation] The considerations raised above were received too late for incorporation in this draft. Attention will be given to them in a subsequent draft. Estimated half-life values of PDBE in different environmental compartments are scarce in the literature. In table 2.1 half-life estimates found in literature are summarized.

<table>
<thead>
<tr>
<th>Environmental compartment</th>
<th>Half-life estimate (d)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil</td>
<td>150</td>
<td>Palm 2001, Palm et al. 2002</td>
</tr>
<tr>
<td>Aerobic sediment</td>
<td>600</td>
<td>Palm 2001, Palm et al. 2002</td>
</tr>
<tr>
<td>Water</td>
<td>150</td>
<td>Palm 2001, Palm et al. 2002</td>
</tr>
<tr>
<td>Air</td>
<td>19</td>
<td>Palm et al. 2002 Vulykh et al. 2004</td>
</tr>
</tbody>
</table>

*(Note: Caution should be used in relying on half-life estimates derived from this program, now called EPI Suite (http://www.epa.gov/opptintr/exposure/docs/episuite.htm). EPI Suite's intended use is chemical screening only and may not be be appropriate for consideration of substances for global control. The most recent literature should be checked for actual measured half-lives and results found reported (or an indication that information is not available). The analysis should include data on degradation products and their fate – that is, need empirical information; speculation (e.g., potential toxic equivalent (TEQ) with dibenzofuran/dibenzodioxin coplanar analogs or other concerns) may play a role but should be clearly identified.) The considerations raised above were received too late for incorporation in this draft. Attention will be given to them in a subsequent draft.

Several studies using sediment cores show that PeBDE congeners deposited in European marine sediments in the beginning of 1970s still are present in significant amounts, indicating high persistency in sediments (Covaci et al. 2002a, Nylund et al. 1992, Zegers et al. 2000, Zegers et al. 2003). The industrial production and use in Europe started in the beginning of the 1970s, with a reduction in more recent years. This is reflected in the sediment core profiles, with no occurrence before this date, and an increase in levels after, with a levelling off in more recent years.

2.2 Bioaccumulation

2.2.1 Studies on bioaccumulation and biomagnification in local food webs

Several studies have focused on PeBDE's potential for bioaccumulation and biomagnification. The studies show an increase of concentrations in biota with increasing trophic level in pelagic and Arctic food webs. The calculated bioconcentration factors (BCFs), bioaccumulation factors (BAFs) and biomagnification factors (BMFs) indicate PeBDEs potential for bioaccumulation and biomagnification. In Table 2.2 calculated values in the literature are summarized. The octanol-water partition coefficient for PeBDE in those studies is 6.5 – 7.4. The more recent studies are described in the following text.

<table>
<thead>
<tr>
<th>Environmental compartment</th>
<th>Calculated bioconcentration factors (BCFs); bioaccumulation factors (BAFs) and biomagnification factors (BMFs) for PeBDE (BDE-99) in the literature from environmental studies in pelagic and Arctic food webs. The data are calculated using the mean lipid weight</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>------------</td>
</tr>
</tbody>
</table>

Table 2.2 Calculated bioconcentration factors (BCFs); bioaccumulation factors (BAFs) and biomagnification factors (BMFs) for PeBDE (BDE-99) in the literature from environmental studies in pelagic and Arctic food webs. The data are calculated using the mean lipid weight.
concentrations. In the study performed by Sørmo et al. 2006 the values in brackets are BMFs calculated from mean whole body concentrations.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Organism</th>
<th>Area</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCF</td>
<td>Cyprinus carpio</td>
<td>Japan</td>
<td>17 700</td>
<td>CITI, 2000</td>
</tr>
<tr>
<td>BAF</td>
<td>Dreissena polymorpha</td>
<td>Lake Mälaren, Sweden</td>
<td>1.8</td>
<td>Lithner et al. 2003</td>
</tr>
<tr>
<td>BMF</td>
<td>Guillemot egg/herring</td>
<td>Baltic sea</td>
<td>17</td>
<td>Sellström 1996</td>
</tr>
<tr>
<td></td>
<td>Grey seal/herring</td>
<td>Baltic sea</td>
<td>4.3</td>
<td>Sellström 1996</td>
</tr>
<tr>
<td></td>
<td>Atlantic Salmon/Salmon/sprat</td>
<td>Baltic sea</td>
<td>5.9</td>
<td>Burreau et al. 2000</td>
</tr>
<tr>
<td></td>
<td>Herring</td>
<td>The Northern Atlantic Sea</td>
<td>3.8</td>
<td>Burreau et al. 2000</td>
</tr>
<tr>
<td></td>
<td>Net plankton/Benthic organisms</td>
<td>Lake Ontario, Canada</td>
<td>0.8</td>
<td>Burreau et al. 2000</td>
</tr>
<tr>
<td></td>
<td>Benthic organisms/Forage fish</td>
<td>Lake Ontario, Canada</td>
<td>0.65</td>
<td>Alaee et al. 2002</td>
</tr>
<tr>
<td></td>
<td>T. libellula/Copepods</td>
<td>Svalbard, Arctic</td>
<td>(19.0)</td>
<td>Alaee et al. 2002</td>
</tr>
<tr>
<td></td>
<td>G. wilkitzkii/Copepods</td>
<td>Norway</td>
<td>2.1 (1.6)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar cod/Copepods</td>
<td>Svalbard, Arctic</td>
<td>1.9 (1.2)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar cod/T. inermis</td>
<td>Norway</td>
<td>3.4 (1.3)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar cod/T. libellula</td>
<td>Svalbard, Arctic</td>
<td>0.04</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar cod/G. wilkitzkii</td>
<td>Norway</td>
<td>(0.1)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Ringed seal/T. inermis</td>
<td>Svalbard, Arctic</td>
<td>26.8</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Ringed seal/T. libellula</td>
<td>Norway</td>
<td>(54.5)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Ringed seal/G. wilkitzkii</td>
<td>Svalbard, Arctic</td>
<td>43.1</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Ringed seal/Polar cod</td>
<td>Norway</td>
<td>(60.0)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td>0.6 (3.9)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Norway</td>
<td>13.7</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td>(56.6)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Norway</td>
<td>0.3</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td>(0.29)</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Norway</td>
<td>3.4</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td>11</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Norway</td>
<td>8.0</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td>1.0</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Norway</td>
<td>5.9</td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Arctic Canada</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Arctic Canada</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Arctic Canada</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Greenland</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Arctic</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
<tr>
<td></td>
<td>Polar bear/Ringed seal</td>
<td>Svalbard, Arctic</td>
<td></td>
<td>Sørmo et al. 2006</td>
</tr>
</tbody>
</table>

BDE analyses of zebra mussels (Dreissena polymorpha) were included in a larger study undertaken in and around the city of Stockholm, Sweden (Lithner et al., 2003). Mussels were collected from a background site and transplanted in baskets to other downstream sites in Lake Mälaren, Saltsjön and in several small lakes. Freshwater flows from Lake Mälaren, through the middle of Stockholm, then out into the brackish Baltic Sea via Saltsjön. Five BDE congeners (BDE-47, BDE-99, BDE-100, BDE-153 and BDE-154) were determined. The congener pattern was dominated by BDE-47 and BDE-99 and was similar to the penta-mix PBDE technical product. Bioaccumulation factors (BAFs) for the various compounds studied were estimated using data from suspended particulate matter (SPM) collected in sediment traps in 1998-99 at the same sites in Riddarfjärden and Saltsjön (Broman et al., 2001). The concentrations on SPM were assumed to reflect water concentrations. BAFs were calculated using lipid weight concentrations in mussels and organic carbon based concentrations in the SPM.
When compared to other compounds (PCBs, DDTs, HCB), the BDEs had the highest BAFs, ranging from 1 to 2. The BAF (=level in mussel/level in SPM) for PeBDE was 1.8.

Concentrations of BDE-47 and BDE-99 in Lake Ontario pelagic food web show increasing concentrations with increasing trophic position (Alaee et al. 2002). In this study concentrations of PBDEs in archived plankton, *Mysis*, *Diporeia*, alewife, smelt, sculpin and lake trout samples collected in 1993 were determined; and trophodynamics of PBDEs in the Lake Ontario pelagic food web was investigated. Lake Ontario pelagic food web consists of three trophic levels. Lake trout (*Salvelinus namaycush*) are a top predator fish species in Lake Ontario, which feed on forage fish including alewife (*Alosa pseudoharengus*), rainbow smelt (*Osmerus mordax*) and slimy sculpin (*Cottus cognatus*); in turn these fish feed on *Mysis* and *Diporeia*, which feed on phytoplankton, and zooplankton sampled as net plankton. Concentrations were increasing at each step up the food chain. The exception to this trend was the biomagnification of BDE-99 from benthic organisms to forage fish, which had a biomagnification factor of 0.8. This is an indication of the breakdown of BDE-99. In fact, the PBDE profile in the plankton; *Mysis* and *Diporeia* resembled the PeBDE formulation, which indicates that BDE-99 bioaccumulates in the invertebrates and starts to be metabolized by forage fish.

A recent study of an arctic food chain shows the same result (Sørmo et al. 2006) as Alaee’s study. Concentrations of polybrominated diphenylethers (PBDEs) were investigated in an Arctic marine food chain, consisting of four invertebrate species, polar cod (*Boreogadus saida*), ringed seals (*Pusa hispida*) and polar bears (*Ursus maritimus*). The most abundant PBDEs, BDE-47 and BDE-99 were found in detectable concentrations even in zooplankton, the lowest trophic level examined in this study. Most of the investigated PBDEs biomagnified as a function of trophic level in the food chain. A noticeable exception occurred at the highest trophic level, the polar bear, in which only BDE-153 was found to increase from its main prey, the ringed seal, indicating that polar bears appear to be able to metabolize and biodegrade most PBDEs. The authors suggested that this discrepancy in the fate of PBDEs among the different species may be related to greater induction of oxidative detoxification activities in the polar bear. Absorption and debromination rates may be more important for bioaccumulation rates of PBDEs in zooplankton, polar cod and ringed seals. BDE-99 showed no biomagnification from pelagic zooplankton to polar cod, probably as a consequence of intestinal or tissue debromination of BDE-99 in the fish. Also among pelagic zooplankton, there was no increase in concentrations from calanoid copepods to *T. libellula*. Lipid-weight based concentrations (LWCs) and whole-body based concentrations (WBCs) of PBDEs were used to assess biomagnification factors (BMFs). Whole body concentrations gave the most realistic BMFs, as BMFs derived from LWCs seem to be confounded by the large variability in lipid content of tissues from the investigated species. This study demonstrates that PeBDEs have reached measurable concentrations even in the lower trophic levels (invertebrates and fish) in the Arctic and biomagnifies in the polar bear food chain.

Polybrominated diphenyl ethers (PBDEs) were determined in adipose tissue of adult and sub adult female polar bears sampled between 1999 and 2002 from sub-populations in Arctic Canada, eastern Greenland, and Svalbard, and in males and females collected from 1994 to 2002 in north-western Alaska (Muir et al. 2006). Only four congeners (BDE-47, BDE-99, BDE-100, and BDE-153) were consistently identified in all samples. BDE-47 was the major PBDE congener representing from 65% to 82% of the ΣPBDEs. Age was not a significant covariate for individual PBDEs or ΣPBDE. Higher proportions of BDE-99, BDE-100, and BDE-153 were generally found in samples from the Canadian Arctic than from Svalbard or the Bering-Chukchi Sea area of Alaska. All four major PBDE congeners were found to biomagnify from ringed seals to polar bears. The polar bear-seal BMFs were relatively consistent despite the large distances among sites. The exceptions were the BMFs for BDE-99, BDE-100, and BDE-153 in East Greenland which were lower than those at all other sites. This may imply differences in the transformation of PBDEs in the marine food web leading to polar bears or to food web differences. Species differences in bioaccumulation and biotransformation of PBDEs have been noted for fishes and this could lead to differences in congener patterns in fish-eating mammals and their predators.
Studies of the biomagnification of tri- to deca-BDEs were carried out in three different food chains, two in the Baltic Sea and one in the Atlantic Ocean (Law 2005). All the tri- to hepta-BDE congeners biomagnified, but showed a maximum biomagnification for the PeBDEs.

2.2.2 Monitoring results indicating bioaccumulation

A large range of studies show concentrations of concern in top predators. High levels in top predators are usually an indication on the potential of a compound to bioaccumulate in the top predator food chain.

Several studies (Jaspers et al. 2004, Herzke et al. 2005, Lindberg et al. 2004, D’Silva et al. 2004, Law et al. 2005, Sinkkonen et al. 2004, Sellström et al. 2003) indicate that PeBDE is widespread in top predatory birds in Europe, such as peregrine falcon (Falco peregrine), merlin (Falco columbarius), goshawk (Accipiter gentilis), golden eagle (Aquila chrysaetos), and buzzard (Buteo buteo). High levels are detected in top predatory eggs of white-tailed sea eagle, peregrine falcon, osprey, and golden eagle (Herzke et al. 2005, Lindberg et al. 2004). High levels have also been detected in European harbour porpoises (Phocoena phocoena) (Thron et al. 2004 and Covaci et al. 2002).

In the Arctic, PeBDE is detected in high levels in top predatory birds and mammals (Verbal et al. 2005, Verbal et al. 2004, Nordstrom et al. 2002, Herschel et al. 2003, Onramp et al. 2004a and b, Wolkers et al. 2004, Thron et al. 2004, Thomas et al. 2004, Komodo et al. 2002), such as glaucous gulls (Larus hyperboreus), polar bears (Ursus maritimus), ringed seals (Phoca hispida) and beluga whales (Delphinapterus leucas).

2.3 Potential for long-range environmental transport

2.3.1 Environmental studies on transport and distribution

There are several factors indicating long-range transboundary transport of PeBDE in the environment. It has a high persistency in air, with a half-life of 11-19 days (Palm et al. 2002, Vulykh et al. 2004)). Monitoring studies have detected a widespread occurrence in the European (ter Shure et al. 2004, Lee et al. 2004, Jaward et al. 2004 a and b, Harrad and Hunter 2004, Harrad et al. 2004) and Arctic (AMAP 2002 and AMAP 2005, Peltola et al. 2001) atmosphere.

Jaward et al. (2004a) studied a total of 71 passive air samples using semi permeable membrane devices (SPMDs) for eight BDE congeners (BDE-28, BDE-47, BDE-49, BDE-75, BDE-99, BDE-100, BDE-153 and BDE-154) during a six week period in 2002 at remote/rural/urban locations across 22 countries in Europe. BDEs were detected in ca. 50% of the samples, and the equivalent ΣBDE air concentrations estimated from the passive sampler data ranged from 0.5 to 250 pg m$^{-3}$. The focus of the most elevated concentrations was the UK, which has a history of PBDE production and has also been a major user of PBDE formulations due to stringent fire regulations within the country. The UK is clearly a regional source for BDEs to the European atmosphere and, in contrast, levels reaching Europe from the west (over the Atlantic Ocean) are low. Other high values were detected in urban centres in mainland Europe – samples from Athens, Bilthoven (Netherlands), Geneva, Milan and Seville, for example. Non-detectable/very low values occurred in remote/background sites, especially in Iceland, Ireland, Norway and Sweden, and values in Eastern Europe were generally low. BDE-47 and BDE-99 contributed ca. 75% to ΣBDE, similar to their proportion in the Bromkal 70-5DE pentamix technical product.

Model results indicate that PBDEs will largely partition to organic carbon in soil and sediment and that their persistence will be strongly influenced by degradation rates in these media that are not well known, only a small proportion of PBDEs exist in air and water, suggesting that these compounds have limited LRAT potential (Prevedouros et al. 2004, Gouin and Harner 2003). This corresponds with PeBDEs affinity for carbon, low solubility in water (1.0 µg/L) and low vapour pressure (7.6 x 10-6 Pa). However, Gouin and Harner (2003) suggest that because of their physical–chemical properties, PBDEs may experience active surface–air exchange as a result of seasonally and diurnally fluctuating temperatures. Subsequently, this may result in the potential for LRAT of the PBDEs through a series of deposition/volatilisation hops, otherwise known as the “grasshopper” effect. This assumption is supported by
Lee et al. (2004) detected atmospheric concentrations of BDEs at two rural/semirural sites in England, and 1 remote site on the west coast of Ireland in 2001 and in 2000, respectively. $\Sigma$BDE concentrations at Mace Head, Ireland, were 0.22 to 5.0 pg m$^{-3}$ with a mean of 2.6 pg m$^{-3}$ and were controlled primarily by advection. $\Sigma$BDE concentrations at Hazelrigg (NW England) were 2.8 to 37 pg m$^{-3}$ with a mean of 12 pg m$^{-3}$, and at Chilton (SW England) were 3.4 to 33 pg m$^{-3}$ with a mean of 11 pg m$^{-3}$. The congener profile was, on average, similar to that of the commercial penta-mix PBDE formulation. At the two English sites in the summer, BDE concentrations were strongly influenced by temperature, indicating that land/air exchange processes play an important role in determining atmospheric concentrations.

BDEs were determined in soil samples collected along a latitudinal transect through the UK and Norway, at remote/rural woodland (both coniferous and deciduous) and grassland sites (Hassanin et al., 2004). Concentrations for $\Sigma$BDE ranged from 65 to 12,000 ng kg$^{-1}$ dry weight. BDE congeners BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154, the major constituents of the commercial pentamix PBDE formulation, dominated the average congener pattern in the soils. This was interpreted as evidence that transfer of the congeners from materials treated with the penta-mix product from source to soil occurs with broadly similar efficiency, and that there is little degradation of the congeners by processes acting either during atmospheric transport or within the soils themselves. There was evidence of latitudinal fractionation of the BDE congeners, with the relative amounts of BDE-47 and the lighter congeners increasing to the north (with increasing distance from source areas) while the proportion of BDE-99 and the heavier congeners decreased. Plots of BDE congener concentrations against percentage soil organic matter yielded different slopes for different congeners. Steeper slopes were generally observed for lighter congeners such as BDE-47, indicating that they have undergone some air-surface exchange (“hopping”), whilst those of heavier congeners such as BDE-153 were close to zero, indicating that they are retained more effectively by soil following deposition. A Japanese study detected seasonal variations in the partitioning of PBDEs between the gas and particulate phase. The fraction of particulate PBDEs was higher in samples collected in winter than those in the summer (Hayakawa et al., 2004). PeBDE is expected to be transported in the environment mostly by being absorbed onto particles due to its low volatility, low solubility and high affinity for carbon compounds. Results from environmental studies indicate that PBDE is transported on air borne particles, and that they are susceptible to wet deposition (ter Schure et al., 2004, ter Schure and Larsson 2002). Further transport depends on the fate of the particles. Fate after deposition on land depends on level of wind erosion that varies with season. Fate after deposition into the sea depends on oceanographic processes, as water layering and transport by currents in the surface layers.

Ter Schure et al. (2004) collected air and atmospheric bulk deposition samples on the island of Gotland in the Baltic Proper during a 10 week period in autumn 2001. The sampling site was chosen because of its central position in the Baltic Sea, and because of the absence of local point sources of pollution. Ten BDE congeners were determined (BDE-17, BDE-28, BDE-47, BDE-85, BDE-99, BDE-100, BDE-153, BDE-154, BDE-183 and BDE-209). The median $\Sigma$BDE concentration ($\Sigma$BDE is the sum of the concentrations of the congeners determined in each study) was 8.6 pg m$^{-3}$, and the BDEs were mainly associated with particles. A comparison to levels of PCB in the atmosphere indicated that, as PCB concentrations in Baltic air have been declining, the input of BDEs by atmospheric deposition to the Baltic Proper now exceeds that of the PCBs by a factor of almost 40 times.

BDEs were determined in precipitation falling in southern Sweden during a two week period in 2000 (ter Schure and Larsson, 2002). The particle-associated and “dissolved” phases were separated during sampling. 65 ± 18% of $\Sigma$BDE was found to be particle-associated. The volume weighted mean concentration of $\Sigma$BDE (nine congeners) in rain was 209 pg l$^{-1}$, and the total deposition rate was $2 \pm 1$ ng $\Sigma$BDE m$^{-2}$ day$^{-1}$. The congener profile in both phases of the total deposition was dominated by BDE-209, and thereafter BDE-47, BDE-99 and BDE-183, representing inputs from all three commercial PBDE formulations. The authors found that particle associated BDEs are effectively removed during small precipitation episodes, and that particle scavenging was an important mechanism for the wet deposition of BDEs.
A model assessment of potential for long-range transboundary atmospheric transport and persistence of PeBDE have been carried out by EMEP (Co-operative programme for monitoring and evaluation of the long-range transmission of air pollutants in Europe). The values of LRTP were considered to be strongly influenced by environmental processes, such as degradation, deposition, gas/particle partitioning, and gaseous exchange with underlying surface. The main process of removal from the atmosphere for the two congeners BDE-47 and BDE-99 was found to be deposition to land and seawater, 78% to land and 15% to sea for BDE-47 and 77% to land and 21% to sea for BDE-99. Only 7% of BDE-47 and 2% of BDE-99 was degraded. Calculated half-life in air was 7 days for BDE-47 and 11 for BDE-99. The findings showed a spatial distribution of BDE-47 that covers the Arctic, Europe, the Mediterranean Sea and northern Africa. BDE-99 spreads over longer distances and spreads to the Arctic, Atlantic Ocean, Asia and Africa. Transport distances (TD) were calculated for the two congeners. It was 2300 km for BDE-47 and 2600 km for BDE-99 (EMEP 2004).

The EU risk assessment (EU 2000) concluded that the major part of releases end up in soil. From soil, PeBDE can be expected to be moved mainly through leaching with water in the suspended solids fraction or through wind erosion where it occurs. A small part in the soil can be volatilised, especially in the warm season, and so may be considered a plausible alternative mechanism for transport in addition to volatilization and advective transport of vapor identified in the literature. Although PeBDE has low water solubility, it has been detected in lakes and seas, and can be transported with water in the soluble and particle phases (Peltola et al. 2001). Occurrence in migratory birds and fish indicate the possibility of transport by migration of animals, but the main route seems to be through the atmosphere.

2.3.2. Levels in remote areas

There are several studies showing the occurrence of PeBDE in remote areas in Europe as well (Vives et al. 2004, Hassanin et al. 2004 and Zenegg et al. 2003). Levels in remote regions are considered to be an indication on long-range transport.

A larger study was performed detecting BDEs in trout (three species) from eleven high mountain lakes in Europe (566 to 2,485m altitude) (Vives et al., 2004). These lakes were selected as being far from local pollution emission sources, and it was considered that the only source of BDEs to these lakes was as a result of atmospheric transport and deposition. The major congeners identified (of 39 determined) were BDE-47 and BDE-99, followed by BDE-100, BDE-153, BDE-154 and BDE-28, and these congeners were found in all samples analysed. The highest concentrations of ΣBDE in fish muscle and liver were found in Lochnagar, Scotland, 1.2 and 11 µg kg⁻¹ wet weight, respectively (177 and 366 µg kg⁻¹ on a lipid basis). No correlation was observed between the occurrence of these compounds and altitude, latitude or temperature, and the authors inferred that the environmental distribution of the BDEs has not, as yet, reached a steady-state.

3. EXPOSURE

3.1 Levels
PeBDE has a widespread occurrence in the environment globally. A large material of monitoring data exist with detected levels in marine and terrestrial birds, sea mammals, mammals, sediments, soil, seafood and fish. A global study by Ueno et al. (2004) of PeBDE in skipjack tuna (Katsuwonus pelamis) shows a wide spread occurrence in the offshore waters of various regions in the world. Table 2.3 gives an overview over the levels found in different parts of the world.

Exposure of the environment and biota in remote regions is of special concern for the preservation of natural resources, since it diminish the occurrence of untouched natural environment and can be a threat to vulnerable species and ecosystems. In the Arctic,
together with other pollutants of concern, PeBDE is detected in high levels in top predatory birds and mammals (Verreault et al. 2005, Verreault et al. 2004, Norstrøm et al. 2002, Herzke et al. 2003, Vorkamp et al. 2004a and b, Wolkers et al. 2004, Thron et al. 2004, Thomas et al. 2004, Ikomomou et al. 2002) showing that the Arctic food webs are seriously affected. Wolkers et al. (2004) detected levels of PeBDE in beluga whales (Delphinapterus leucas) in the Arctic, a species protected by the Convention on migratory species (the Bonn convention). ΣBDE concentrations (geometric mean; 22 congeners) were 234, 161 and 29 µg kg⁻¹ in juvenile, adult male and adult female beluga.

In fact, there are detected high levels of PeBDE in several species, with populations of concern protected by the Bonn convention. Several studies (Jaspers et al. 2004, Herzke et al. 2005, Lindberg et al. 2004, D’Silva et al. 2004, Law et al. 2005, Sinkkonen et al. 2004, Sellström et al. 2003, Kannan et al. 2005, Ramu et al. 2005 and Wolkers et al. 2004) indicate that PeBDE is widespread in peregrine falcon (Falco peregrine), merlin (Falco columbarius), goshawk (Accipiter gentiles), golden eagle (Aquila chrysaetos), buzzard (Buteo buteo), beluga whales (Delphinapterus leucas), irrawaddy dolphins (Orcaella brevirostris), and Indo-Pacific humpback dolphin (Sousa chinensis), all protected by the Bonn convention. High levels are also detected in eggs of peregrine falcon and golden eagle (Herzke et al. 2005, Lindberg et al. 2004). In a study on PBDE-levels in peregrine falcon eggs in Sweden (Lindberg et al. 2004) individual BDE concentrations were as high as 39,000 µg kg⁻¹ lipid weight, some of the highest concentrations seen in wildlife so far.

The populations of harbour porpoises (Phocoena phocoena) in the North and Baltic seas are protected through the Bonn Convention. Studies have detected high levels in those populations (Thron et al. 2004 and Covaci et al. 2002). In a study by Thron et al. (2004) animals with poor body condition (lower mean blubber thickness) had much higher concentrations than other individuals. Only females showed decreasing concentrations with age, indicating elimination via transfer from mother to offspring.

The harbour porpoise is together with peregrine falcon and merlin also on the list for strictly protected (endangered) species in the convention on the conservation of European wildlife and natural habitats (the Bern convention). The species white-tale sea eagle is on the list for endangered species in the Bern Convention. Levels of concern are detected in both individuals and eggs (Herzke et al. 2005). Beluga whales and irrawaddy dolphins are on list for protected (vulnerable) species. High levels are found in white-beaked dolphin (Lagenorhynchus albirostris), another endangered species. The parties of this convention undertake to take appropriate measures to ensure the conservation of endangered and vulnerable species and their habitats.

Table 2.3 Levels of PeBDE (BDE-99) in different parts of the world (LW=Lipid weight, DW=Dry weight).

<table>
<thead>
<tr>
<th>Country/Region</th>
<th>Organism/compartment</th>
<th>Levels of PeBDE</th>
<th>References</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Europe</td>
<td>Atmosphere, Gas phase</td>
<td>10-120 pg/m³</td>
<td>Jaward et al. 2004</td>
<td>22 countries</td>
</tr>
<tr>
<td>Japan</td>
<td>Atmosphere, Particulate, Gas phase</td>
<td>0.05-0.9 pg/m³, 0.05-19' pg/m³</td>
<td>Hayakawa et al. 2004</td>
<td>' measured in the summer</td>
</tr>
<tr>
<td>Sweden</td>
<td>sediments</td>
<td>&lt;0.7-51.4 ng/g DW</td>
<td>Palm et al. 2002</td>
<td>Rivers at point source</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>soil</td>
<td>78 – 3200 ng/g DW</td>
<td>Hassanin et al. 2004</td>
<td></td>
</tr>
<tr>
<td>Western Europe</td>
<td>sediments</td>
<td>&lt;0.2-6.9 ng/g DW</td>
<td>Palm et al. 2002</td>
<td>Estuaries</td>
</tr>
<tr>
<td>Japan, Osaka</td>
<td>sediments</td>
<td>9-28 ng/g DW</td>
<td>Palm et al. 2002</td>
<td></td>
</tr>
<tr>
<td>North Ocean, Pacific Ocean</td>
<td>Skipjack tuna</td>
<td>0.18-2.1 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td></td>
</tr>
<tr>
<td>Country</td>
<td>Animal Type</td>
<td>PBDE Concentration</td>
<td>Author(s)</td>
<td>Location/Region</td>
</tr>
<tr>
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</tr>
<tr>
<td>Japan</td>
<td>Skipjack tuna</td>
<td>1.1-1.7 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td>Offshore waters</td>
</tr>
<tr>
<td>East China Sea</td>
<td>Skipjack tuna</td>
<td>2.4-4.7 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td></td>
</tr>
<tr>
<td>Taiwan</td>
<td>Skipjack tuna</td>
<td>4.7 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td>Offshore waters</td>
</tr>
<tr>
<td>Philippines</td>
<td>Skipjack tuna</td>
<td>2.1 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td>Offshore waters</td>
</tr>
<tr>
<td>Brazil</td>
<td>Skipjack tuna</td>
<td>1.9 ng/g LW</td>
<td>Ueno et al. 2005</td>
<td>Offshore waters</td>
</tr>
<tr>
<td>Canada</td>
<td>Atlantic tomcod</td>
<td>77 ng/g LW</td>
<td>Law et al. 2003</td>
<td></td>
</tr>
<tr>
<td>Chilika Lake, India</td>
<td>Irrawaddy dolphin</td>
<td>0.12-0.78 ng/g LW</td>
<td>Kannan et al. 2005</td>
<td>endangered species</td>
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<tr>
<td>Hong Kong</td>
<td>Indo-Pacific humpback dolphin</td>
<td>33.6-720 ng/g LW</td>
<td>Ramu et al. 2005</td>
<td>Coastal waters 12% of ΣPBDEs</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>White beaked dolphin</td>
<td>1480 ng/g LW</td>
<td>Law et al. 2003</td>
<td>Endangered species</td>
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<tr>
<td>Hong Kong</td>
<td>Finless porpoises</td>
<td>27.6-117.6 ng/g LW</td>
<td>Ramu et al. 2005</td>
<td>Coastal waters 12% of ΣPBDEs</td>
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<td>Japan</td>
<td>Northern fur seal</td>
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<td>Kajiwara et al. 2004</td>
<td>Pacific coast 12% of ΣPBDEs</td>
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<td>Svalbard, Norway</td>
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<td>0.7-4.7 ng/g LW</td>
<td>Gabrielsen et al. 2004</td>
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<tr>
<td>Canadian Arctic</td>
<td>Polar bear</td>
<td>1.04-11.3 ng/g LW</td>
<td>Muir et al. 2006</td>
<td></td>
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<tr>
<td>Bjørnøya, Norway</td>
<td>Glacous gulls</td>
<td>0-7.9 ng/g LW</td>
<td>Herzke et al. 2003</td>
<td></td>
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<tr>
<td>Norway</td>
<td>White-tailed sea eagle</td>
<td>6-184 ng/g LW</td>
<td>Herzke et al. 2005</td>
<td>In eggs, Endangered Species</td>
</tr>
<tr>
<td>Sweden</td>
<td>Peregrine falcons</td>
<td>110-9200 ng/g LW</td>
<td>Lindberg et al. 2004</td>
<td>Endangered species</td>
</tr>
<tr>
<td>Australia</td>
<td>Melon-headed whale</td>
<td>4.8 ng/g LW</td>
<td>Law et al. 2003</td>
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<tr>
<td>Canada</td>
<td>Beluga whale</td>
<td>108 ng/g LW</td>
<td>Law et al. 2003</td>
<td>Vulnerable species</td>
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<tr>
<td>Netherlands</td>
<td>mussels</td>
<td>0.3-11 ng/g LW</td>
<td>Law et al. 2003</td>
<td>Marin+freshwater</td>
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<tr>
<td>Sweden</td>
<td>frog</td>
<td>5.6 ng/g LW</td>
<td>De Wit et al. 2004</td>
<td></td>
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<tr>
<td>Canada</td>
<td>Zooplankton</td>
<td>0.46 ng/g LW</td>
<td>Law et al. 2003</td>
<td></td>
</tr>
</tbody>
</table>

### 3.2 Trends

Most trend analysis show an increase in concentrations of PBDEs in the environment and in humans from the beginning of the 1970s, with a peak around the mid-1990s and a stabilisation or subsequent levelling off in Europe (Covaci et al. 2002, Fångström et al. 2005, Thomsen et al. 2005 and Knudsen et al. 2005), but with a continuous increase in the Arctic (Vorkamp et al. 2005, AMAP 2002 and AMAP 2005). PeBDEs are reported in the studies to follow the same trend as ΣPBDEs.

In the Asia-Pacific region a study on northern fur seals on the Pacific coast of Japan shows an increase of PBDEs to about 150 times between 1972 and 1994, and then levels decreased to about 50% in 1998 (Kajiwara et al. 2004). The reduction in PBDEs values was assumed to be due to the voluntary phase out of PeBDE in Japan in 1990. BDE-99 levels showed the same pattern as ΣPBDEs.
The results from a modelling exercise utilizing the European variant (EVn) BETR multimedia environmental fate model are presented for the technical PeBDE product (Prevedouros et al. 2004). To predict future atmospheric concentration trends, the model was used in its fully dynamic mode over the period 1970-2010. It was predicted that atmospheric concentrations peaked around 1997, and then declining with an overall “disappearance” half-life of 4.8 years. The model steady state simulations gave generally good agreement with measured data for BDE-47 and BDE-99.

Three dated sediment cores from locations in Western Europe were analysed for 14 BDE congeners (Zegers et al., 2003). Cores from the Drammenfjord (Norway), the western Wadden Sea (The Netherlands) and Lake Woserin (Germany) showed a time dependent pattern in the distribution of BDEs since the start of production of PBDE formulations. Two of the three commercial formulations could be distinguished. The penta-mix formulation is clearly present from the beginning of the 1970s. This is in agreement with data for the industrial production of this formulation. In the cores from the Netherlands and Germany, concentrations of BDE congeners associated with the penta-mix were levelling off in the most recent layers (1995 & 1997), whereas those in the Drammenfjord were still increasing in 1999. The absence of all BDE congeners in the older (deeper) layers of all three cores, as well as in several 100 to 150 million year old layers of clay from Kimmeridge, UK, indicated that these BDE congeners are not produced naturally.

3.3. Bioavailability

Environmental studies on bioavailability have detected uptake of PeBDE in soil organisms (Matscheko et al. 2002), sediment dwelling organisms (Magnusson et al. 2003) and aquatic organisms (Lithner et al. 2003, Voorspoels et al. 2003, Marsch et al. 2004, Kierkegaard et al. 2004, and Sinkkonen et al. 2004), making PeBDEs way into the food webs evident. Subsequent bioaccumulation and biomagnification of the compound has been detected and described in Section 2.2.2.

Soil exposed to PBDEs in various ways was analyzed for BDE-47, BDE-66, BDE-99, BDE-100, BDE-153, BDE-154 and BDE-183 (Matscheko et al., 2002). Earthworms collected at all soil sampling sites were analyzed as well. The BDE congener profile in all soil samples was dominated by BDE47 and BDE99. Accumulation of the compounds in earthworms from the sites yielded a direct relationship between the concentrations in the soil and concentrations in the worms. The biota-soil accumulation factors (BSAFs) of BDE congeners BDE47, BDE99 and BDE100 were around 5 (organic matter/lipids). Thus, earthworms living in contaminated soils will accumulate tissue BDE concentrations and, as these animals represent the base of the terrestrial food chain for many organisms, this form a pathway for the accumulation of BDEs in organisms at higher trophic levels.

The western Scheldt estuary (SE) is subject to a variety of suspected PBDE sources, such as a brominated flame retardant manufacturing plant, Antwerp harbour, and the textile industry located further upstream. BDE concentrations in samples of biota, including crab, shrimp, starfish, benthic fish (such as dab, goby, plaice and sole) and gadoid fish (such as bib and whiting) from the estuary (SE) were compared to those in samples from the Belgian North Sea (BNS) beyond the mouth of the estuary (Voorspoels et al., 2003). Eight BDE congeners (BDE-28, BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, BDE-183 and BDE-209) were determined. Concentrations observed in the SE samples were up to 30 times higher than in those from the BNS, with an increasing gradient towards Antwerp. Concentrations in the BNS ranged from 0.02 to 1.5 μg kg⁻¹ wet weight in benthic invertebrates and goby, from 0.06 to 0.94 μg kg⁻¹ wet weight in fish muscle, and from 0.84 to 128 μg kg⁻¹ wet weight in fish liver. The corresponding ranges in samples from the SE were from 0.2 to 30, 0.08 to 6.9, and from 15 to 984 μg kg⁻¹ wet weight, respectively. The ratio BDE-99/BDE-100 was found to be highly location- and species-dependent, possibly relating to differences in metabolism. In shrimp, the value of this ratio (4:1) was very similar to that observed in the Bromkal formulation and in SE sediment, and was similar in shrimp from both the BNS and SE, implying both that these congeners are readily bioavailable and that shrimp lack the ability to metabolise either congener. On a lipid weight basis, concentrations of BDE-47 ranged from 3 to 108 μg kg⁻¹ lipid weight in samples from the BNS, and from 8 to 1,550 μg kg⁻¹ lipid weight in SE samples. BDE-47 was the most abundant congener in all samples, comprising 43 to 75% of ΣBDE.
Thomas et al. (2004) conducted an input-output balance study of BDEs on three captive, juvenile grey seals. The animals were fed a diet of herring for six months, and the study was performed during the last three months of this period. BDE analysis was undertaken using GC-ECNIMS. Consistently high absorption (89 - 99%) was observed for all BDE congeners studied (BDE-28, BDE-47, BDE-49, BDE-99, BDE-100, BDE-153, BDE-154 and BDE-209).

Hydroxylated BDEs (OH-BDEs) have been detected and identified as metabolites in several species after exposure to specific BDE congeners but have also been found to occur as natural products in marine sponges and ascidians (Marsch et al., 2004). Methoxylated BDEs (MeO-BDEs) have also been reported as natural products present in marine sponges and green algae. Thus, the origin of these substances can be natural, anthropogenic or both. Nine OH-BDEs and six MeOBDEs were identified in Baltic Sea salmon (Salmo salar) blood using newly synthesized standards (Marsch et al., 2004). All of the identified OH- and MeOBDEs were substituted with four or five bromine atoms and five also had one chlorine substituent. Fourteen have the methoxy or hydroxy group substituted in the ortho-position to the diphenyl ether bond. The structures of several of the compounds support natural rather than anthropogenic origins. However, at least one of the OH-BDEs (4’-OH-BDE-49) may be a hydroxylated metabolite of BDE-47.

3.4 Human exposure

Studies have shown that food is the main route for human exposure, but also exposure to indoor air at home and workplaces, due to levels in products like furniture and electronic devices. Fish and agriculture products are the main food sources of PeBDE for humans, and mothers milk for the nursing child. Fatty fish from contaminated areas are a major source (Sjödin et al. 2003).

PeBDE has been detected in various foods (VKM 2005, Burniston et al. 2003 and Bocio et al. 2003) as well as in indoor dust (Shoeib et al. 2004 and Wilford et al. 2005).

There are several hazard assessments in EU and U.S. looking into the exposure of humans (VCCEP 2003, COT 2004, VKM 2005). They conclude that the available information is inadequate to fully characterize the risks.

Several studies have detected levels of PeBDE in sewage sludge (Matscheko et al. 2002, Fabrellas et al. 2004, Motche and Tanner 2004 and Sjödin et al. 2003). Sewage sludge is considered to be one of the main sinks for PBDEs. The application of sewage sludge to agricultural land is one of the reasons for detected levels of PeBDE in food products. This can explain the detected levels in vegetables and root crops in experimental studies. Levels in fish and root crops can be the source of exposure to domestic animals like chickens and pigs, and the source of PBDEs in meat products for human nourishment.

A Canadian global study showed that PeBDE is widespread in human milk in populations all over the world (Ryan 2004). There are data on levels in human blood serum and milk from USA, Canada, Mexico, Japan, the EU region, the Arctic region and Scandinavia.

Increasing levels between the 1980s to the 2000s have been observed in mother’s milk from Sweden as well as in blood from Germany and Norway (Sjödin et al. 2003). A more recent study in Sweden (Fängström et al. 2005) assessed the temporal trends of polybrominated diphenyl ethers (PBDEs), in mothers’ milk in the Stockholm area. The pooled samples were covering the time period 1980 to 2004, with emphasis on samples from the last ten years. Concentrations of BDE-47, BDE-99 and BDE-100 reached a peak in the mid-1990s and are now clearly showing decreasing levels. The concentrations are however still much higher than in 1980.

The objective of a recent Norwegian study was to complete and extend a previous study on time trends of PBDEs in Norwegian pooled serum samples (Thomsen et al. 2005). These levels was compared with levels in other human samples from Norway in order to put together an overview of the PBDE body burden in the general population from 1977 to 2004. The temporal trend of the sum of 7 PBDEs (28, 47, 99, 100, 153, 154 and 183) in the pooled serum from the present study are in close agreement with the levels found in a previous study.
by the same authors, except for the pools from 1991 and 2002 which were found to be considerably higher than expected from earlier results of preceding and following years. This was surprising as the pools contained at least twenty individual samples (mean age 40 – 50 years). In the samples from 2002, the mean of sum seven PBDEs is 3.8 ng/g lipid (serum from the youngest group excluded) and 3.5 ng/g lipids in men age 25-59 years. In general, for similar time periods the levels in breast milk seem to be somewhat lower than in the serum, but the same overall trend is observed. This confirms that the PBDE body burdens have risen rapidly from 1977 to about 1997, but now seem to have stabilized or even to have decreased. This is in accordance with the trends observed in Swedish breast milk. The PBDE level was previously found to be about twice as high in a serum pool from infants up to four years of age compared to serum pools from elderly persons. This finding was confirmed in the Norwegian study. However, in 2002, children between the ages of 5 and 14 years showed higher levels of PBDEs than the average adult.

Contemporary PBDE concentrations in Europe and Asia are remarkably similar with low median values on a lipid basis for all countries and relatively small variations. The situation in North America is completely different with median values for individual studies in the range of 20-50 ng/g LW (Ryan 2004). The levels of breast adipose tissue taken from women living in San Francisco Bay area in 2000 were almost two orders of magnitude higher than what has been reported in human milk from Sweden (Sjödin et al. 2003).

In a preliminary study screening levels of PBDEs in plasma and milk samples from Mexican women detected the levels were well above European levels of PBDEs reported so far (López et al. 2004). The mean level of PBDEs (with BDE-209 excluded) in Mexican women living in urban areas was approx. 20 ng/g LW in plasma. The levels in women living in rural areas in Mexico were however comparable with women living in rural areas in Sweden. (BDE-209 levels were only detected in women living in the Mexican city).

Ryan (2004) detected a big individual variation in levels in the general population in a study from Canada. The values span more than three orders of magnitude, with a few values showing a much greater level.

Levels detected in the Canadian Arctic in Ryan study (2004) were increasing. Values in human milk from the Faroe Islands showed the same trend (Fängström et al. 2004).

Two studies in Australia indicated that levels of PBDEs in Australian breast milk and blood serum are higher than in Europe but lower than in North America (Harden et al. 2004 and 2005).

Table 2.5 Data on mean levels of PeBDE(BDE-99) (ng/g LW) in humans from different parts of the world.

<table>
<thead>
<tr>
<th>Data</th>
<th>Country/region</th>
<th>Levels</th>
<th>References</th>
<th>Year</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>blood</td>
<td>The Netherlands</td>
<td>0.8</td>
<td>Weiss et al. 2004</td>
<td>unknown</td>
<td></td>
</tr>
<tr>
<td>blood</td>
<td>Norway</td>
<td>1.0</td>
<td>Thomsen et al. 2004</td>
<td>1999</td>
<td></td>
</tr>
<tr>
<td>blood</td>
<td>Mexico</td>
<td>2.0</td>
<td>López et al. 2004</td>
<td>2003</td>
<td>Urban population</td>
</tr>
<tr>
<td>blood</td>
<td>Australia</td>
<td>2.3</td>
<td>Harden et al. 2004</td>
<td>2003</td>
<td></td>
</tr>
<tr>
<td>milk</td>
<td>Germany</td>
<td>0.2</td>
<td>Harden et al. 2004</td>
<td>2000</td>
<td></td>
</tr>
<tr>
<td>milk</td>
<td>Sweden</td>
<td>0.3</td>
<td>Fängström et al. 2005</td>
<td>2003</td>
<td>Urban population</td>
</tr>
<tr>
<td>milk</td>
<td>Mexico</td>
<td>0.6</td>
<td>López et al. 2004</td>
<td>2003</td>
<td>Rural population</td>
</tr>
<tr>
<td>milk</td>
<td>Sweden</td>
<td>0.5</td>
<td>López et al. 2004</td>
<td>2003</td>
<td>Rural population</td>
</tr>
<tr>
<td>milk</td>
<td>United Kingdom</td>
<td>0.9</td>
<td>Harden et al. 2004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>milk</td>
<td>Faroe Islands</td>
<td>1.0</td>
<td>Fängström et al. 2004</td>
<td>1999</td>
<td>Rural population</td>
</tr>
<tr>
<td>milk</td>
<td>Australia</td>
<td>1.9</td>
<td>Harden et al. 2005</td>
<td>2002/2003</td>
<td></td>
</tr>
<tr>
<td>milk</td>
<td>Canada</td>
<td>4</td>
<td>Ryan et al. 2002</td>
<td>2002</td>
<td>Rural population</td>
</tr>
</tbody>
</table>
In Sweden, occupational exposure to PBDE has been identified among electronics recycling personnel (Sjödin et al., 1999) and in technicians responsible to repair and maintain computers (Jacobsson et al., 2002). Also workers in industry manufacturing PeBDE, polyurethane foam and electronic equipments can be exposed to PeBDE.

### 4. HAZARD ASSESSMENT FOR ENDPOINTS OF CONCERN

Evidence to date suggests that the major congeners of the technical PeBDE formulation, BDE-47 and BDE-99, are likely to be the more toxic and bioaccumulative than other PBDE compounds congeners. The toxicology of PBDEs is not well understood, but PBDEs have been associated with tumours, neurodevelopmental toxicity and thyroid hormone imbalance. The neurotoxic effects of PBDEs are similar to those observed for PCBs. Children exposed to PBDEs are prone to subtle but measurable developmental problems. It is presumed that PBDEs are endocrine disrupters, but research in this area is scant (Siddiqi et al. 2003).

#### 4.1 Effects in wildlife

In a review article on toxic effect of brominated flame retardents Darnerud (2003) concluded that exposure to PBDEs gives rise to adverse effects in experimental *in vivo* models, and depending on type of product different effects are seen, occurring at varying dose levels. Generally, the technical PeBDE products cause effects at the lower dosages. The critical effects of PeBDEs are those on neurobehavioral development and, although somewhat less sensitive, thyroid hormones in offspring (from 0.6 to 0.8 and 6 to 10 mg/kg body wt., respectively) (Darnerud 2003).

Recent studies show that exposure to BDE-47 can cause growth inhibition in colonies of the plankton algae (*Skeletonema costatum*) and a depression on reproductive output of the zooplankton *Daphnia magna* (Källqvist et al. 2006).

Blubber biopsy and blood samples were collected from weaned grey seal (*Halichoerus grypus*) pups and juveniles during 1998 and 1999 (Hall et al., 2003). Fifty four post-weaned pups and fifty five first year juveniles were studied, of which thirteen 13 were recaptured post weaned pups. The median concentrations of ΣBDE (14 congeners) were 0.17 and 0.46 µg kg$^{-1}$ lipid weight in the blubber of the pups and the juveniles, respectively. The study indicated that thyroid hormone levels in the blood of grey seals during their first year of life were significantly, and positively, related to ΣBDE concentrations in blubber, after accounting for the effects of possible confounding variables. Such an association is not, in itself, sufficient evidence for a causal relationship, but is in accordance with the hypothesis that these compounds can act as endocrine disrupters in grey seal pups.

#### 4.2. Effects in mammals:

Darnerud (2003) concluded in his review that for PeBDEs, the critical effects among the available studies seem to be developmental neurotoxicity and, generally at somewhat higher doses, altered thyroid hormone homeostasis. Regarding the neurotoxicity in mice, no clear mechanism could be defined but effects of the PeBDEs both via thyroid hormone disruption and directly on signal transmission in brain have been discussed. For example, PBDEs were capable to induce cell death of cerebellar granule cells in culture (Reistad et al., 2002). The LOAEL value for PeBDE could be set to 0.6–0.8 mg/kg body wt., based on the most sensitive effect observed, neurobehavioral effects during early development (Darnerud 2003).

In a hazard assessment by the Committee on food safety in Norway (VKM 2005) the following toxic effects of exposure to BDE-99 or the technical pentamix formulation was reported; neurotoxicity, effects on neurobehavioral development, effects on the thyroid hormone system and histopathological alterations in the thyroidea and liver.

<table>
<thead>
<tr>
<th>milk</th>
<th>USA</th>
<th>28</th>
<th>Pâpke <em>et al.</em> 2001</th>
<th>2000</th>
<th>Urban population</th>
</tr>
</thead>
</table>

Table 2.6. Overview of No Observed Effect level (NOEL) and Lowest Observed Effect Level (LOEL) after oral administration of BDE-99 congener or commercial penta-mix formulations. Bold values are the lowest LOEL or NOEL detected. Most of the studies are in line with the OECD test guidelines and the others have a quality assessed to be adequate.
<table>
<thead>
<tr>
<th>PBDE</th>
<th>Duration</th>
<th>Dose</th>
<th>NOEL mg/kg/day</th>
<th>LOEL mg/kg/day</th>
<th>Endpoint</th>
<th>Species</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDE-99</td>
<td>s.d</td>
<td>0.8 or 12.0 mg/kg</td>
<td>n.d.</td>
<td>0.8</td>
<td>Neurotoxicity</td>
<td>mouse</td>
<td>Eriksson et al. 2001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Behaviour, motor activity level and learning</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDE-99</td>
<td>s.d</td>
<td>0.6, 6, or 30 mg/kg</td>
<td>n.d.</td>
<td>0.6</td>
<td>Developmental-and neurotoxicity</td>
<td>mouse</td>
<td>Branchi et al. 2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Behaviour - hypoactive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDE-99</td>
<td>s.d</td>
<td>0.4, 0.8, 4.0, 8.0, or 16 mg/kg</td>
<td>0.4</td>
<td>0.8</td>
<td>Developmental-and neurotoxicity</td>
<td>mouse</td>
<td>Viberg et al. 2004 Sand et al. 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Behaviour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDE-99</td>
<td>s.d.</td>
<td>0.06 and 0.3 mg/kg to pregnant female</td>
<td>n.d.</td>
<td>0.06</td>
<td>Developmental-and neurotoxicity</td>
<td>rat, F1 gen.</td>
<td>Kuriyama et al. 2005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Behaviour (increased activity)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDE-99</td>
<td>s.d.</td>
<td>0.06 and 0.3 mg/kg to pregnant female</td>
<td>0.06</td>
<td>0.3</td>
<td>Reduced testis size and number of sperms</td>
<td>rat, F1 gen.</td>
<td>Kuriyama et al. 2005</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>30 d</td>
<td>0.01, 0.05, 0.1, 0.5, or 1.0 mg/kg/dag</td>
<td>1</td>
<td>n.d.</td>
<td>Growth, food intake, hematology, histopathology clinical chemistry</td>
<td>rat</td>
<td>Great lakes Chemical Corporation 1985</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>30 d</td>
<td>0, 3, 30, or 60 mg/kg/dag</td>
<td>3</td>
<td>30</td>
<td>Liver weight, puberty, reproduction, liver enzymes, T&lt;sub&gt;4&lt;/sub&gt;-reduction</td>
<td>Male rat</td>
<td>Stoker et al. 2004</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>30 d</td>
<td>0, 3, 30, or 60 mg/kg/dag</td>
<td>n.d.</td>
<td>3</td>
<td>T&lt;sub&gt;4&lt;/sub&gt;-reduction</td>
<td>Female rat</td>
<td>Stoker et al. 2004</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>35 d</td>
<td>0, 1, 10 eller 30 mg/kg/dag</td>
<td>1</td>
<td>10</td>
<td>T&lt;sub&gt;4&lt;/sub&gt;-reduction</td>
<td>pregnant rat</td>
<td>Zhou et al. 2002, Zhou et al. 2001</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>90 d</td>
<td>0-0.44 mg/kg/dag</td>
<td>n.d.</td>
<td>0.44</td>
<td>Liver enzymes</td>
<td>rat</td>
<td>Carlson 1980</td>
</tr>
<tr>
<td>Penta mix DE-71</td>
<td>90 d</td>
<td>0, 2.10, eller 100 mg/kg/dag</td>
<td>0-2</td>
<td>2-10</td>
<td>Hepatocyto-megali Tyreoida hyperplasi</td>
<td>rat</td>
<td>Great lakes Chemical Corporation 1984</td>
</tr>
</tbody>
</table>

n.d.=not defined, s.d.=single dose
PBDE mixture, DE-71 (containing BDE-47, BDE-99, BDE-100, BDE-153, BDE-154) delays the puberty and suppresses the growth of androgen-dependent tissues in male Wistar rat following a peri-pubertal exposure. These effects suggest that DE-71 may be either inducing steroid hormone metabolism or acting as an androgen receptor (AR) antagonist (Stoker et al. 2005).

Talsness et al. (2005) evaluated the effects of environmentally relevant concentrations (low doses) of BDE-99 on the female reproductive system in rats. Ultra structural changes compatible with altered mitochondrial morphology were observed in the ovaries of the F1 offspring. No statistically significant changes in ovarian follicle counts were observed. External and skeletal anomalies were detected in offspring (F2) from two different dams (F1) with early developmental exposure to 300 μg BDE-99/kg BW. Exposure to BDE-99 resulted in female reproductive tract changes in the F1 generation which were apparent at adulthood.

In utero exposure to a single low dose of BDE-99 disrupts neurobehavioral development and causes permanent effects on the rat male reproductive system apparent in adulthood (Kuriyama et al. 2005). In a study by Kuriyama et al. (2005) the effects of developmental exposure to BDE-99 on juvenile basal motor activity levels and adult male reproductive health was assessed. The exposure to low-dose BDE-99 during development caused hyperactivity in the offspring at both time points (postnatal days 36 and 71) and permanently impaired spermatogenesis by the means of reduced sperm and spermatid counts. The doses used in this study of 60 and 300 μg/kg BW are relevant to human exposure levels, being approximately 6 and 29 times, respectively, higher than the highest level reported in human breast adipose tissue. This is the lowest dose of PBDE reported to date to have an in vivo toxic effect in rodents and supports the premise that low-dose studies should be encouraged for hazard identification of persistent environmental pollutants.

The study by Viberg et al. (2004) shows that neonatal exposure to BDE-99 can induce developmental neurotoxic effects, such as changes in spontaneous behaviour (hyperactivity), effects that are dose-response related and worsen with age. The changes are seen in C57/B1 mice of both sexes. Spontaneous behaviour (locomotion, rearing, and total activity) was observed in two-, five- and eight month-old mice.

4.3 Toxicity to humans
Several hazard assessments have been produced in EU and in U.S. The conclusions in the hazard assessments elaborated are the lack of sufficient knowledge of the toxicology of PeBDE to assess the risk to humans (COT 2004, VKM 2005 and VCCEP 2003). The toxicological importance for humans of detected effects in laboratory animals is not clear. There is still not enough knowledge of the mechanisms, half-life and metabolism of PeBDE in experimental animals and humans (VKM 2005).

The conclusion in the hazard assessment by the Committee on food safety in Norway was that the exposure through food and mother’s milk is considerably lower than the observed NOEL in laboratory mammals (VKM 2005). But long-time exposure to lower doses of PeBDE can cause health effects, since PeBDE accumulates in the human body. Since the half-life of PeBDE in humans is not known it is not possible today to conclude on long-time exposure effects.

Vulnerable groups could however be pregnant women, embryos and infants, because of effects on the thyroid hormone balance, and the embryo’s development of the central nervous system. During pregnancy, maintenance of the thyroid hormone balance is a physiological challenge. Embryos and infants are particularly vulnerable for reductions in thyroid hormone levels (VKM 2005). Infants are exposed to PeBDE through the diets of their mothers’ milk, since PeBDE is lipophilic and accumulates in the milk (VKM 2005).

3. SYNTHESIS OF INFORMATION
The pentabromodiphenylether (PeBDE) commercial product is a mixture of primarily tetra-through hexaBDE congeners (plus trace amounts of triBDE and 0-1% heptaBDE). It is used
for flame retardant purposes as an additive in consumer products. The ratio of the PBDE-congeners in commercial PeBDE mixtures is different in different regions of the world.

A substantial range of studies on PeBDE are identified. New findings support the conclusion that PeBDE’s properties fulfil the screening criteria in Annex E of the Stockholm Convention.

PeBDE is released into the environment during the manufacture of the commercial PeBDE mixture, the manufacture of products, during their use and after they have been discarded as waste. The main source in North America and Western Europe has been products with polyurethane foam, but is now mainly phased out. The information is too limited to draw conclusions on the importance of other uses, like textiles, electric and electronic products, drilling oil fluid and rubber products. Dismantling and reuse of electric and electronic consumer goods can be an extensive source for releases of PeBDE. In addition detailed information on use is lacking for many regions of the world.

The releases are to air, water and soil. The major part of the releases ends up in soil. The distribution between the environmental compartments is: soil>>water>air. The main part of PeBDE in the environment is bound to particles; only a small amount is transported in its gaseous phase or diluted in water.

Due to PeBDEs high persistency in air, the main route for long-range transport is through the atmosphere. Modelling and environmental studies indicate that the transport is through a series of deposition/volatilisation hops towards the poles. Long-range transport through water and emigrating animals is also likely. Several studies show that PeBDE in soil and sediments is bioavailable, enters the food chain and that it bioaccumulates and biomagnifies in the food webs, ending up in high levels in top predators.

PeBDE is widespread in the global environment and in humans. Vulnerable ecosystems and species are affected, among them several endangered species. Some individuals of endangered species show high levels of concern. The potential for the toxic effects in wild life and mammals is evident.

The exposure to humans is through food, use of products and indoor air and dust. PeBDE transfers from mothers to embryos and lactating infants. The detected levels are considerably lower than observed NOELs in laboratory mammals. But knowledge is too scarce to conclude on the effects of long-term exposure. Vulnerable groups can be pregnant women, embryos and infants.

Most countries have ceased their production. Uses of PeBDE have been phased-out in several countries, but are still on the market in big regions of the world.

Table 1. POP characteristics of PeBDE (from “Annex to decision POPRC-1/3” in UNEP/POPS/POPRC.1/10)

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Meets the criterion (Yes/No)</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potential for Long-Range Atmospheric Transport</td>
<td>Yes</td>
<td>PeBDE has a low vapour pressure (9.6.10⁻⁸–4.7.10⁻⁵ Pa) and modelling data show an estimated half-life in air greater than two days. The estimated half-lives for PBDE-47 and PBDE-99 in air are between 10 and 20 days. Monitoring data show that the substance is found in remote areas. PeBDE congeners have been found in Arctic air with a concentration range from &lt;1 to 20 pg/m³. There is also a substantial amount of monitoring data in marine mammals, birds, fish, lake sediments, etc., in remote areas.</td>
</tr>
<tr>
<td>Toxicity (Adverse Effects)</td>
<td>Yes</td>
<td>There is evidence of reproductive toxicity in invertebrates and fish. The EC₅₀ for larval development for marine copepod ranged between 13 and 4 mg/L for PBDE-47 and PBDE-99,</td>
</tr>
</tbody>
</table>
respective. The lowest-observed-adverse effect level (LOAEL) in rodents for developmental neurotoxicity and liver toxicity ranged from 0.6 to 10 mg/kg body weight/day

<table>
<thead>
<tr>
<th>Persistence</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deposits of PBDE congeners that were present in marine sediments a few decades ago are still present in clearly quantifiable amounts</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bioaccumulation</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log Kow is greater than 5 (log Kow values 6.46–6.97). The reported bioconcentration factors for <em>Cyprinus carpio</em> are 66,700 for PBDE-47 and 17,700 for PBDE-99. Data from around the world demonstrate increasing levels of PeBDE congeners with rising trophic position. Recent publications confirm food chain transfer in the Arctic.</td>
<td></td>
</tr>
</tbody>
</table>

Note: The inclusion of this table has been proposed. A table with conclusions from this risk profile seems to be more adequate.

### 4. Concluding statement

Based on the information in this risk profile and otherwise made available to the POPRC, PeBDE is likely, as a result of long-range environmental transport, to cause significant adverse effects on human health or the environment, such that global action is warranted.

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67 PBDE-47 and PBDE-99 are two major congener components of the PeBDE commercial mixture (i.e., 2,2’,4,4’-tetrabromodiphenyl ether and 2,2’,4,4’,5-pentabromodiphenyl ether respectively).
RISK MANAGEMENT EVALUATION

Executive Summary

Background

Commercial Pentabromodiphenyl ether (C-PeBDE) is a mixture of brominated flame retardants (BFRs), mainly isomers of Pentabromodiphenyl ether (PeBDE) and Tetrabromodiphenyl ether (TeBDE). Brominated flame retardants are a group of brominated organic substances that inhibit or suppress combustion in organic material. They are or have been used almost exclusively in the manufacture of flexible polyurethane (PUR) foam for furniture and upholstery in homes and vehicles, packaging, and non-foamed PUR in casings and electronic equipment (EE). They are also used to some extent in specialized applications in textiles and in industry. The chemical properties of PeBDE have lead to its wide-spread occurrence in the environment and in humans, and there is evidence of its toxicity, of this reason C-PeBDE causes concern in many regions of the world.

There are national and international standards for fire safety for some product groups. This applies for example to electrical equipment, industrial packaging, upholstered furniture, curtains, electronic household appliances and electrical cables. These standards specify the flame-retarding properties that are required but not which flame retardants are to be used. Until now, brominated flame retardants have been considered to be the cheapest and most efficient. Today, it has become increasingly more common to replace these substances either with flame retardants without bromine or by changing the design of the product so that there is no need for the continued use of flame retardants. It has also become important to avoid the use of products containing flame retardants if this is not absolutely necessary on the basis of fire safety. Accordingly, some manufacturers have already replaced C-PeBDE with cost-competitive non-POPs alternatives in all uses, including PUR and electronics.

High levels of PeBDE are detected in the environment, the substance has severe toxic properties and has been shown to be persistent and bioaccumulative. It thus represents a potential risk for future generations. Concentrations in wildlife and in humans have also increased significantly (RPA, 2000). Those findings have resulted in voluntary and regulatory phase-outs of PeBDE in several regions in the world. Since this is a global, transboundary problem, global actions to phase out C-PeBDE should be considered.

Conclusion and recommendation

Having evaluated the risk profile for commercial PeBDE (C-PeBDE), and having concluded that this chemical is likely, as a result of long-range environmental transport, to lead to significant adverse effects on human health and the environment, this risk management evaluation has been prepared, as specified in Annex F of the Convention.

In accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends that the Conference of the Parties to the Stockholm Convention consider listing of brominated diphenyl ethers with 4 or 5 bromines in Annex A, and specifying the related control measures.

1. Introduction

1.1 Chemical identity of the proposed substance

Background

In 2005 Norway nominated commercial Pentabromodiphenyl ether (C-PeBDE) to be listed as a persistent organic pollutant (POP) under Annex A of the Stockholm Convention. The convention is a global treaty to protect human health and the environment from POPs. Twelve
substances (or groups of substances) are currently listed under the convention. POPs are chemicals that remain intact in the environment for long periods, become widely distributed geographically, accumulate in living organisms and can cause harm to humans and the environment.

The POP Review Committee is considering a global ban on the production and use of C-PeBDE. As part of this consideration a questionnaire was sent to Parties and Observers to the Convention, asking for information about C-PeBDE as defined in Annex F of the Convention. Based on the information from the questionnaires and other sources, Norway has been responsible for the drafting of the present Risk Management Evaluation of a global ban on C-PeBDE.

PeBDE is a brominated flame retardant (BFR). Because of its chemical and toxic properties and wide spread occurrence in the environment and in humans PeBDE causes concern in many regions in the world. Brominated flame retardants are a group of brominated organic substances that inhibit or suppress combustion in organic material. They are or have been used almost exclusively in the manufacture of flexible polyurethane (PUR) foam for furniture and upholstery in homes and vehicles, packaging, and non-foamed PUR in casings and electric and electronic equipment (EE). To some extent they have also been used in specialized applications in textiles and in various other uses.

Chemical identity of the proposed substance
Commercial pentabromodiphenyl ether (C-PeBDE) refers to mixtures of bromodiphenyl ether congeners in which the main components are 2,2', 4,4'-tetrabromodiphenyl ether (BDE-47 CAS No. 40088-47-9) and 2,2',4,4',5-pentabromodiphenyl ether (BDE-99 CAS No. 32534-81-9), which have the highest concentration by weight with respect to the other components of the mixture. Hexabrominated diphenylethers (HeBDE) species can also comprise a significant portion of C-PeBDE. The formulation of C-PeBDE used in North America and Europe contains 4-12% HeBDE.

The numbering system for the PBDEs is the same as that used for polychlorobiphenyls (PCBs) (Ballschmiter et al. 1993).

The acronym PBDE is used for the generic term polybromodiphenyl ether, covering all congeners of the family of brominated diphenyl ethers. It is sometimes abbreviated to BDE.

1.2 Conclusions of the Review Committee of Annex E information
Annex E of the Stockholm Convention requires a Risk Profile to be developed to evaluate whether the chemical is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted. A Risk Profile for C-PeBDE was developed and accepted in 2006 (UNEP, 2006). The POP Review Committee concluded as follows:

“Pentabromodiphenyl ether (C-PentaBDE) is a synthetic mixture of anthropogenic origin with no known natural occurrence. It can be concluded therefore that the presence of components of C-PentaBDE in the environment is the result of anthropogenic activities. Long range transport must be responsible for its presence in areas such as the Arctic region, remote from sites of production and release. PentaBDE degrades slowly in the environment and can bioaccumulate and biomagnify in mammals and piscivorous birds.

The phase out of C-PentaBDE production and use has led to a reduction in current use, but many materials in use, such as polyurethane foams and plastics in electronic equipment, contain PeBDE which is slowly released to the environment. This release will be accelerated at end-of-life of such materials, especially during recovery and recycling operations.

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68 Aldrin, cglordane, DDT, dieldrin, endrin, heptachlor, hexachlorobenzene, mirex, toxaphene, PCbs, dioxins and furans.
Although levels of PeBDE in human blood and milk, and in other environmental species, are falling in Europe, they continue to increase in North America and the Arctic region.

Based on the information in this risk profile, C-PentaBDE, due to the characteristics of its components, is likely, as a result of long-range environmental transport and demonstrated toxicity in a range of non-human species, to cause significant adverse effects on human health and the environment, such that global action is warranted.

1.3 Data sources
In general, the information submitted from countries is limited. In particular, there is a lack of reliable data on production and consumption, and cost data of using alternatives. In addition, information concerning the positive and negative impacts on society of regulations is limited. Sufficient data on control actions already taken have been submitted, and some data on the use of control measures and on control and monitoring capacity in developed countries are available from some questionnaires.

Other data sources
Submissions with reference to more comprehensive studies like the risk reduction strategy study submitted by UK on behalf of the EC (EPA 2000) and the study of management options developed under the Convention on Long Range Transboundary Pollution submitted by Norway, have made it possible to do this risk management evaluation.

Information on national and regional regulations, or voluntary phase out of the use and production of C-PeBDE from developing countries are available, but information from developing countries is limited.

Most information on use and production patterns is from around 2000, when background material was gathered for management evaluations in the developed regions. This information has been updated in this document with complementing studies up to 2007. After 2000 production and new use of C-PeBDE has been phased out in most of the developed regions. There is no new information on use in US, where the production has been voluntarily phased out. Information was received about recent regulatory action in Australia and China.

The collected information would be inadequate for a quantitative analysis of the risks of phasing out production and use of C-PeBDE. However, the available data are sufficient to prepare a qualitative risk management evaluation consistent with the requirements of the Stockholm Convention.

1.4 Status of the chemical under international conventions
While C-PeBDE is being considered for listing under the Stockholm Convention, PeBDE is treated under several other international conventions. Below is a brief overview of the most important ones, based on UNEP (2006).

The OSPAR Convention
The Convention for the Protection of the Marine Environment of the North-East Atlantic (the OSPAR Convention) is guiding international cooperation on the protection of the marine environment of the North-East Atlantic. OSPAR’s objective is to make every endeavour to move towards the cessation by the year 2020 of discharges, emissions and losses of hazardous substances which could reach the marine environment.

In 1998 the OSPAR Commission placed PBDEs on its “List of Chemicals for Priority Action”. An OSPAR Commission background document on PBDEs was reviewed by Sweden in 2001. The next full review of this document is not planned before 2008. At the 4th North Sea Conference, it was decided to phase out the use of brominated flame retardants by 2020.

The UNECE Convention on Long-range Transboundary Air Pollution
The United Nations Economic Commission for Europe (UNECE) works for sustainable economic growth among its 55 member countries. The UNECE Convention on Long-range Transboundary Air Pollution requires Parties to endeavour to limit and, as far as possible, gradually reduce and prevent air pollution including long-range transboundary air pollution. The Convention has been extended by eight protocols. The Protocol for POPs focuses on a list of 16 substances that have been singled out according to agreed risk criteria for total ban, elimination at a later stage or restrictive use. In 2005, C-PeBDE was nominated as a new POP to the Convention by Norway. In December 2005 PeBDE was considered by the Executive Body of the Convention to meet the screening criteria for POPs. In 2006 the management options C-PeBDE were assessed to give a basis for later negotiations on restrictions.

The Rotterdam Convention

The Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade is a multilateral environmental agreement designed to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals. It is an instrument to provide importers of products with the power to make informed decisions on which chemicals they want to receive and to exclude those they cannot manage safely.

The Convention entered into force in 2004 and today has more than 100 states as Parties. The EU notified PeBDE to the Convention in 2003. To become a candidate ban of the substance must be notified by two Parties. However it is not at this time a candidate for consideration to be added to Annex III of the convention (a list of pesticides and industrial chemicals that have been banned or severely restricted in countries or regions for health or environmental reasons).

Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and their Disposal

Should C-PeBDE be listed under the Stockholm Convention, and thus accepted as a POP, it will come under the aegis of the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and their Disposal. The Basel Convention is the most comprehensive global environmental agreement on hazardous and other wastes. The Convention has 169 Parties and aims to protect human health and the environment against the adverse effects resulting from the generation, management, transboundary movements and disposal of hazardous and other wastes. The Basel Convention came into force in 1992.

Under the Convention 5 guidelines on POP waste were developed and adopted in 2006.

Strategic Approach to International Chemicals Management (SAICM)

Should C-PeBDE be listed under the Stockholm Convention, and thus accepted as a POP, it will come under the aegis of the Strategic Approach to International Chemicals Management (SAICM), a policy framework for international action on chemical hazards that was adopted by the International Conference on Chemicals Management on 6 February 2006, in Dubai, United Arab Emirates. SAICM supports the achievement of a goal agreed at the 2002 Johannesburg World Summit on Sustainable Development, which was to ensure that by the year 2020 all chemicals are produced and used in ways that minimize adverse impacts on human health and the environment. SAICM includes substances with POPs characteristics as a class of chemicals to be prioritized for halting of production and use, and substitution by safer alternatives (SAICM Overarching Policy Strategy 2002).

1.5 Any national or regional control actions taken

Most developed countries have taken some actions to limit the production and use of PeBDE.

- Australia: PeBDE is effectively banned for use in new articles. Imports of articles containing BFRs are not regulated.
- EU: placing on the market and use in concentrations higher than 0.1 % by mass is banned from 2004 (EU-Directive 2003/11/EC). Use in electrical and electronic
appliances was phased out from July 1st, 2006 under the EU's Restriction of Hazardous Substances in electrical and electronic equipment. Products containing more than 0.25 % PeBDE are classified as hazardous waste when they are discarded.

- US: the industry voluntarily ceased production from 2005, and the use is forbidden in some states. USEPA requires notification and Agency review prior to restart of manufacture for any use (see rule at 40 CFR Part 721:10000).
- Japan: use stopped voluntarily in 1990.
- Norway and Switzerland: both countries have banned production, import, export and marketing and use of PeBDE and mixtures containing 0.1 percent per weight or more of PeBDE. Products containing more than 0.25 % PeBDE are classified as hazardous waste when they are discarded. In Norway recycling and reuse of PeBDE and materials with PeBDE are not allowed.
- Canada: no production of PeBDE, regulations on use are in preparation. PeBDE was proposed to be added to the Virtual Elimination List in Canada in 2004.
- China: use of PeBDE in EE products was banned from 1. March 2007. There should currently be no production of C-PeBDE in China.

2. Production, use and release of PeBDE
2.1 Levels and trends of production and use

Overall demand and production

Based on the last available market information on C-PeBDE from Bromine Science and Environmental Forum (BSEF), total global demand has decreased from 8,500 tons in 1999 to 7,500 tons in 2001. The estimated cumulative use of C-PeBDE since 1970 was 100,000 t in 2001 according to BSEF.

Table 2.1 Total global demand of C-PeBDE by region in 2001. Metric tons and percent.

<table>
<thead>
<tr>
<th></th>
<th>America</th>
<th>Europe</th>
<th>Asia</th>
<th>Rest of the world</th>
<th>Total</th>
<th>Percent of total world usage of BFRs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penta-mix PBDE formulation</td>
<td>7,100</td>
<td>150</td>
<td>150</td>
<td>100</td>
<td>7,500</td>
<td>4 %</td>
</tr>
</tbody>
</table>

Source: BSEF (2001)

The US EPA (2007) estimates that US production and import were between 4,500 and 23,000 tons in 2002. This indicates a somewhat larger US market in 2001/2002 than the number in Table 2.1. However, since there should be no current production of C-PeBDE in Europe, Japan, Canada and US, remaining production would be located in other parts of the world. According to BSEF there are some indications that since the late 1990s China may have produced C-PeBDE for the US market as well.

C-PeBDE has been produced in Israel, Japan, US and the EU (Peltola et al., 2001 and TNO-report 2005). A patent on a technical mixture containing PeBDE was issued for China in 1999. As produced in China, the technical mixture contained a different ratio of its constituents (that is, different proportions of congeners) than C-PeBDE produced in Europe and the US.

The major producer of BFR in Israel, the Dead Sea Bromine Group, declares in a public statement on their web site that their products do not contain C-PeBDE. This is to comply with the ban in EU, which is an important market for the company.

The last producer of C-PeBDE in the US, the Great Lakes Chemical Corporation (now Chemtura Corporation), voluntarily ended its production in 2004. In 2001 alone, almost 70,000 metric tons of polybrominated diphenyl ethers (PBDEs) were produced globally, almost half of which were used in products sold in the US and Canada. Before the phase-out in US the majority of PeBDE formulation produced globally were used in North America (>97%). At the end of 2004, approximately 7.5% of the more than 2.1 billion pounds of flexible polyurethane foam produced each year in the US contained the commercial PeBDE formulation (Washington State 2006).
Production of C-PeBDE in the former EU (15) ceased in 1997 (EU 2000). Usage in the EU (15) declined during the second half of the 1990s and was estimated to be 300 metric tonnes in 2000 (used solely for PUR production) (EU 2000). The use of PeBDE was banned in the EU (25) in 2004. Use of PBDE in electrical and electronic appliances was phased out from 1 July 2006.

Results from a survey in Canada in 2000 indicated that approximately 1,300 tonnes of commercial products containing PBDE were imported into Canada. Based on quantities reported, C-PeBDE was imported in the greatest volume. PeBDE was proposed to be added to the Virtual Elimination List in Canada in 2004.

No information was found for Eastern European countries outside EU or for most countries in the Asia-Pacific region. No information was available from countries in Africa or Latin America.

2.2 Use of C-PeBDE

Use of C-PeBDE as a flame retardant

Since 1999 the most common use (95-98%) of PeBDE has been in flexible polyurethane (PUR) foam. This foam contains between 10 and 18% of the commercial C-PeBDE formulation. Flame retarded, flexible PUR foam is used mainly for furniture, including upholstery in domestic furnishing, and in the automotive and aviation industries.

Other uses are in rigid polyurethane elastomers in instrument casings, in epoxy resins and phenol resins in electric and electronic appliances, and construction materials. C-PeBDE can also be used in small amounts in textiles, paints, lacquers, in rubber goods (conveyor belt, coating and floor panels) and in oil drilling fluids. Levels range from 5-30% by weight. Up to the early 1990s C-PeBDE was used in printed circuit boards, which was usual for FR2 laminates (phenol resins) in Asia. FR2 laminates are used in household electronics (television, radio, and video), vehicle electronics, and white goods (washing machines, kitchen appliances, etc.).

According to information from the BSEF member companies their sales for use of C-PeBDE in hydraulic fluid (in the form of a mixture) in petroleum drilling and mining stopped 10-20 years ago.

Australia has reported use of C-PeBDE in manufacture of polyurethane foams for refrigerators, packaging and for use as potting agents, and in epoxy resin formulations supplied into aerospace market, laminating systems and adhesive systems. The US has reported use of C-PeBDE in the production of components for military airplanes. EU had an exemption for aircraft emergency evacuation systems that expired march of 2006.

A recent study, has indicated use of C-PeBDE in older computers (Betts 2006).

2.3 Global future demand for flame retardants

According to a market analyst consultant company, global demand for flame retardants is expected to grow by 4.4 percent per year to 2.1 million metric tons in 2009, valued at USD 4.3 billion (Fredonia Group, 2005). Growth will largely be driven by demand in developing countries in Asia (China in particular), Latin America and Eastern Europe. The growth in demand is expected for most flame retardants. Globally, growth is expected to be largest for bromine compounds, mainly due to high growth rates in China. Demand for use in electrical and electronic applications is expected to grow fastest. Higher value products will continue to make inroads as substitutes for less environmentally friendly compounds, especially in Western Europe, and as chlorine compounds begin to be replaced in China by bromine- and phosphate-based and other flame retardants (Fredonia Group, 2005).

As electronic circuits become smaller and more densely packed, and their plastic components are subjected to higher temperatures, the need for flame retardants increases. Construction
markets are expected to be the second fastest growing market for flame retardants globally. An exception is China, where the second fastest growth will be from motor vehicles followed by textiles, both rapidly growing industries in that country. Plastics will continue to replace other materials such as metals and glass in a wide range of products in order to lower cost and weight and to improve design and production flexibility. Their usage is widespread and growing in transportation, building products and electrical and electronic products. Plastics must be made flame retardant for many applications. As a result, 75% of all flame retardants are used in plastics (Freedonia Group 2005).

Environmental restrictions vary by region. In Western Europe, Japan and to a lesser extent North America, such restrictions will especially limit growth of chlorinated compounds which might be considered as in-kind replacements for PBDEs. The ban on some brominated flame retardants in Western Europe is not expected to spread substantially to other regions (Freedonia Group 2005), but it drives the development of electrical and electronic equipment without the banned substances for sale on the world market. Dozens of Asian, European, and US companies announced in 2005 that they have developed or are developing electrical and electronic equipment that does not contain C-PeBDE. In Asia more than 90% of electronic manufacturers already make products compliant with the EU ban on PeBDE. Officials from electronics companies and industry consultants consider that most electric and electronic equipment sold on the world market were in compliance with the ban in EU in 2005, due to the difficulties of keeping product streams separate ((Environmental International reporter 2006).

2.4 Emissions from production of C-PeBDE and products using C-PeBDE as input

PeBDE is synthesized from diphenyl ether by brominating it with bromine in the presence of a powdered iron/Friedel-Crafts catalyst. The producers of C-PeBDE have reported that the major routes of PeBDE release to the environment during production are filter waste and rejected material, both of which are disposed of in landfills. Waste water releases of PeBDE may also occur from spent scrubber solutions (RPA, 2000). The emissions to air from production of PeBDE is assumed to be none or negligible (RPA 2000, Van der Gon et al. 2005).

According to RPA (2000) emissions from polyurethane production are assumed to occur prior to the foaming process when handling the additives (discharges to water) and during the curing (emissions to air). In the phase prior to foaming, releases to waste water are estimated at 0.1 kg/tonne handled PeBDE. Releases to air may also occur during the curing phase of the foam, when the temperature of the foam stays elevated for many hours depending on the production block size. Emissions to air at this phase are estimated to be 1 kg/tonne PeBDE, but it is assumed that some of the volatilized PeBDE condenses in the production room ending up in the waste water. RPA (2000) concludes that 0.6 kg of PeBDE is released into waste water and 0.5 kg into air for each ton of C-PeBDE used in PUR production. Global annual production of PUR-foam in 2000 (containing 10-18% of C-PeBDE), has been estimated to be 150,000 tons (Alaee et al. 2003). Global annual releases of PeBDE from PUR-foam production are estimated in table 2.2.

Table 2.2 Global production and use of C-PeBDE in PUR-foam production and estimation of associated releases in 2000. PUR-foam containing 10-18% of C-PeBDE.

<table>
<thead>
<tr>
<th>Production of PUR-foam</th>
<th>Use of C-PeBDE to PUR-foam production</th>
<th>Releases of PeBDE into waste water from PUR-foam production</th>
<th>Emissions of PeBDE into air from PUR-foam production</th>
</tr>
</thead>
<tbody>
<tr>
<td>150,000 tons/year</td>
<td>15,000 – 27,000 tons/year</td>
<td>9,000 - 16,200 kg/year</td>
<td>7,500-13,500 kg/year</td>
</tr>
</tbody>
</table>

Source: Document developed under the Task Force on POPs; Exploration of management options for PeBDE (http://www.unece.org/env/popsxg/5thmeeting.htm)

There is limited information on emissions of PeBDE from manufacture of products other than PUR-foam, but emissions to air are assumed to be negligible. Major releases will be to waste water or solid waste. There is no information on the use of C-PeBDE in manufacturing.
processes from Eastern European countries outside the EU. Modeling indicates that emissions during manufacture of products containing C-PeBDE are minor in comparison to the consumption.

2.5 Emissions from use of C-PeBDE-containing products

TNO (2005) concludes that the major releases of PeBDE to air stem from products and equipment which contain the substance as flame retardant.

**Indoor equipment**

PeBDE is a component in indoor dust, and several studies have examined the extent of human exposure (UNEP 2006). Indoor dust is considered to be one of the main sources of human exposure (UNEP 2006).

Several studies have detected PeBDE in indoor air and dust stemming from products like textiles, furniture and electronic devices (Shoeib et al., 2004, Stapleton et al. 2005, and Wilford et al., 2005). Controlled chamber experiments have detected volatilization of PeBDE from PUR-foam, used in furniture (Wilford et al. 2005). Experimental data shows that TeBDE and PeBDE are released from electronic appliances, such as TV sets and computer monitors (Danish EPA, 1999). This is supported by a recent study, indicating use of C-PeBDE in older computers (Betts 2006).

PeBDE has been identified as an additive flame retardant in textiles in different national substance flow analyses in the ECE-region (Danish EPA 1999, Norwegian EPA 2003). In a Norwegian substance flow analysis manufacturers of furniture textile have stated that the textile contains 0.45 % PeBDE. Textiles used in the public sector, the transport sector and business sector in many countries have stringent rules for flammability.

C-PeBDE is used solely as an additive chemical. Although the vapor pressures of its constituents are low, some fraction can volatilize from the products during their whole life-cycle (RPA, 2000). The release of BFRs from products in use will depend on two factors:

- h) Release of BFRs from the surface
- i) Migration of BFRs in the polymer

When released from products, the BFRs are likely to adsorb to particles. The particles (dust) may adhere to surfaces within the appliances, on other surfaces in the indoor environment or be spread to the outdoor environment. When the appliances are dismantled for reprocessing some of the dust will be released to the workplace air. Compared to an office environment the exposure from dismantling of the appliances may be several orders of magnitude higher (Danish EPA, 1999).

In RPA (2000) 3.9 % PeBDE of the amount of C-PeBDE present in products was estimated to be released annually through volatilization during their anticipated lifetime of 10 years, using a worst-case scenario. Global annual releases of PeBDE from new use of PUR-foam in articles are estimated in Table 2.3. Similar detailed information for other uses is not found in the literature.

**Table 2.3 Global production and use of PeBDE in PUR-foam in products and estimated releases during their service life in 2000 (Metric ton/year).**

<table>
<thead>
<tr>
<th>Production of PUR-foam</th>
<th>Content of C-PeBDE in PUR-foam</th>
<th>Releases of PeBDE during the lifetime of the products</th>
</tr>
</thead>
<tbody>
<tr>
<td>150,000</td>
<td>15,000 – 27,000</td>
<td>585 – 1,053</td>
</tr>
</tbody>
</table>

Source: Document developed under the Task Force on POPs; Exploration of management options for PeBDE (http://www.unece.org/env/popsxg/5thmeeting.htm)

**Outdoor equipment**

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While material vaporized from outdoor equipment will be widely dispersed at low concentration in the air, particles of polymer (foam) products which contain PeBDE can be released to the environment from C-PeBDE-containing outdoor equipment. These particles are primarily released to the urban/industrial soil compartment (75%), but may also be released to surface waters (24.9%) or air (0.1%). The release can occur during the lifetime of the product (due to weathering, wear and tear etc.) and at waste disposal particularly when products are dismantled or subject to other mechanical processes. This can apply to the following outdoor applications of PVC (RPA, 2000):

j) Car undercoating,
k) Roofing material,
l) Coil coating,
m) Fabric coating,
n) Cables and wires, and profiles,
o) Shoe soles.

The emission factors for these releases are in RPA (2000) estimated to 2-10% over the lifetime of the product, with the higher factor being applied to products subject to high wear rates (such as car undercoating and shoe soles), and 2% during disposal operations. The releases in the EU region were in 2000 estimated to be 15.86 tonnes PeBDE per year to industrial soil, 5.26 tonnes per year to surface water and 0.021 tonnes per year to air. No estimates of global releases are found in the literature.

According to information obtained from the bromine industry historic uses of hydraulic fluid (in the form of a mixture) in petroleum drilling and mining can have resulted in excessive amounts released to the environment. No estimates of those releases are found in the literature.

2.6 Emissions from waste containing C-PeBDE

Waste can be generated from production of C-PeBDE, manufacturing processes of C-PeBDE-containing products and when C-PeBDE-containing products end up as waste. There is limited information in the literature concerning releases from C-PeBDE-containing waste.

Waste generated from production of C-PeBDE

In the production of C-PeBDE producers have stated that the major sources of waste release were filter waste and reject material. Waste water releases of PeBDE may also occur from spent scrubber solutions (RPA, 2000). C-PeBDE-containing waste was put on landfill (RPA, 2000). In the US this waste is disposed of in landfills that are permitted to handle hazardous chemical waste. In the EU, wastes containing more than 0.25% PeBDE are classified and treated as hazardous waste. Waste from production of C-PeBDE is considered negligible.

Waste generated from manufacturing processes of products containing C-PeBDE

Blocks of PUR foam generally have to be cut into the required size/shape of the final product. This operation usually occurs after the blocks have cured and cooled. For some applications PUR foam can be produced in a mould of the desired shape, and then cutting is not required.

The flame retardant lost during these processes will stay in the scrap foam. Foam scrap is often recycled into carpet underlay (rebond), particularly in the United States (EU has been an exporter of scrap foam (around 40,000 tonnes/year) to the United States for this use (RPA 2000)). Other uses for scrap foam such as regrinding and subsequent use as filler in a variety of applications (e.g. car seats, addition to virgin polyol in the manufacture of slabstock foam) have been reported. It is also possible that scrap foam is deposited on landfill or incinerated in many countries.

During the production of printed circuit boards a substantial part of the laminate is cut off and ends up in solid waste. C-PeBDE is no longer used for production of printed circuit boards in most producer countries. The information on the use of C-PeBDE in other manufacturing processes of EE-appliances is too limited to enable the drawing of conclusions about waste generation, but it is known that most of the waste ends up as solid waste. This waste is put
into landfills or incinerated, as is waste generated from production of building materials, textiles and furniture.

**When products containing C-PeBDE become waste**

In the EU, wastes containing PeBDE are covered by regulations governing plastics containing BFRs. These plastics must be separated from EE-appliances prior to recovery and recycling by December 2006. After extrusion of metals the plastic fraction is disposed of or burned in municipal waste incinerators.

Vehicle hulks are stored outdoors and then dismantled in shredder plants. In some countries regulations require that components containing hazardous substances are separated before shredding. This applies, obviously, for smaller components that are easy to dismantle. For most plastic and textile components this is not done, and flame retardants in those components end up in the waste fraction from the shredder plant that is put into landfills or sometimes incinerated.

Other products containing PeBDE is also put on landfills or incinerated when they end up as waste.

**Releases from landfills and incineration**

Polymer (foam) particles containing PeBDE could leach from landfills into soil, water or groundwater. However, it is not currently possible to assess the significance of this type of process. The amount of PeBDE put on landfill or incinerated in the EU is estimated to be 1,036 tonnes/year (RPA, 2000).

Given the physic-chemical properties of the substance (low water solubility, high octanol-water partition coefficient) it is considered very unlikely that significant amounts of PeBDE will leach from landfills as the substance would be expected to adsorb strongly onto soils (RPA, 2000). However, Norwegian screening studies have measured concentrations of PeBDE of concern in the leaching water from landfills (Fjeld et al. 2003 and 2004).

At the operating temperatures of municipal waste incinerators almost all flame retardants will be destroyed. However, based on experience with other organic compounds, trace amounts could pass through the combustion chamber (Danish EPA, 1999). Studies of municipal waste incineration facilities have detected levels of PeBDE in both gaseous and particulate fractions in the air in the vicinity of the facility. The levels were above background levels of PeBDE (Agrell et al. 2004, Law 2005, ter Shure et al. 2004).

Potentially toxic products such as brominated dibenzo-p-dioxins and dibenzofurans, may be released during incineration of waste containing C-PeBDE (Danish EPA, 1999), just as their chlorinated analogues may be produced during combustion of wastes containing chlorinated materials. While, the technologies used in modern well-run waste incinerators to manage chlorinated dioxins and dibenzofurans emissions are believed to be adequate for controlling emissions of brominated and mixed bromo/chloro species as well (OECD 2001), these substances could be released during open burning of C-PeBDE-containing materials.

**2.7 Emissions from recycling and dismantling activities**

**Electrical and Electronic (EE) waste recycling plants**

The analyses of dismantled FR2 printed circuit boards in electrical scrap show that about 35% of the PBDE used consists of PeBDE. Based on market information it has been assumed that 25% of FR2 laminates in older appliances were treated with the commercial mixture of PeBDE (Swiss agency 2002).

Prevedouros et al. (2004) estimated production, consumption, and atmospheric emissions of PeBDE in Europe between 1970 and 2000 based on literature data. According to their study, the flow of PeBDE in disposed EE-appliances is estimated to be in the range of 17-60 metric tons per year within the time period 2000-2005. An experimental Swiss study on substance flow in a modern recycling plant showed a much higher flow of PeBDE than expected from the literature study. The study revealed that the majority of producers and importers have
insufficient information about the content of chemical compounds in the products they market (Swiss agency, 2002).

In Morf et al. (2005), the average concentration in EE-appliances was estimated to 34 mg/kg PeBDE. The highest amount was found in the plastic fraction of EE-appliances (125 mg/kg). If a recycling process is not equipped with an efficient air pollution control device as was used in the modern plant on which the experimental study was conducted, a significant flow of dust-borne PeBDEs may be transferred into the environment. In plants with off-gas filtering, around 65% of the PeBDE will be collected (Morf et al. 2005).

Studies of the working conditions in recycling plants have detected levels of PeBDE in the indoor air, and indicate that PeBDE also can be spread as diffuse emissions from recycling plants. The authors of a national substance flow analysis carried out for Switzerland, covering the whole life cycle of penta-, octa-, and decabDE as well as TBBPA, concluded that EE waste equipment accounts for the largest flow of the investigated BFRs compared to other waste fractions, such as, for example, automotive shredder residues and construction waste (Swiss agency, 2002).

**Dismantling of vehicles**

In a substance flow analysis of BFR in Switzerland, the concentrations of PeBDE in plastics were estimated to be 0.044 g/kg in road vehicles produced in 1998 and 0.089 g/kg in road vehicles produced in 1980. These concentrations refer to the amount of PeBDE in the total weight of plastics in cars exclusive of EE plastic components. Up to the end of the 1980s, 100% of all unsaturated polyester (UP) resins was treated with BFR, primarily Decabromodiphenyl ether (DeBDE) but also PeBDE and tetrabromobisphenol-A (TBBPA).

The first step in the recycling of vehicles is fragmentation in a shredder, where the metals are separated from other materials. Plastics, rubber, paper, wood, dirt, etc. end up in several fractions of shredder residues. The plastic parts mainly end up in a fraction called "fluff".

It could be assumed that there are diffuse emissions from shredder plants, but these have not been estimated. The conditions for emissions can be assumed to be similar as for recycling plants of EE-appliances. For plants not equipped with an efficient air pollution control device a significant flow of dust-borne PBDEs may be transferred into the environment. In plants with off-gas filtering a large portion of PeBDE will end up in the collected fraction of the gas.

**Dismantling of buildings and other constructions**

In Switzerland, 5% of the PUR insulating foams produced in 1990 was used in the building industry and contained 220 g/kg PeBDE (Swiss agency 2000).

Thermoplastic sheeting used to be treated with BFRs at concentrations between 1.3 and 5% by weight. According to a study for Denmark, 10 – 20 % of the plastic sheeting used in bridges and underground structures are possibly treated with flame retardants (Danish EPA 1999). There are indications of use of PeBDE in PVC plastic sheetings. There is no detailed information on the use of BFR. In the substance flow analysis made in Switzerland, 5% of products produced in 1990 with PVC plastic sheeting were assumed to contain PeBDE. The amount of PeBDE was estimated to be 49 g/kg PVC sheeting. Emissions of dust-borne PeBDE can be assumed to be released during dismantling activities. The information is too limited to quantify those emissions.

### 3. Summary information relevant to the risk management evaluation

#### 3.1 Identification of possible control measures

There are in principle several control measures that could be implemented to reduce the use of C-PeBDE and/or reduce the environmental impacts associated with the use of the substance. The control measures are:

- **Voluntary commitments by industry** to reduce the environmental impacts associated with the use of C-PeBDE by reducing their production and/or use. Commitments have
been successfully implemented in some countries, where industry voluntarily has phased out the production of PeBDE. It is likely that this measure works well when the actions needed are cheap, and/or it is obvious to the industry that it is facing a long term phase-out of the substance.

- **A ban/restriction on the use of C-PeBDE**, either as a whole or on a sectoral basis through both unilateral and multilateral fora. This could either be an outright ban on use in all sectors, in some products or restrictions on the concentrations in products. If agreed on by a sufficient number of countries this could be an effective measure if properly enforced. Some countries have already taken such actions.

- **Eco-labeling schemes**, which either requires that products containing PeBDE are labeled or more likely that products containing less harmful substances are given an eco-friendly label. This would inform users about the most environmental-friendly products, enabling them to choose these products if they want.

- **Economic instruments** like taxes on sales of C-PeBDE or taxes on sales of products containing the mixture. Alternatively trading of permits to buy and use C-PeBDE could be implemented. Both these instruments would give users economic incentives to reduce use of the substance.

- **A deposit-refund system for C-PeBDE**, implying that a deposit (tax) is paid by the buyer of a product containing C-PeBDE, which is wholly or partly refunded when the buyer returns the product to a certified waste treatment facility when it should be no longer used. This could be an effective measure to ensure proper waste handling and reduce release to the environment when a product ends up as waste, but would require identification of those wastes containing C-PeBDE. However, since national and regional bans already in place will reduce the amount of C-PeBDE entering the market, such a tax is unlikely to generate funds on the scale required for dealing with legacy problems.

- **Various control measures at the production or waste handling facilities** ensuring safe work environment and good manufacturing practice, end-of-pipe controls reducing emissions to the environment, regulations on waste handling of products etc. These measures could be applied at the production plants for C-PeBDE, at the plants using C-PeBDE as input in their production and at the waste handling facilities. If properly designed and enforced this could be an effective tool to reduce releases from the sources in question.

The properties of the different control measures are discussed below.

### 3.2 Efficacy and efficiency of possible control measures in meeting risk reduction

**General considerations**

Since the components of C-PeBDE of concern are released to the broader as well as the work environment during the manufacturing process of the mixture, when C-PeBDE is blended into products, during the lifetime use of the product and when the product ends up as waste, several control measures might have to be applied to reduce release of the components of C-PeBDE to the environment.

The choice of control measure for the remaining use of C-PeBDE and emissions of its components must take into account that most developed countries have phased out production of C-PeBDE, and that the ultimate long term goal is to phase out the global production and use of C-PeBDE and emissions of its components. Further risk reduction options should be examined against the following criteria (RPA, 2000):

- **Effectiveness**: the measure must be targeted at the significant hazardous effects and routes of exposure identified by the risk assessment. The measure must be capable of reducing the risks that need to be limited within and over a reasonable period of time.

- **Practicality**: the measure should be implementable, enforceable and as simple as possible to manage. Priority should be given to commonly used measures that could be carried out within the existing infrastructure.

- **Economic impact**: the impact of the measure on producers, processors, users and other parties should be as low as possible.

- **Monitorability**: monitoring should be possible to allow the success of risk reduction to be assessed.
Voluntary commitments and eco-labeling schemes can yield reductions in use of C-PeBDE and emissions of its components with rather low costs for producers at an early stage of a phase out process. They can stimulate reduced use of C-PeBDE in areas where costs are low, and voluntary schemes have been successfully used in several countries to reduce use of C-PeBDE. These measures could eventually also contribute to reductions in countries where so far few or no actions are taken to reduce the use of C-PeBDE. However, the global effectiveness of voluntary measures depends on the culture of the particular society, which is often influenced by experience, and these factors will be especially important when considering the adoption of measures in developing countries. The willingness among the affected industries to take necessary action to limit global use of C-PeBDE will also be important. Free riders tend to be a problem in many of the voluntary schemes that have been tried to curb emissions of various substances in the developed countries. Since the ultimate goal is to phase out C-PeBDE, other measures seem more likely to be effective at this stage.

Economic instruments could ultimately lead to a phase out of the use of C-PeBDE if the tax or permit price are high enough to make switching to alternative solutions profitable. Economic instruments have not proved effective in most developed countries when a total phase-out over short time is required, and have not been used for substances with the high level of concern of POPs. The experiences with their practical use are limited in most developing countries. It would therefore complicate things to introduce this measure. Since use of economic instruments is not at present allowed under the Stockholm Convention, further consideration should not be given to such measures.

Standards aiming at reducing the concentrations of C-PeBDE in products or for instance using additional additives would not be very effective, since concentrations have already been minimized to the degree possible to minimize costs (RPA, 2000). Besides, this measure would only target some parts of the value chain for C-PeBDE, leaving out important sources for release to the outer and work environment. However, standards could be used to ensure environmentally benign waste handling.

It seems that in the current situation a global ban on production and use of C-PeBDE by listing the substance under the Stockholm Convention would be the most appropriate measure. Since suitable, more environmentally benign alternatives exist for all use of C-PeBDE (see below), a ban should cover all sectors. Eventually, some very special uses of C-PeBDE where alternatives are not efficient enough and/or very costly could be exempted for a time-limited transition period. However, it must be considered whether a ban could lead to substitution of C-PeBDE with other environmentally harmful substances and if this should warrant any action. A ban would eliminate emissions from the manufacturing of C-PeBDE, and also eliminate release from the production and use of C-PeBDE in new products. It would ensure that there would be no new production and use of C-PeBDE in the future. But it would not affect the emissions from C-PeBDE in products already in use. For most products the use of C-PeBDE as a flame retardant has already been replaced by other more environmentally friendly flame retardants, with some producers taking voluntary action ahead of regulation. Substitution has largely occurred in the developed countries. One country has reported need for an exemption for use of C-PeBDE in military aeroplanes, due to the lack of alternatives that meet the special demands for fire safety. Some time could be allowed for testing and technical modifications to develop alternatives and to be able to meet the special demands on fire safety in products with special needs. This would require a phase out plan for the use of these products, which would be warranted under Annex B of the Stockholm Convention, although listing in Annex A with time-limited exemptions would serve the same end.

**Waste handling**

A ban on production and use of C-PeBDE would not in itself affect emissions of its components of concern from waste handling, where they can present a technical and legacy problem. However, listing a substance under the Stockholm Convention implies a ban on recycling and reuse of stockpiles of C-PeBDE itself. Article 6 in the Convention requires that wastes and stockpiles are handled in a safe, efficient and environmentally sound manner, so that the content is destroyed or irreversibly transformed, taking into account international rules, standards and guidelines. The article also bans disposal operations that lead to recovery, recycling, reclamation, direct use or alternative use of POPs material.
Article 6 clearly bans recycling of products and articles containing C-PeBDE if it results in new use of C-PeBDE as a constituent of new articles. Recycling and reuse of plastic material containing BFRs has been practiced but this would no longer be allowed if C-PeBDE were present in the new material at more than trace levels. This would be especially important for recycling of electronic articles containing C-PeBDE and for shredder plants with more diverse inputs, and so the consequences for this type of industry may need to be considered further.

A special challenge could be to separate C-PeBDE-containing articles from those without the substance, since most articles are not labeled telling what they contain. However, there is information about articles that have contained C-PeBDE in the past and which articles it is used in today, like electronic articles, textiles and isolation material and casing materials. National authorities would have to make surveys to get more detailed national information about C-PeBDE content in different articles becoming waste.

Also, targets for phase out of the use of existing products containing C-PeBDE and the collection of these could be considered according to Annex B of the Convention.

Since there are substantial stocks of products containing C-PeBDE in use, national authorities could consider some additional measures to limit releases. These measures could range from establishing collection points where people can deliver their used products to more actively promoting and encouraging people to deliver their waste products. A deposit-refund system does not seem appropriate since sales of new products containing C-PeBDE would no longer be allowed and their presence has become a legacy problem. However, paying people a fee to deliver their products would be an option, although a source of funding for such an operation is not obvious.

A special challenge would be to ensure proper handling of C-PeBDE-containing waste material/articles in developing countries. Since these countries have limited experience in handling this kind of waste, they would need practical help and information as well as financial help to ensure environmentally benign handling of this waste. The assistance could include how to dismantle C-PeBDE-containing articles, treat the various parts and the methods of environmentally sound treatment of the final C-PeBDE.

3.3 Information on alternatives (products and processes)

There are three ways to provide flame retardancy in products without using BFRs:
- substitute them with another flame retardant in a given material (i.e. plastic or foam);
- substitute them with another flame retardant in a different type of plastic or foam; or
- redesign the product so that there is no need for using flame retardants.

With the phasing out of C-PeBDE in important markets, manufacturers are actively identifying alternatives. Some companies, such as IKEA, have already phased out all C-PeBDEs globally. Another factor encouraging the development of alternatives is the fact that many governments and large corporations have developed green procurement guidelines that prohibit the use of PBDEs in electronic products.

The alternative flame retardants for C-PeBDE listed in Table 3.1 are mostly gathered from companies and all the chemicals listed are already in use. The human health or environmental impacts of these alternatives have not been investigated by the authors. For example, hexabromocyclododecane listed in Table 3.1 as an alternative for C-PeBDE in coatings and adhesives is not a preferable alternative. This compound already causes concern because of its chemical properties in several countries and regions. RPA (2000) suggests that only tetrabromobenzate (TBBE) and chlorinated alkyl phosphate esters, tri (2-chloroisopropyl) phosphate (TCPP) in particular, followed by phosphate esters, are relevant chemical alternatives to PeBDE. However, since that time other alternatives may have been developed and commercialized and should also be considered.

<table>
<thead>
<tr>
<th>Substrate (material, Products in which)</th>
<th>Alternative flame retardants for C-PeBDE by substrate.</th>
</tr>
</thead>
</table>

Table 3.1 Alternative flame retardants for C-PeBDE by substrate.
<table>
<thead>
<tr>
<th>Material Type</th>
<th>Substrate Uses</th>
<th>Retardants in Commercial Materials</th>
<th>Non-flammable or Containing Halogen-free Flame Retardants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epoxy</td>
<td>• Printed circuit boards • Electronic component encapsulation • Technical laminates</td>
<td>• Reactive nitrogen and phosphorus constituents • Ammonium polyphosphate • Aluminium trihydroxide</td>
<td>• Polyphenylene sulphide</td>
</tr>
<tr>
<td>Unsaturated polyester</td>
<td>• Technical laminates • Plastic parts in Transportation</td>
<td>• Ammonium polyphosphate • Aluminium trihydroxide • Dibromostyrene • Tetrabromophthalic anhydride-based diol • Tetrabromophthalic anhydride • Bis (tribromophenoxy) ethane</td>
<td>None identified</td>
</tr>
<tr>
<td>Rigid polyurethane foam</td>
<td>• Insulation of cold storage plants/freezing rooms, pipes, etc.</td>
<td>• Ammonium polyphosphate • Red phosphorus • Tetrabromophthalate diol • Tetrabromophthalic anhydride-based diol • Bisphosphate</td>
<td>• Some applications: mineral wool or other technical solutions</td>
</tr>
<tr>
<td>Flexible polyurethane foam</td>
<td>• Furniture • Components in Transportation</td>
<td>• Ammonium polyphosphate • Melamine • Reactive phosphorus polyols • Tetrabromophthalic anhydride derivative • Bromo-alkyl phosphates • Reofos NHP (halogen-free phosphorus flame retardant) • Bisphosphate</td>
<td>None identified</td>
</tr>
<tr>
<td>Laminates</td>
<td></td>
<td>• Triaryl phosphate isopropylated</td>
<td>None identified</td>
</tr>
<tr>
<td>Adhesives</td>
<td></td>
<td>• Tetrabromophthalate diol • Tetrabromophthalic anhydride based diol • Hexabromocyclododecane • Reomol® TOP • Bis (tribromophenoxy) ethane</td>
<td>None identified</td>
</tr>
<tr>
<td>Coatings</td>
<td></td>
<td>• Tetrabromophthalate Diol • Tetrabromophthalic anhydride based diol • Hexabromocyclododecane • Triaryl phosphate • Bis (tribromophenoxy) ethane</td>
<td>None identified</td>
</tr>
</tbody>
</table>

Alternatives to C-PeBDE in PUR foam
The US EPA Design for the Environment completed an assessment of alternatives to PeBDE in PUR which was released in September 2005 (US EPA, 2005). The agency has established a Furniture Flame Retardancy Partnership with a broad set of stakeholders to assess environmentally safer chemical alternatives to PeBDE and to investigate other technologies for improving furniture fire safety. Leading US flame-retardant chemical manufacturers identified 14 chemical formulations that are viable substitutes for PeBDE in large-scale production of low-density flexible polyurethane foam, see Table 3.2. The US EPA assessed the hazards, potential exposures and tendency to bioaccumulate and to persist in the environment for the chemicals in each formulation. The alternatives are drop-in replacement chemicals for PeBDE. Existing storage and transfer equipment as well as foam production equipment can be used without significant modification. Alternatives compatible with existing process equipment at foam manufacturing facilities are the most cost effective, because they do not require the plants to modify their processes or purchase new equipment. The assessment looked into hazardous effects, persistence and bioaccumulation. The report did not assess the environmental impact of the use of those alternatives to the use of PeBDE, and was mainly focused on comparison of the impact of use in the production process of PUR-foam. The levels of bioaccumulation and persistence reported for the identified chemical formulations were low or moderate, with an exception for organic phosphate esters, which have high persistence. The assessment assigned high aquatic toxicity concern to most of the alternatives and low or moderate concern for human health effects.

Most of the commercial products were mixtures, and some information about constituents is included in Table 3.2.

Table 3.2 Alternative Flame-Retardant Chemical Formulations

<table>
<thead>
<tr>
<th>Albemarle Corporation</th>
<th>Ameribrom, Inc. (ICL Industrial Products)</th>
<th>Great Lakes Chemical Corporation</th>
<th>Supresta (Akzo Nobel)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAYTEX® RX-8500</td>
<td>FR 513 Proprietary reactive brominated flame retardant, proprietary aryl phosphate, triphenyl phosphate CAS 115-86-6</td>
<td>Firemaster® 550 Proprietary halogenated aryl esters, proprietary triaryl phosphate isopropylated, triphenyl phosphate</td>
<td>Fyro® FR-2 Tris(1,3-dichloro-2-propyl) phosphate CAS 13674-87-8</td>
</tr>
<tr>
<td>SAYTEX® RZ-243</td>
<td>Firemaster® 552 Proprietary halogenated aryl esters, proprietary triaryl phosphate isopropylated, triphenyl phosphate</td>
<td>AB053 Tris(1,3-dichloro-2-propyl) phosphate</td>
<td></td>
</tr>
<tr>
<td>ANTI BLAZE® 195</td>
<td>ANTIBLAZE® V-500 Proprietary chloroalkyl phosphate and triphenyl phosphate</td>
<td>AC003 Proprietary organic phosphate ester, triphenyl phosphate</td>
<td></td>
</tr>
<tr>
<td>ANTI BLAZE® 205</td>
<td>ANTIBLAZE® 180 Tris(1,3-dichloro-propyl) phosphate CAS 13674-87-8</td>
<td>AC073 Proprietary aryl phosphates, triphenyl phosphate</td>
<td></td>
</tr>
<tr>
<td>ANTI BLAZE® V-500</td>
<td>ANTIBLAZE® 180 Tris(1,3-dichloro-propyl) phosphate CAS 13674-87-8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Chemicals other than these fourteen formulations are currently used for other types of foam and in niche markets for low-density polyurethane foam. The chemicals are used to flame retard high-density, flexible polyurethane foam. Chemical companies and foam manufacturing facilities have experimented with their use in low-density flexible foams with moderate success. Generally the use of these chemicals either results in scorching of the foam (a discoloration and indication of localized overheating during production) or a negative effect on the physical properties of foam. Also, many formulations of these chemicals are available only as solids, making them less desirable as drop in substitutes for PeBDE. Since the commercial mixture PeBDE is liquid, addition of a solid flame retardant may require changes such as additional mixing steps and alteration of the process times. In some cases, these changes can have significant effects on foam quality or cost-effectiveness of production.

Three of the most commonly used chemicals that various reports have suggested may be viable alternatives to PeBDE are melamine, tris(1,3-dichloro-2-propyl) phosphate (TDCPP) (or TCPP) and ammonium polyphosphate (APP). There are numerous international manufacturers of melamine and its derivatives, which are non-halogenated flame retardants, typically supplied as a crystalline powders. Flame retardants based on melamine are currently used in flexible polyurethane foams, intumescent coatings (those which swell on heating and thus provide some measure of flame retardancy), polyamides and thermoplastic polyurethanes. They are used effectively in Europe in high-density flexible polyurethane foams but require 30 to 40 percent melamine per weight of the polyol. However, melamine and its derivatives display several toxic effects. These include changed electrolyte composition of urine, teratogenic effects in fertilized rainbow trout eggs, and reproductive effects in snails and houseflies (Daugherty 1982). Melamine also caused chronic injury to the male rat bladder due to stones formed during exposure to the chemical which correlated strongly with carcinoma (Danish EPA 1999).

TDCPP is a chlorinated phosphate ester that is often used in polyurethane foam formulations. It is used in high-density foam and has been used in low-density foams when light scorching (discoloration) is not a primary concern. TDCPP has been identified as having moderate hazard concerns for persistence, acute and chronic ecotoxicity, reproductive and developmental effects, genotoxicity and carcinogenicity (US EPA 2005).

APP is an additive flame retardant containing nitrogen and phosphorus, typically supplied in a crystalline form. It is currently used to provide flame retardancy in flexible and rigid polyurethane foams, as well as in intumescent laminations, molding resins, sealants and glues. APP does not accumulate in the food chain but metabolizes into ammonia and phosphate. It is not thought to be acutely toxic to humans (Leisewitz et al. 2000). However, chemical manufacturers and foam manufacturing trade groups do not consider it to be an alternative for PeBDE on a large scale. Reasons for this are that APP is typically incorporated as a solid, it has adverse effects on foam properties and processing and it is not considered to be as effective as a fire retardant compared to other alternatives.

White foam has become the industry standard for flame-retarded, low-density foam in the mattress and bedding industries, and in many upholstered furniture applications in the United States. While the colour of the foam, however, is not a determinant of its flame retardancy, manufacturers seem to be reluctant to use discoloured/scorched foam for many applications because this is an indication of thermal stress on the foam which may lead to premature failure of the foam during its service life. Greater acceptance of discolored foams would allow manufacturers to choose from a wider variety of alternative flame retardants. Barrier fabrics are allowing mattress manufacturers to mask the colour.
Non-chemical alternatives to C-PeBDE in PUR foam

Non-chemical alternatives are also identified in US EPA (2005). Three currently available, alternative technologies for flame retarding furniture include barrier technologies, graphite impregnated foam and surface treatment. Graphite impregnated foam and surface treatments have limited commercial uses. Barrier technologies are predominantly used in mattress manufacturing rather than residential upholstered furniture. But there is considerable interest in future applications of these technologies for the furniture industry as well.

In addition to the following technologies, it should be noted that some furniture designs exclude the use of filling materials, and even fabric altogether. Design therefore, should be considered when evaluating alternative means for achieving flame retardancy in furniture. Flame-retardant barrier materials can be a primary defence in protecting padding for furniture and mattresses. Manufacturers can layer barrier materials to improve the flame retardancy of their products. This layering approach allows a product to maintain its fire resistance even if one layer is compromised. There are many types of barrier materials available, and some layers in the composites may be chemically treated to improve flame retardancy. Fabrics composed of natural fibres such as cotton may be chemically treated with phosphonitrillic chlorides, for example, but any hazards associated with these chemical treatments have not been assessed in this report.

Fabrics composed of synthetic fibres that are inherently flame retardant are also flame-retardant barrier materials. Plastic films derived from flame-retardant resins are also flame-retardant barrier materials. These materials are designed and manufactured to meet specific flammability standards. This also explains the large number of flame-retardant barrier materials that are available. Flame-retardant barrier materials can be characterized by cost, resulting in three primary groups. The first group of flame-retardant materials is the chemically treated, primarily boric acid treated, cotton-based materials. These materials are the least expensive flame-retardant barrier materials available. Mattress manufacturers that base their material decisions predominantly on cost prefer these flame retardants. Though estimates of exposure assume that use of boric acid-treated cotton will not significantly increase boron intake by the wearers, there is no information available on the release of boron in dust form consumer items (Leisewitz et al. 2000). The second group of flame-retardant materials is a blend of inexpensive natural fibres and expensive synthetic fibres. Synthetic fibres used in these blends include VISIL, Basofil, Polybenzimidazole, KEVLAR, NOMEX and fiberglass. Smaller manufacturers of furniture and mattresses in niche markets use these materials. These blends are commonly used in bus and airplane seating. The third group of flame-retardant materials is composed solely of expensive, high-performance synthetic fibres. They are generally used in industrial or high-performance applications such as firemen’s coats and astronaut space suits.

Given the range of alternative flame retardants available, a wise course would be to examine the manufacturing processes, evaluate the use of synthetic materials, and give preference to those that pose least risk.

Alternatives to C-PeBDE in EE-appliances

As of mid-November 2005, a number of big manufacturers were phasing out all PBDEs. Examples of alternative flame retardants processes currently being utilized include:

- Bromine-free circuit boards for TVs, VCRs and DVD players (Sony).
- Phosphorus-based flame retardants for printed circuit boards (Hitachi).
- Flame resistant plastic without deca-BDE (Toshiba).
- Halogen-free materials and low-voltage internal wires (Panasonic/Matsushita).

Manufacturing firms expects increased costs due to compliance with the EU ban on use of hazardous chemicals in EE-appliances, including PeBDE among a range of other substances. Among the world producers of EE-appliances 35% expect the price of their products to increase by less than 5%, another 23% of the producers expect an increase between 5 and 10%; 6% of the producers expect prices to increase by more than 10% (Environmental International Reporter, 2006).
Leisewitz et al. (2000) says that no problems should arise from the use of zinc borate, magnesium hydroxide or expandable graphite as alternatives to the brominated flame retardants.

Alternatives for C-PeBDE in textiles
There are bromine-free flame retardant alternatives. Some of them are not environmentally sound, such as antimony trioxide and borax. There are also durable flame retardant materials, such as wool and polyester fibres. Some manufacturers claim that a ban on the use of C-PeBDE in textiles will give poorer quality and durability of the textile.

Table 3.3 Bromine-free flame retardant chemicals for textiles.

<table>
<thead>
<tr>
<th>Textile</th>
<th>Alternative flame retardants in commercial materials</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVC, plastic coating of worker clothes</td>
<td>Antimony trioxide</td>
</tr>
<tr>
<td>Working clothes, Uniforms for off-shore,</td>
<td>Tetraakis(hydroxymethyl) phosphonium chloride (THPC)</td>
</tr>
<tr>
<td>electricity plants, military sector, police,</td>
<td></td>
</tr>
<tr>
<td>health sector</td>
<td></td>
</tr>
<tr>
<td>Cotton/polyester (bedclothes, clothing,</td>
<td>Phosphonitrilic chloride (PNC)</td>
</tr>
<tr>
<td>worker clothes, protective clothing) used in</td>
<td></td>
</tr>
<tr>
<td>public institutions, the off-shore sector,</td>
<td></td>
</tr>
<tr>
<td>ship and hotels</td>
<td></td>
</tr>
<tr>
<td>Cotton/polyester (worker clothes, protective</td>
<td>Proban (organic phosphorous compounds)</td>
</tr>
<tr>
<td>clothing) used in the off-shore sector, ship</td>
<td></td>
</tr>
<tr>
<td>and hotels</td>
<td></td>
</tr>
<tr>
<td>Carpets, textiles in the transport sector</td>
<td>Aluminium hydroxide</td>
</tr>
<tr>
<td>Tent, tarpaulin</td>
<td>Aluminium hydrate</td>
</tr>
<tr>
<td>Furniture textiles in the health sector,</td>
<td>Ammonium compounds</td>
</tr>
<tr>
<td>offices, industry and transport sector</td>
<td></td>
</tr>
<tr>
<td>Furniture for living room and bedroom</td>
<td>Borax</td>
</tr>
<tr>
<td>Mixed fibres wool and cellulose</td>
<td>Melamine</td>
</tr>
</tbody>
</table>


Table 3.4 Durable inherent flame retardant materials

<table>
<thead>
<tr>
<th>Material</th>
<th>Application</th>
</tr>
</thead>
<tbody>
<tr>
<td>PHD-cold foam</td>
<td>Mattresses and furniture</td>
</tr>
<tr>
<td>Flame retardant polyester fibre (Trevira CS)</td>
<td>Curtains, table clothes in public institutions, schools, vehicles, oil rigs</td>
</tr>
<tr>
<td>Wool</td>
<td>Furniture textiles for domestic use</td>
</tr>
<tr>
<td>Mixed fibres wool and cellulose</td>
<td>Bed clothes to baby carriages and beds</td>
</tr>
</tbody>
</table>


3.4 Summary of information on impacts on society of implementing possible control measures

Benefits of phasing out C-PeBDE
The most obvious benefits to the global society of phasing out C-PeBDE would be reduced releases to air, water and soil of the substance, as well as releases in workplace settings (UNEP, 2006). The major part of the releases of PeBDE ends up in soil and sediments, since in the environment the substance is bound to particles. PeBDE in soil or sediments is readily incorporated into the food chain and bioaccumulates in the fatty tissues of top predators, including humans. The substance is widespread in the global environment. Levels of PeBDE have been found in humans in all regions of the world (UNEP, 2006).

Most trend analyses show a rapid increase in concentrations of PeBDE in the environment and in humans from the early 1970s to the end of the 1990s. In the US, where PeBDE was in high use until recently and where it remains in such materials as polyurethane foam incorporated into consumer products, there has been a build-up in human tissue. Components found in C-PeBDE appear in a number of ecosystems and species, including
several endangered species. Some individuals of these species show levels high enough to be of concern. The potential for the toxic effects in wild life, including mammals, is evident.

Potential exposure of humans is through food, use of products containing C-PeBDE, and contact with indoor air and dust. PeBDE transfers from mothers to embryos and lactating infants. UNEP (2006), in its assessment, concludes that PeBDE is likely to cause significant adverse effects on human health or the environment, such that global action is warranted (see chapter 1.2). Continued use will entail a potentially large cost.

When considering phasing out C-PeBDE, there is also a need to weigh the potential risk of further production and use of the substance against the potential risk of production and use of alternative substances. This is particularly the case when it comes to the use of PeBDE as flame retardant. Fire prevention is important to protect human safety and avoid social and economic losses due to fire. Thus, to the extent that the alternative substances are less efficient at preventing flames and fires, there would be a loss for society of replacing PeBDE with these less efficient substances. In addition, many toxic substances are released during fires; hence fire prevention can yield environmental benefits. Using less flame retardant substances could therefore yield an environmental loss if fires become more frequent. However, according to US EPA (2005), the available alternatives function as well as PeBDE.

The discussion above leads to the conclusion that most of the alternatives are in themselves less hazardous to the environment than PeBDE. Indeed, few of the substitutes are classified as dangerous for the environment, though complete information is lacking in many cases. It is hard to find reliable assessments of the net environmental benefits of substituting PeBDE with other substances, because – among other reasons - of various properties of the substances and the increased amounts that will be needed to replace PeBDE. The criteria for assessing possible candidate substances have been published by the Danish EPA (1999).

Lack of data makes it impossible precisely to assess and quantify the overall net benefits of phasing out C-PeBDE. However, given the documented harm associated with PeBDE in the environment, its persistence and bioaccumulation, and given that most developed countries have already phased it out, there can be little doubt that the overall benefits are considered positive.

**Costs of phasing out C-PeBDE**

The incremental costs for users of C-PeBDE of replacing it with other substances in their products or re-designing the product itself to eliminate the need for additives would obviously have to be considered. Each affected plant would have its own suite of costs incurred by the phase out of C-PeBDE, so it is hard to make an overall assessment with any accuracy. Some producers may have to invest in new production equipment, but for most users this seems not to be necessary since there are available ‘drop in’ replacements. In general, the costs of buying many of the alternatives seem to be similar or slightly lower than buying C-PeBDE. However, for some alternatives there may be an increase in costs associated with a need for higher loads (RPA, 2000).

Listing C-PeBDE in the Stockholm Convention would oblige Parties to adopt measures or guidance, as specified in the Convention, for the handling of wastes contaminated with C-PeBDE. For those countries who have not yet adjusted their waste handling practices for C-PeBDE, adopting such measures will involve additional costs, in both developed and developing countries. In addition to containment technology and provisions for special handling, these measures could extend, for example, to the upgrading of waste treatment plants. No data are available on the costs experienced by countries that have adopted such measures. We have not seen reliable figures for the estimated costs of phasing out C-PeBDE, but most studies state that these cost are “low”. Allied to this economic analysis is the fact that most users in developed countries have phased out C-PeBDE seemingly without any great challenges.

Potential incremental costs of using alternative substances should be included in the analysis. In addition, an estimate might be made, albeit with great difficulty, of the reduced cost of damage to ecosystems and to public health when materials like C-PeBDE are removed from
the market. The Polluter Pays principle, under which such costs should be internalized by the producer and/or the user, is seldom applied (at least without regulatory assistance), and so no good estimates are available of the potential cost of damage avoided. Legacy problems such as that likely to be posed by the presence of PeBDE in the environment do not lend themselves to management under the terms of the Polluter Pays Principle since the original ‘polluter’ often cannot be identified or is no longer in business.

It could be discussed whether the costs for producers of C-PeBDE of closing the production and eventually switch to production of other substances should be included in a cost benefit analysis. These costs could be considered as part of an ordinary restructuring of production due to changes in market demand. No assessments of such restructuring costs for producers are found in the literature. Costs incurred by national governments related to regulation, enforcement and compliance activities should be included in the assessment. No such cost figures are found, but overall costs are likely to be low in developed countries where the systems for monitoring and control activities are in place. However, the costs could be considerable for developing countries without these systems. On the other hand the implementation of the Stockholm Convention will require these systems to be established, and cannot be considered a cost for listing C-PeBDE. It could however be extra costs for the waste handling of products and materials containing C-PeBDE.

3.5 Other considerations

All Parties to the Convention would be responsible for the monitoring and enforcement of a worldwide ban on the production and use of C-PeBDE if it were to be listed as a POP. It should be relatively simply to control the production, export and imports of C-PeBDE, but the same cannot be said about production, export and import of products and materials that contain PeBDE (RPA, 2000). Most developed countries have the required systems to deal with those problems, but it could be an extra cost for developing countries, if C-PeBDE is listed. The treaty does not ban products already in use, but poses an obligation on proper waste handling. This could also imply extra costs for developing countries. It could be extremely difficult and expensive to quantify the chemicals in products. But because of national and international standards for fire safety for some product groups, the industry already have the obligation to have this knowledge. There is also an expanding amount of knowledge about use in products, from surveys in the developed world. The threat of infringers being excluded from the world market should discourage producers from placing such products on the market. Most of the alternatives are less-than or as expensive as C-PeBDE, and there should therefore be less economic incentives to produce new products containing C-PeBDE. For electric articles it has been found to be impractical and costly to divide the product streams, to apply with the ban in EU. The market is therefore already phasing out use of banned BFR in electric articles globally. National authorities would have to make surveys to get more detailed national information about C-PeBDE content in different articles becoming waste. Monitoring and control with the ban could be done through spot tests, aiming on distributors and producers. But the development of more appropriate methodologies for determining the chemical species present in new flame retarded consumer goods and other products would facilitate a ban.

According to the Bromine Science and Environment Forum, all developed countries have in place monitoring and control capacities as well as legislative tools to restrict the use of C-PeBDE. Thus, the main challenge in this area would be for the developing countries to get sufficient capacities in place.

4. Synthesis of information

4.1 Summary of evaluation

Pentabromodiphenyl ether (PeBDE) is a brominated flame retardant (BFR). BFRs are a group of brominated organic substances that inhibit or suppress combustion in organic material. They have been used almost exclusively in the manufacturing of flexible polyurethane (PUR) foam for furniture and upholstery in homes and vehicles, packaging, (non-foamed) PUR in
casings and electronic equipment (EEE). To some extent they have also been used in specialized applications in textiles and in various other uses.

Because of its chemical and toxic properties and wide spread occurrence in the environment and in humans, PeBDEs cause concern in many regions of the world. The substance has shown to be persistent and bioaccumulative, and thus a potential risk for future generations. Concentrations in wildlife and in humans have also increased significantly. Those findings have resulted in voluntary and regulatory phase-outs of this compound in several regions of the world. Most developed countries have either banned the production and use of PeBDE or put restrictions on use. There are national and international standards for fire safety for some product groups. This applies for example to electrical material, industrial packaging, upholstered furniture, curtains, electronic household appliances and electrical cables. These standards specify the flame-retarding properties that are required. Until now, brominated flame retardants have been the cheapest and therefore considered to be the most efficient in cost-effectiveness terms. However, it has become increasingly common to replace these substances either with flame retardants without bromine, or to change the design of the product so that there is no need for the continued use of chemical flame retardants. It has also become important to avoid the use of products containing flame retardants if such is not absolutely necessary on the basis of fire safety.

Suitable alternatives seem to exist for almost all use of PeBDE. However, some of the alternative substances are also hazardous, and the impacts of some are not properly investigated. Still, overall benefits from phasing out the use of C-PeBDE are assumed to be positive. Costs of phasing out C-PeBDE are generally perceived to be “low” due to the fact that most developed countries have already phased out C-PeBDE without meeting excessive challenges. Cost-competitive non-POP alternatives are available and have been taken up by companies as replacements for PeBDE in PUR-foam and electronic equipment.

4.2 Elements to a risk management strategy

Since the dissemination of PeBDE into the environment is a global, transboundary problem, global actions to phase out C-PeBDE should be considered. A global ban on production and use of C-PeBDE covering all sectors, to be achieved by listing C-PeBDE under Annex A of the Stockholm Convention, would be the most appropriate measure, given that most developed countries have already banned production. Eventually, some very special uses of C-PeBDE (military airplanes, space suits etc.) where alternatives are not efficient enough and/or very costly could be exempted from the ban for a time-limited transition period. Developed countries have in place all monitoring and control capacities as well as legislative tools to enforce a ban. Thus, the main enforcement challenge would be for the developing countries to get sufficient capacities in place.

Several countries have reported that they would have problems regulating a commercial mixture of PeBDE. Most national regulations concern compounds. It will therefore be more practical, rather than listing C-PeBDE under the Convention, as was earlier envisaged by the POPRC, to list brominated diphenylethers with 4 or 5 bromines. All mixtures with one of the isomers of Tetrabromodiphenyl or Pentabromodiphenyl ether will then be covered by the conditions in the Convention, except when they occur as trace. The Convention could set lower limits for these listed substances, so that mixtures containing concentrations below these levels (traces, for example) would not be covered. Complete coverage of the components of the C-PeBDE would require also the listing of Hexabromodiphenyl ether, with the same lower limit, since it can comprise up to 12% of the commercial product. This could be an issue for the listing of commercial Octabromodiphenyl ether, which also contains appreciable amounts of the HeBDE.

Provision of guidance on criteria for the selection of alternatives to PeBDE should be part of the risk management strategy for the elimination of this substance. It will be important to discourage the replacement of PeBDE with other environmentally harmful substances.
A ban would eliminate emissions from the manufacture of C-PeBDE and products containing it. It would not affect the emissions from C-PeBDE in products already in use. Recycling and reuse of products containing C-PeBDE would not be allowed, if it results in new use of the isomers of TeBDE or PeBDE as a constituent of new products, since these activities are banned under Article 6 of the Convention. Recycling and recovery can occur, but only if the new product does not contain the isomers of TeBDE and PeBDE. Additional regulations might need to be considered when products are treated to recover the valuable materials such as metals that are contained in them, and the components of C-PeBDE is inadvertently released to the environment. This would especially be important for recycling of electronic articles containing C-PeBDE and for shredder plants handling these and other products, like vehicles. Some components in the waste fraction can be sorted out, but for most EE appliances this will not be practical. Thus, new regulations might require installation of air pollution control devices on some incinerators and plants, and that would be costly for them. However, most developed countries already have other restrictions that require off-gas filtering of the emissions from recycling and shredder plants. These source categories could be added to Annex C.

Consideration was given to listing of brominated diphenylethers with 4 or 5 bromines in Annex B, with targets to be set for the phase out of the use of specific existing products containing C-PeBDE. However, collection of such products would be a major task and the likely complexity of such schemes militated against such a recommendation. The general rules on waste handling in the Stockholm Convention will, of course, apply to C-PeBDE once brominated diphenylethers with 4 or 5 bromines is listed.

Waste fractions containing C-PeBDE should be handled as hazardous waste. This is already done in large parts of the UN ECE region. This could impose extra costs on some countries and sectors. Ways to ensure collection of articles containing C-PeBDE, and the setting of targets, should therefore be left to each country.

The solutions for waste handling should to a large extent depend on local conditions and be designed to fit into existing systems and traditions, taking the general rules of the Convention into consideration, including the general guideline on waste handling for POPs in the Basel Convention. The listing under the Stockholm Convention will imply the development of a guideline for waste containing the isomers of TeBDE and PeBDE under the entity of Basel, that have to be considered as well.

5. Concluding statement

Having evaluated the risk profile for C-PeBDE, and having concluded that this chemical is likely, as a result of long-range environmental transport, to lead to significant adverse effects on human health and the environment, such that global action is warranted, this risk management statement has been prepared in accordance with the content specified in Annex F of the Convention.

In order to ensure that listing under the Convention achieves the aim of preventing the use of the hazardous components of the C-PeBDE mixtures, the Committee recommends to the Conference of the Parties to the Stockholm Convention that, in accordance with paragraph 9 of Article 8 of the Convention and specifying the related control measures, brominated diphenylethers with 4 or 5 bromines be listed in Annex A.
References


### B.10. Perfluorooctane sulfonate - SUMMARY

#### SUMMARY

10. PFOS

Draft Risk Management Evaluation May 2007


Risk Profile UNEP/POPS/POPRC.2/17/Add5


| Composition | PFOS is a fully fluorinated anion, which is commonly used as a salt or incorporated into larger polymers. PFOS and its closely related compounds, which contain PFOS impurities or substances which can give rise to PFOS, are members of the large family of perfluoroalkyl sulfonate substances. PFOS can be formed by degradation from a large group of related substances, referred to as PFOS-related substances |
| Uses | Uses include: fire fighting foams, carpets, leather/apparel, textiles/upholstery, paper and packaging, coatings and coating additives, industrial and household cleaning products, pesticides and other insecticides, photographic industry, photolithography and semiconductor manufacturing, hydraulic fluids, and metal plating. PFOS-related substances have been used to provide soil, oil and water resistance to textiles, apparels, home furnishings and upholstery, carpets, and leather products. PFOS-related substances have been used in the packaging and paper industries in both food packaging and commercial applications to impart grease, oil and water resistance to paper, paperboard and packaging substrates. 3M PFOS-based products were sold in the past to a variety of formulators to improve the wetting of water-based products marketed as alkaline cleaners, floor polishes (to improve wetting and levelling), denture cleansers and shampoos. Several of these products (alkaline cleaners, floor polishes, shampoos) were marketed to consumers; some products were also sold to janitorial and commercial services. A number of the alkaline cleaners were spray-applied. |
| Releases | PFOS and PFOS-related substances can be released to the environment at their manufacture, during their use in industrial and consumer applications and from disposal of the chemicals or of products or articles containing them after their use. |
| Fate | PFOS is extremely persistent. It has not shown any degradation in tests of hydrolysis, photolysis or biodegradation in any environmental condition tested. The only known condition whereby PFOS is degraded is through high temperature incineration. PFOS meets the criteria for the potential for long-range transport. This is evident through monitoring data showing highly elevated levels of PFOS in various parts of the northern hemisphere. It is especially evident in the Arctic biota, far from anthropogenic sources. PFOS also fulfills the specific criteria for atmospheric half-life. |
| Effects | PFOS has demonstrated toxicity towards mammals in sub-chronic repeated dose studies at low concentrations, as well as rat reproductive toxicity with mortality of pups occurring shortly after birth. Environmental toxicity data for PFOS is predominantly found for aquatic organisms such as fish, invertebrates and algae, and for birds. PFOS is toxic to aquatic |
organisms with mysid shrimp and *Chironomus tentans* being the most sensitive organisms.

| Exposure | Most notable and alarming are the high concentrations of PFOS that have been found in Arctic animals, far from anthropogenic sources. PFOS has been detected in higher trophic level biota and predators such as fish, piscivorous birds, mink, and Arctic biota. Also, predator species, such as eagles, have been shown to accumulate higher PFOS concentrations than birds from lower trophic levels. Even with reductions in manufacturing of PFOS by some manufacturers, wildlife, such as birds, can continue to be exposed to persistent and bioaccumulative substances such as PFOS simply by virtue of its persistence and long-term accumulation. Concentrations in Canadian Arctic polar bear are among the highest in polar bears worldwide but the exposure concentrations are not considered an anomaly given similar concentrations in polar bears in other North America and European Arctic locations and high concentrations in other wildlife globally as shown above. |
| Status | PFOS was added to the list of Chemicals for Priority Action under OSPAR in June 2003. Persistent Organic Pollutants Protocol to the Long-Range Transboundary Air Pollution Convention ("LRTAP"): The Executive Body of the UNECE LRTAP Convention agreed that PFOS be considered a POP as defined under the Protocol on POPs and requested that the UNECE Task Force on POPs continue with the review of the substance and exploring management strategies. |
| Alternatives | The draft risk management evaluation received responses from various industries which claimed that the following uses have no technically feasible alternatives: photo imaging, photo mask, semi-conductor, aviation hydraulic fluids, and manufacture of ant baits for leaf-cutting ants. The industries also claim that the following uses have alternatives but would have to be gradually phased in: metal plating and fire fighting foam. |
B.10.a. Perfluorooctane sulfonate– DETAILED PROFILE

Perfluorooctane sulfonate
Background

In September 2005, the government of Sweden made a proposal for listing perfluorooctane sulfonate (PFOS) and 96 PFOS-related substances in Annex A of the Stockholm Convention on Persistent Organic Pollutants (POPs).

Introduction

Perfluorooctane sulfonate is a fully fluorinated anion which is used as such in some applications or incorporated into larger polymers. Perfluorinated substances with long carbon chains, including perfluorooctane sulfonate, are both lipid-repellent and water-repellent. Therefore, perfluorooctane sulfonate-related substances are used as surface-active agents in various applications. The extreme persistence of these substances makes them suitable for high-temperature applications and for applications in contact with strong acids or bases. They are used in a wide variety of applications e.g. in textiles and leather products; metal plating; food packaging; fire fighting foams; floor polishes; denture cleansers; shampoos; coatings and coating additives; in the photographic and photolithographic industry; and in hydraulic fluids in the aviation industry.

Perfluorooctane sulfonate can be formed by degradation from a large group of related substances, referred to as perfluorooctane sulfonate-related substances. Perfluorooctane sulfonate and 96 perfluorooctane sulfonate-related substances are part of the nomination. All these substances are members of a large family of perfluoroalkylated substances, in which also some substitutes for perfluorooctane sulfonate can be found.

Data Sources:
— Hazard Assessment of Perfluorooctane Sulfonate and its Salts. OECD, 2002

1. Chemical and Physical properties

1.1 Names and registry numbers

| Chemical name: | Perfluorooctane Sulfonate (PFOS) |
| Molecular formula: | \( C_{17}F_{17}SO_3^- \) |
| Synonyms: | 1-Octanesulfonic acid, \( C_8F_{17}SO_3^- \) acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro; 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8-heptadecafluoro-1-octanesulfonic acid; 1-Octanesulfonic acid, heptadecafluoro-; 1-Perfluorooctanesulfonic acid; Heptadecafluoro-1-octanesulfonic acid; Perfluoro-n-octanesulfonic acid; Perfluorooctylsulfonic acid |

Trade names: There is a list of 96 substances which could degrade to perfluorooctane sulfonate in the environment.

PFOS, as an anion, does not have a specific CAS number. The parent sulfonic acid and some of its commercially important salts are listed below:

- Perfluorooctane sulfonic acid (CAS No. 1763-23-1)
- Potassium salt (CAS No. 2795-39-3)
- Diethanolamine salt (CAS No. 70225-14-8)
- Ammonium salt (CAS No. 29081-56-9)
- Lithium salt (CAS No. 29457-72-5)
1.2 Structure

![Figure 1. Structural formula of PFOS shown as its potassium salt](image)

Molecular weight: 506.1 (potassium salt)

The physical and chemical properties of the potassium salt of PFOS are listed in Table 2.

Table 2. Physical and chemical properties of PFOS potassium salt. (Data from OECD, 2002, unless otherwise noted).

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance at normal temperature and pressure</td>
<td>White powder</td>
</tr>
<tr>
<td>Molecular weight</td>
<td>538 g/mol</td>
</tr>
<tr>
<td>Vapour Pressure</td>
<td>$3.31 \times 10^{-4}$ Pa</td>
</tr>
<tr>
<td>Water solubility in pure water</td>
<td>$519 \text{ mg/L (20 } \pm 0.5^\circ\text{C)}$</td>
</tr>
<tr>
<td></td>
<td>$680 \text{ mg/L (24 - 25}^\circ\text{C)}$</td>
</tr>
<tr>
<td>Melting point</td>
<td>$&gt; 400^\circ\text{C}$</td>
</tr>
<tr>
<td>Boiling point</td>
<td>Not measurable</td>
</tr>
<tr>
<td>Log $K_{OW}$</td>
<td>Not measurable</td>
</tr>
<tr>
<td>Air-water partition coefficient</td>
<td>$&lt; 2 \times 10^{-6}$ (3M, 2003)</td>
</tr>
<tr>
<td>Henry’s Law Constant</td>
<td>$3.09 \times 10^{-9}$ atm m$^3$/mol pure water</td>
</tr>
</tbody>
</table>

PFOS can be formed (by environmental microbial degradation or by metabolism in larger organisms) from PFOS-related substances, i.e., molecules containing the PFOS-moiety depicted in Figure 1. Although the ultimate net contribution of individual PFOS-related substances to the environmental loadings of PFOS cannot be predicted readily, it is considered here that any molecule containing the PFOS moiety can be a precursor to PFOS.

The majority of PFOS-related substances are polymers of high molecular weights in which PFOS is only a fraction of the polymer and final product (OECD, 2002). PFOS-related substances have been defined somewhat differently in different contexts and there are currently a number of lists of PFOS-related substances (Table 3). The lists contain varying numbers of PFOS-related substances that are thought to have the potential to break down to PFOS. The lists overlap to varying extents depending on the substances under consideration and the overlap between national lists of existing chemicals.

Table 3. Number of PFOS-related substances as proposed by UK – DEFRA, US – EPA, OECD, OSPAR, and Canada

<table>
<thead>
<tr>
<th>Source</th>
<th>Number of PFOS-related substances</th>
</tr>
</thead>
<tbody>
<tr>
<td>UK – DEFRA (2004)</td>
<td>96</td>
</tr>
<tr>
<td>OECD (2002)</td>
<td>172 (22 classes of perfluoroalkyl sulfonate substances)</td>
</tr>
<tr>
<td>OSPAR (2002)</td>
<td>48</td>
</tr>
<tr>
<td>Canada (2004)</td>
<td>~ 50</td>
</tr>
</tbody>
</table>

A large number of substances may give rise to PFOS and thus contribute to the contamination problem. DEFRA, UK (2004), has recently proposed a list of 96 PFOS-related substances. However, the properties of the 96 substances have not generally been determined. They may have very different environmental characteristics such as solubility, stability and ability to be absorbed or metabolised (3M, insert publication date). Nevertheless,
it is expected that all of these substances would give rise to the final degradation product of PFOS.

Environment Canada’s ecological risk assessment defines PFOS precursors as substances containing the perfluorooctylsulfonyl (C₈F₁₇SO₂, C₈F₁₇SO₃) moiety that have the potential to transform or degrade to PFOS. The term “precursor” applies to, but is not limited to, some 50 substances identified in the ecological assessment. However, this list is not considered exhaustive as there may be other perfluorinated alkyl compounds that are also PFOS precursors. This information was compiled based on a survey to industry, expert judgement and CATABOL modelling, in which 256 perfluorinated alkyl compounds were examined to determine whether non-fluorinated components of each substance were expected to degrade chemically and/or biochemically and whether the final perfluorinated degradation product was predicted to be PFOS.

In order to avoid excluding substances that may be PFOS precursors, PFOS-related substances/potential PFOS precursors are defined in this document as all molecules having the following molecular formula: C₈F₁₇SO₂Y, where Y = OH, metal salt, halide, amide and other derivatives including polymers. This definition has been proposed by the EU (EU COM 2005).

2. **Persistence**

Perfluorooctane sulfonate is extremely persistent. A study on the hydrolysis of perfluorooctane sulfonate in water has been performed at a range of temperatures and pH values with no observable degradation; the half-life of perfluorooctane sulfonate was determined to be over 41 years. Biodegradation of perfluorooctane sulfonate has also been evaluated under aerobic and anaerobic conditions. No apparent degradation occurred.

3. **Bioaccumulation**

Perfluorooctane sulfonate does not accumulate in fatty tissue, as is typical of many persistent organic pollutants. This is because perfluorooctane sulfonate is both hydrophobic and lipophobic. Rather, perfluorooctane sulfonate binds to proteins in the blood and the liver. The octanol-water partition coefficient (logKow) is not measurable for perfluorooctane sulfonate.

The kinetic bioconcentration factor in bluegill sunfish (Lepomis macrochirus) for whole fish was determined to be 2,796. In another study, on rainbow trout (Oncorhynchus mykiss), bioconcentration factors in liver and plasma were estimated at 2,900 and 3,100 respectively.

Monitoring data from top predators at various locations show highly elevated levels of perfluorooctane sulfonate and demonstrate the substantial bioaccumulating and biomagnifying properties of perfluorooctane sulfonate. The concentrations of perfluorooctane sulfonate found in livers of arctic Polar Bears exceed the concentrations of all other known individual organohalogens. Biomagnification factors for a variety of mammals, birds and fish range from 22 to 160 between two trophic levels.

4. **Potential for long-range environmental transport**

The potassium salt of perfluorooctane sulfonate has a measured vapour pressure of 3.31 × 10⁻³ Pa. As a result of this vapour pressure and a low air-water partition coefficient (< 2 × 10⁻⁶), perfluorooctane sulfonate itself is not expected to volatilize significantly. It is therefore assumed to be transported in the atmosphere predominantly bound to particles, because of its surface-active properties, rather than in a gaseous state. The atmospheric half-life of perfluorooctane sulfonate is expected to be greater than two days, based on its extreme resistance to degradation in all tests performed. The indirect photolytic half-life of perfluorooctane sulfonate has been estimated to be over 3.7 years.
The presence of perfluorooctane sulfonate in a wide variety of Arctic biota, far from anthropogenic sources, demonstrates the capacity of perfluorooctane sulfonate to undergo long-range transport.

5. Adverse effects

Toxicology data are available for rats and monkeys following acute, subchronic and chronic exposures. High doses of perfluorooctane sulfonate (potassium salt) caused death, and at lower doses (sub-milligram), gastrointestinal lesions and loss of weight were observed. Maternal and pup death and toxicity were reported in a multigenerational study. Perfluorooctane sulfonate may affect lung maturation in young rats.

Perfluorooctane sulfonate has shown moderate acute toxicity to fish. The lowest observed LC$_{50}$ (96 hours) was estimated at 4.7 mg/l in fathead minnow (*Pimephales promelas*) exposed to the lithium salt of perfluorooctane sulfonate. The lowest LC$_{50}$ (96 hours) for aquatic invertebrates has been observed in the mysid shrimp (*Mysidopsis bahia*) at 3.6 mg/l. The most sensitive algae appear to be the green algae *Pseudokirchneriella subcapitata* with an IC$_{50}$ (96h, cell density) of 48.2 mg/l.

Reasons for concern

“According to the available data, perfluorooctane sulfonate is extremely persistent in the environment. Due to its physical and chemical properties and considerably long atmospheric half-life and based on findings in environmental samples in distant locations, e.g., the Arctic, it can be assumed that perfluorooctane sulfonate/perfluorooctane sulfonate-related substances can be transported long distances in air, far from their sources. Perfluorooctane sulfonate is associated with serious harmful effects in mammals and aquatic organisms.

The voluntary phase-out of perfluorooctane sulfonate production by the major producer in the USA has led to a significant reduction in the use of perfluorooctane sulfonate-related substances. However, it can be assumed that it is still produced in some countries and there is evidence that it continues to be used in many countries. As perfluorooctane sulfonate-related substances can move in the atmosphere to locations far from their sources, measures taken by single countries or groups of countries are not sufficient to abate the pollution caused by it. Regional action has already been considered necessary and perfluorooctane sulfonate is nominated under the Convention on Long-range Transboundary Air Pollution Protocol on Persistent Organic Pollutants. Due to the harmful persistent organic pollutant properties and risks related to its possible continuing production and use, global action is warranted to eliminate the pollution caused by perfluorooctane sulfonate.”
PFOS is a fully fluorinated anion, which is commonly used as a salt or incorporated into larger polymers. PFOS and its closely related compounds, which contain PFOS impurities or substances which can give rise to PFOS, are members of the large family of perfluoroalkyl sulfonate substances.

The Persistent Organic Pollutants Review Committee (POPRC) has evaluated Annex D at the First meeting of the POPRC, Geneva, 7-11 November 2005, and has concluded that PFOS meets the screening criteria specified in Annex D.

Data sources
This builds on information that has been gathered by the United Kingdom, i.e., in the hazard assessment report prepared by the UK and the USA for the OECD, and in the UK risk reduction strategy:
Some recent information from the open scientific literature (up to October 2005) is also included. Data submitted by Parties and observers, which have been considered, are also included in this report when they add new info.

Summary of available risk information
The hazard assessment of PFOS, prepared by the OECD in 2002, concluded that the presence and the persistence of PFOS in the environment, as well as its toxicity and bioaccumulation potential, indicate a cause of concern for the environment and human health.

An environmental risk assessment, prepared by the UK-Environment Agency, and discussed by the EU member states under the umbrella of the existing substances regulation (ESR DIR 793/93) shows that PFOS is of concern.

The Environment Canada/Health Canada Draft Assessment of PFOS, its Salts and its Precursors was released for public comment in October 2004. The ecological and human health assessments have been revised and should be publicly available soon. The ecological risk assessment has concluded that PFOS is persistent, bioaccumulative, and inherently toxic.

Sweden has made a notification to the European Commission concerning proposed restrictions on marketing and use of PFOS and their 96 known derivatives. The proposed Swedish regulation prohibits products which wholly or partly contain PFOS or PFOS related substances. These products must not be offered for sale or handed over to consumers for individual use or offered for sale and handed over or used commercially.

This prohibition shall not apply to hydraulic fluids intended for use in aircraft.

The UK has notified a national regulation of PFOS and substances that degrade to it. The proposed UK regulation prohibits the import into the United Kingdom of fire fighting foams containing perfluorooctane sulfonate. The regulation also prohibits the supply, storage and use of perfluorooctane sulfonate for any uses and time limited derogations for certain uses.

The UK and Sweden have proposed the following classification for PFOS in EU (2005):
T Toxic
R40 Carcinogen category 3; limited evidence of carcinogenic effect
R48/25 Toxic; danger of serious damage to health by prolonged exposure if swallowed
R61 May cause harm to the unborn child
R51/53 Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment

The EU is now considering a proposal on the prohibition of PFOS and PFOS-related compounds in some products and chemical mixtures.

Norway is now considering a proposal to prohibit the use of fire fighting foams containing PFOS and PFOS-related compounds, which is the major use of these compounds today in Norway.

The Environmental Protection Agency (EPA) in the USA finalized two Significant New Use Rules (SNURs) in 2002, requiring companies to inform the EPA before manufacturing or importing 88 listed PFOS-related substances. The EPA proposed an additional SNUR under section 5(a)(2) of the Toxic Substances Control Act (TSCA) in March 2006 to include within the scope of this regulation another 183 perfluoroalkyl sulfonates (PFAS) with carbon chain lengths of five carbons and higher. The EPA further proposed an amendment to the Polymer Exemption rule in March 2006 which would remove from exemption polymers containing certain perfluoroalkyl moieties consisting of CF3- or longer chains, and would require that new chemical notifications be submitted on such polymers.

Status of the chemical under international conventions

OSPAR: PFOS was added to the list of Chemicals for Priority Action in June 2003.
Persistent Organic Pollutants Protocol to the Long-Range Transboundary Air Pollution Convention ("LRTAP"): Perfluorooctane sulfonate and its precursors were approved under Track A and are currently under Track B review.

SUMMARY INFORMATION RELEVANT FOR THE RISK PROFILE

Sources

1. Production, trade and stockpiles
The main production process of PFOS and PFOS-related substances is electro-chemical fluorination (ECF) and utilized by 3M, the major global producer of PFOS and PFOS-related substances prior to 2000.

   - Direct fluorination, electro-chemical fluorination (ECF):
     \[ C_8H_{17}SO_2Cl + 18 HF \rightarrow C_8F_{17}SO_2F + HCl + \text{by products} \]

The reaction product, perfluorooctanesulfonyl fluoride (PFOSF)\(^{69}\) is the primary intermediate for synthesis of PFOS and PFOS-related substances. The ECF method results in a mixture of isomers and homologues with about 35-40% 8-carbon straight chain PFOSF. However, the commercial PFOSF products were a mixture of approximately 70% linear and 30% branched PFOSF derivate impurities. The global production of PFOSF by 3M until the production ceased is estimated to have been 13,670 metric tones (1985 to 2002), with the largest yearly production volume, 3500 metric tones, in 2000 (3M, Submission to SC, 2006). PFOSF may be further reacted with methyl- or ethyl-amine to form N-ethyl- and N-methyl perfluorooctane sulfamide and subsequently with ethylene carbonate resulting in N-ethyl- and -methyl-perfluorooctane sulfamidoethanol (N-EtFOSE and N-MeFOSE). N-EtFOSE and N-MeFOSE were the principal building blocks of 3M’s product lines. PFOS is formed after the chemical or enzymatic hydrolysis of PFOSF (3M, 1999)

Other production methods for perfluoroalkylated substances are telemerisation and oligomerisation. However, to which extent these methods are applied for production of PFOS and PFOS-related substances is not evident.

\(^{69}\) In the OECD report, 2002, perfluorooctanesulfonyl fluoride is abbreviated POSF.
On 16 May 2000, 3M announced that the company would phase-out the manufacture of PFOS and PFOS-related substances voluntarily from 2001 onwards. The 3M global production of PFOS and PFOS-related substances in year 2000 was approximately 3,700 metric tonnes. By the end of 2000 about 90 % of 3M’s production of these substances had stopped and in the beginning of 2003 the production ceased completely.

3M’s voluntary phase-out of PFOS production has led to a significant reduction in the use of PFOS-related substances. This is due not only to the limited availability of these substances (3M had at the time the greatest production capacity of PFOS-related substances in the world), but also to action within the relevant industry sectors to decrease companies’ dependence on these substances.

The US Environmental Protection Agency (US EPA) compiled a list of non-US companies, which are believed to supply PFOS-related substances to the global market. Of these (and excluding the plant of 3M in Belgium), six plants are located in Europe, six are located in Asia (of which four are in Japan) and one in Latin America (OECD, 2002). However, this list may not be exhaustive or current.

According to the recent submission from Japan to the SC there is one manufacturer in Japan still producing PFOS and with a production amount of 1-10 tonnes (2005). The submission from Brazil states that lithium salt of PFOS is produced but that no quantitative data is available.

2. Uses

Perfluorinated substances with long carbon chains, including PFOS, are both lipid-repellent and water-repellent. Therefore, the PFOS-related substances are used as surface-active agents in different applications. The extreme persistence of these substances makes them suitable for high temperature applications and for applications in contact with strong acids or bases. It is the very strong carbon-fluorine binding property that causes the persistence of perfluorinated substances.

The historical use of PFOS-related substances in the following applications has been confirmed in the US (all), in the UK (the first six), or the EU (the final two) only.

- Fire fighting foams
- Carpets
- Leather/apparel
- Textiles/upholstery
- Paper and packaging
- Coatings and coating additives
- Industrial and household cleaning products
- Pesticides and insecticides

In the UK study (RPA & BRE, 2004), detailed information has been received from the following sectors that currently use PFOS-related substances:

- Use of existing fire fighting foam stock
- Photographic industry
- Photolithography and semiconductor
- Hydraulic fluids
- Metal plating

The sectors presented above account for the UK. However, deviation in the current use pattern between EU countries can not be excluded.

PFOS and its precursors are not manufactured in Canada but rather are imported as chemicals or products for Canadian uses. They may also be components in imported manufactured articles. It is estimated that the majority of PFOS has been used as water, oil,
soil and grease repellents (e.g. on fabric, leather, paper, packaging, rugs and carpets) and as surfactants (e.g. in fire fighting foams, coating additives) (Environment Canada, 2004).

PFOS and its precursors are not manufactured in the US, but can be imported either as chemicals or in products for the specific limited uses that were excluded from regulation. These comprise use as an anti-erosion additive in aviation hydraulic fluids; use as a component of a photoresist substance, including a photo acid generator or surfactant, or as a component of an anti-reflective coating, used in a photomicroolithography process to produce semiconductors or similar components of electronic or other miniaturized devices; use in coatings for surface tension, static discharge, and adhesion control for analog and digital imaging films, papers, and printing plates, or as a surfactant in mixtures used to process imaging films; and use as an intermediate only to produce other chemical substances to be used solely for these uses. Historically, PFOS and its precursors were also used as surfactants in fire fighting foams and in industrial and household cleaning products; in carpet, textile, leather, and paper coatings; and in termite and ant bait insecticide products. Stocks of PFOS and PFOS-containing products that were in existence at the time the US regulations were promulgated in 2002 could continue to be used in any application until they were consumed without violating the regulation, except that the PFOS-related insecticide products are subject to a phaseout agreement prohibiting their use after 2015.

The table below outlines the estimated current demand for PFOS-related substances in these applications in the EU (RPA & BRE, 2004).

<table>
<thead>
<tr>
<th>Industry Sector</th>
<th>Quantity (kg/year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Photographic industry</td>
<td>1,000</td>
</tr>
<tr>
<td>Photolithographic and semi-conductors</td>
<td>470</td>
</tr>
<tr>
<td>Hydraulic fluids</td>
<td>730</td>
</tr>
<tr>
<td>Metal plating</td>
<td>10,000</td>
</tr>
</tbody>
</table>

In the survey on production and use of PFOS and related substances performed by OECD in 2004 data concerning PFOS were difficult to separate from data on other substances e.g. PFAS.

**Fire Fighting Foams**

Water is vital and effective in extinguishing a majority of fires. However, when fighting fires involving flammable liquids (Class B), water tends to sink below the burning fuel due to its specific gravity and, thus, has little effect in extinguishing the fires (and in some cases could even result in the flammable liquid spilling out of its contained area). Fire fighting foams were therefore developed for use on flammable liquids fires and have proven to be one of the most important and effective tools for dealing with such fires. Fire fighting foams are produced by a combination of foam concentrate (the form in which it is stored) and water, which is then aspirated with air to form the finished foam. The resulting foam forms a low-density blanket that extinguishes fires from flammable liquids.

The fire fighting foams can be grouped in two main categories:

- Fluorine containing foam types (some of them consist of PFOS-related substances)
- Fluorine-free foam types

Since the announcement of the voluntary cessation of production of PFOS-related substances by 3M, the presence of PFOS in fire fighting foams has gradually decreased (RPA & BRE, 2004). Historically, in Canada, the most significant imports of PFOS, itself, were in the form of the potassium salt, used for fire-fighting foams (Environment Canada, 2004). Canada has also identified that existing stocks of PFOS-containing fire fighting foams could be a continued significant source of releases.

An industry survey conducted in the US by the Fire Fighting Foam Coalition in 2004 reported that the total inventory of aqueous film-forming foam in the US was approximately 9.9 million gallons, of which about 45% was PFOS-based stocks produced before 2003, with the other 55% comprised of telomere-based foams.
Textile, Carpet and Leather Protection

PFOS-related substances have been used to provide soil, oil and water resistance to textiles, apparels, home furnishings and upholstery, carpets, and leather products. They were used because they were able to modify the surface properties of these materials to provide repellence and resistance. When applied to a material’s surface, the perfluorocarbon chain tends to be oriented away from the surface, lowering the surface energy of the material, thereby creating a protective barrier. Since 3M’s withdrawal from the market, PFOS-related substances are used to a much smaller extent for these applications (RPA & BRE, 2004).

Paper and Packaging Protection

FOS-related substances have been used in the packaging and paper industries in both food packaging and commercial applications to impart grease, oil and water resistance to paper, paperboard and packaging substrates. According to 3M, fluorochemicals were used for both food contact applications (plates, food containers, bags and wraps) and non-food applications (folding cartons, containers and carbonless forms and masking papers). Since 3M’s withdrawal from the market, PFOS related substances are used to a much smaller extent for these applications (RPA & BRE, 2004).

Coatings and Coating Additives

3M indicates that prior to its voluntary phase out of PFOS production, the company would sell fluorochemical polymer coatings and coating additives which were used undiluted or diluted with water or butyl acetate to impart soil or water repellence to surfaces (including printing circuit boards and photographic film). These polymers contained fluorocarbon residuals at a concentration of 4% or less. Other applications for aqueous coatings are to protect tile, marble and concrete. It is unclear which of these products were actually based on PFOS-related substances.

A (DATE) survey in the UK among members of the British Coatings Federation (BCF) showed that the use of PFOS-related substances for these purposes is very limited (RPA & BRE, 2004).

Industrial and Household Cleaning Products (Surfactants)

3M PFOS-based products were sold in the past to a variety of formulators to improve the wetting of water-based products marketed as alkaline cleaners, floor polishes (to improve wetting and levelling), denture cleansers and shampoos. Several of these products (alkaline cleaners, floor polishes, shampoos) were marketed to consumers; some products were also sold to janitorial and commercial services. A number of the alkaline cleaners were spray-applied.

With regard to the UK cleaning products industry, the responses received to-date do not indicate the use of PFOS-related substances in industrial and household cleaning products. Based on information provided in product registers, the Swedish National Chemicals Inspectorate (KemI) has indicated that PFOS-related substances are still being used in Sweden for both industrial and household use (RPA & BRE, 2004).

Photographic Industry

PFOS-based chemicals are used for the following purposes in mixtures in coatings applied to photographic films, papers, and printing plates:

- Surfactants
- Electrostatic charge control agents;
- Friction control agents;
- Dirt repellent agents; and
- Adhesion control agents

According to the European Photographic Chemicals Industry Sector Group of CEFIC (EPCI), PFOS coating aids have a combination of surface-active properties that are unique and not found with any other type of coating aid. Only small quantities of PFOS materials are required to function as coating aids in imaging media. This property is important because the required addition of non-photoactive materials to coatings in significant quantities diminish the ability of
the imaging material to form the sharpest images. In short, thinner coatings make clearer, sharper images (RPA & BRE, 2004).

Photolithography and Semiconductors

**Photoresist**
Semiconductor manufacturing comprises up to 500 steps, of which there are four fundamental physical processes:
- Implant
- Deposition
- Etch
- Photolithography

Photolithography is the most important step towards the successful implementation of each of the other steps and, indeed, the overall process. It shapes and isolates the junctions and transistors; it defines the metallic interconnects; it delineates the electrical paths that form the transistors; and joins them together. Photolithography reportedly represents 150 of the total of 500 steps mentioned above (RPA & BRE, 2004).

Photolithography is also integral to the miniaturisation of semiconductors: Miniaturisation makes integrated circuits smaller, cheaper, faster and better, which is critical to continuing the electronic revolution (ESIA, 2003), and to EU manufacturers remaining competitive in the global market.

**Chemical amplification** makes the photolithography process more efficient and depends on a catalyst to chemically amplify the effect of the exposing light. The catalyst-precursor is called a photo-acid generator (PAG). The catalytic process is most effective when the photo acid produced from the PAG is a strong Bronsted acid. The first generations of 248 nm resist were formulated without PFOS in the PAG. PFOS PAG was included in formulations to improve performance.

**Antireflective Coatings**
A number of resist suppliers sell antireflective coatings (ARC), subdivided into Top (TARC) and Bottom (BARC) coatings and used in combination with deep ultra violet (DUV) photoresist. The process involves placing a thin, top coating on the resist to reduce reflective light, in much the same way and for the same purposes that eyeglasses and camera lenses are coated. TARC depends on good coating properties, water solubility, and an extreme refractive index. PFOS is present in TARC at a total of about 3% by weight.

**Hydraulic Fluids for the Aviation Industry**

Hydraulic fluids were initially used in aircraft to apply brake pressure. As larger and faster aircraft were designed, greater use of hydraulic fluids became necessary. An increase in the number of hydraulic fluid fires in the 1940s necessitated work towards developing fire resistant fluids. The first of these fluids was developed around 1948, when fire resistant hydraulic fluids based on phosphate ester chemistry were developed.

Technological advances in the aviation industry required continuous modifications to the hydraulic fluids to meet the specifications of the aircraft manufacturers. It was found that certain additives (such as the perfluorinated anionic surfactants) could alter the electrical potential at the metal surface and prevent its electrochemical oxidation. As a result, hydraulic fluids based on phosphate ester technology and incorporating additives based on perfluorinated anions are used in all commercial aircraft, and in many military and general aviation aircraft throughout the world, as well as by every airframe manufacturer.

It was discovered that the localised corrosion (referred to as erosion) was a result of a unique combination of factors and that perfluorinated anionic surfactants (such as PFOS) could inhibit erosion (and control damage) of mechanical parts of hydraulic systems that are used in all aircraft. These perfluorinated anions acts by altering the electrical potential at the metal
surface, thereby preventing the electrochemical oxidation of the metal surface under high fluid flow conditions (RPA & BRE, 2004).

**Metal Plating**

The main uses of PFOS-related substances in metal plating are for chromium plating, and anodising and acid pickling.

PFOS related substances lower the surface tension of the plating solution so that mist containing chromic acid from the plating activity is trapped in solution and is not released to air. Before the introduction of PFOS-based applications to control the emissions of hexavalent chromium, mist was controlled through extraction. Hexavalent chromium is a known carcinogen (RPA & BRE, 2004).

**Other**

There is information on other historical or current PFOS applications such as in pesticides, medical applications, mining and oil surfactants, flame retardants and in adhesives. Based on current understanding, these applications represent a minor part of known PFOS applications and are therefore not further elaborated in this dossier.

3. **Releases to the environment**

There is to date very limited information regarding the emissions and pathways of PFOS to the environment. The occurrence of PFOS in the environment is a result of anthropogenic manufacturing and use, since PFOS is not a naturally occurring substance.

Releases of PFOS and its related substances are likely to occur during their whole life cycle. They can be released at their production, at their assembly into a commercial product, during the distribution and industrial or consumer use as well as from landfills and sewage treatment plants after the use of the products (3M, 2000).

Manufacturing processes constitute a major source of PFOS to the local environment. During these processes volatile PFOS-related substances may be released to the atmosphere. PFOS and PFOS-related substances could also be released via sewage effluents (3M, 2000). High local emissions are supported by one study that showed extremely high concentrations of PFOS in wood mice collected in the immediate vicinity to 3M’s fluorochemical plant in Antwerpen, Belgium (Hoff et al., 2004). High concentrations of PFOS were also found in liver and blood from fish collected in the Mississippi River at the immediate vicinity of another of 3M’s fluorochemical plant, Cottage Grove in Minnesota (MPCA, 2006).

Fire training areas have also been revealed to constitute a source of PFOS emissions due to the presence of PFOS in fire-fighting foams. High levels of PFOS have been detected in neighbouring wetlands of such an area in Sweden (Swedish EPA, 2004) as well as in groundwater in the US close to a fire-training area (Moody et al., 2003).

An investigation on the uses of PFOS and PFOS-related compounds in Norway, 2005, show that approximately 90% of the total use is in fire extinguishers (provide REFERENCE). Estimated releases of PFOS related to fire extinguishers are at least 57 tonnes since 1980 to 2003 (2002; 13-15 tonnes). Remaining quantities of fire extinguisher foam in Norway are estimated to be a minimum of 1.4 million litres, which corresponds to an amount of approximately 22 tonnes PFOS. Releases from the municipal sector in Norway, 2002, were estimated to be 5-7 tonnes.

The use of PFOS in semiconductors is estimated to result in a release of 43 kg per year in the EU, according to the Semiconductur Industry Association (SIA) (provide date and Reference). This corresponds to 12 % of the total PFOS use in this application. PFOS released in the USA from semiconductors is estimated to be in the same range (SIA date/ref).

The releases of sulfonated perfluorochemicals, including PFOS or PFOS-related substances, from different product usages have been estimated (U.S Releases Estimation -1997). For example, garments treated with home-applied products, are expected to lose 73 % of the
treatment during cleaning over a 2-year life span. A loss of 34 % to air is expected from spray can products during use, while up to 12.5 % of the original content may be remaining in the cans at the time of disposal.

One route for PFOS and PFOS-related substances to the environment may be through sewage treatment plants (STPs) and landfills, where elevated concentrations have been observed compared to background concentrations. Once released from STPs, PFOS will partially adsorb to sediment and organic matter. A substantial amount of PFOS may also end up in agricultural soil, due to the application of sewage sludge. The primary compartments for PFOS are therefore believed to be water, sediment and soil (RIKZ, 2002).

Dispersion of PFOS in the environment is thought to occur through transport in surface water, or oceanic currents (Yamashita et al., 2005, Caliebe et al., 2004), transport in air (volatile PFOS-related substances), adsorption to particles (in water, sediment or air) and through living organisms (3M, 2003).

One major obstacle when trying to estimate the releases of PFOS to the environment is that PFOS can be formed through degradation of PFOS-related substances. The rate and the extent of that formation are presently unknown. In a study on Swedish STPs, higher concentrations of PFOS were found in the effluents compared to incoming sewage water, which could indicate that PFOS was formed through PFOS-related substances (Posner and Järnberg, 2004).

Environmental fate

1. Persistence
PFOS is extremely persistent. It does not hydrolyse, photolyse or biodegrade in any environmental condition tested (OECD 2002).
A study on the hydrolysis of PFOS in water has been performed following US-EPA OPPTS protocol 835.2210. The study was conducted at pH varying from 1.5 – 11.0 and at a temperature of 50°C, to facilitate hydrolysis, but did not indicate any degradation of PFOS. The half-life of PFOS was set to be greater than 41 years.

A study on the photolysis of PFOS in water following US-EPA OPPTS protocol 835.5270 has been conducted. No evidence of direct or indirect photolysis was observed under any of the conditions tested. The indirect photolytic half-life of PFOS at 25°C was calculated to be more than 3.7 years.
Biodegradation of PFOS has been evaluated in a variety of tests. Aerobic biodegradation of PFOS has been tested in activated sewage sludge, sediment cultures and soil cultures in several studies. Anaerobic biodegradation has been tested in sewage sludge. Neither of the studies demonstrated any signs of biodegradation.

The only known condition whereby PFOS is degraded is through high temperature incineration under correct operating conditions (3M, 2003). Potential degradation at low temperature incineration is unknown.

2. Bioaccumulation
It should be noted that PFOS does not follow the “classical” pattern of partitioning into fatty tissues followed by accumulation, which is typical of many persistent organic pollutants. This is because PFOS is both hydrophobic and lipophobic. Instead, PFOS binds preferentially to proteins in the plasma, such as albumin and β-lipoproteins (Kerstner-Wood et al., 2003), and in the liver, such as liver fatty acid binding protein (L-FABP; Luebker et al., 2002). Because of the unusual physical-chemical characteristics of PFOS, the mechanism of bioaccumulation probably differs from other POPs.

In a study following OECD protocol 305, the bioaccumulation of PFOS in bluegill sunfish (Lepomis macrochirus) has been tested. The whole-fish kinetic bioconcentration factor (BCFK) was determined to be 2796 (OECD, 2002).
In another study on rainbow trout (*Oncorhynchus mykiss*), a bioconcentration factor (BCF) in liver and plasma was estimated to be 2900 and 3100, respectively (Martin, et al., 2003).

When strictly looking at the BCF values, it is clear that these values are below the numeric BCF criteria in Stockholm Convention Annex D (the reported BCF values are below 5000) but, in this particular case, as noted above, the BCF numeric criteria may not adequately represent the bioaccumulation potential of the substance. Thus, monitoring data from top predators at various locations show highly elevated levels of PFOS and demonstrate substantial bioaccumulation and biomagnification (BMF) properties of PFOS. It is notable that the concentrations of PFOS found in livers of Arctic polar bears exceed the concentrations of all other known individual organohalogens (Martin et al., 2004). Based on the concentration of PFOS in predators (e.g., the polar bear) in relation to the concentration in their principal food (e.g., seals), hypothetical BMF values can be calculated. Such data are reported in Table 4. It should be noted that there are uncertainties in these comparisons. Even if either liver or blood concentrations are compared in two species, species differences in specific protein binding in that particular compartment may affect the concentration in the organ without having affected the whole-body concentration of the substance.

Table 4. Measured concentrations of PFOS in biota from various locations. Calculated BMF is shown where applicable.

<table>
<thead>
<tr>
<th>Species and Location</th>
<th>Concentrations of PFOS</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Polar Bear, Canadian Arctic</td>
<td>- Concentrations of PFOS in liver (1700 – &gt; 4000 ng/g) exceeding all other individual organohalogens. &lt;br&gt; - BMF &gt; 160 based on concentrations in Arctic seals.</td>
<td>Martin et al., 2004.</td>
</tr>
<tr>
<td>• Arctic fox, Canadian Arctic</td>
<td>- Very high concentrations of PFOS in liver (6.1 - 1400 ng/g)</td>
<td>Martin et al., 2004.</td>
</tr>
<tr>
<td>• Mink, US</td>
<td>- Very high concentrations of PFOS in liver (40 - 4870 ng/g). &lt;br&gt; - BMF = 22 based on data from fish in the same area. &lt;br&gt; - Another mink study also show very high concentrations of PFOS in liver (1280 - 59 500 ng/g, mean 18 000 ng/g.) &lt;br&gt; - BMF ~145 to ~4000 based on data from their prey such as crayfish (whole body), carp (muscle) and turtles (liver</td>
<td>Giesy and Kannan, 2001 &lt;br&gt; Kannan et al., 2005</td>
</tr>
<tr>
<td>• Seal in the Bothnian Sea, Finland</td>
<td>- Very high concentrations of PFOS in liver (30 – 1100 ng/g). &lt;br&gt; - BMF &gt; 60 based on data from salmon in the same area.</td>
<td>Kannan et al., 2002.</td>
</tr>
</tbody>
</table>

In a study by Kannan et al., 2005, the whole body BCF for round gobies (provide scientific name) was calculated to be approximately 2400, which is comparable with laboratory data. PFOS concentrations in fish (whole body of round gobies) compared to concentrations in liver of salmon results in BMFs of approximately 10-20. In bald eagles, the mean PFOS concentration in the livers, 400 ng/g ww, gives a BMF of four to five when compared to fish at
higher trophic levels in the study. For mink, BMFs from 145 to 4000 can be calculated when based on the mean liver concentration, 18 000 ng/g ww, compared to their prey items such as crayfish (whole body), carp (muscles) and turtles (liver).

In general, data show that animals at higher trophic levels have higher concentrations of PFOS than animals at lower trophic levels, indicating that biomagnification is taking place. For instance, a trophic magnification factor (TMF) of 5.9 was calculated for PFOS based on a pelagic food web including: one invertebrate species, Mysis; two forage fish species, rainbow smelt and alewife; and a top predator fish species, lake trout. A diet-weighted bioaccumulation factor of approximately 3 was determined for the trout (Martin et al., 2004).

Morikawa et al. (2005) showed a high bioaccumulation in turtles. Results from a study performed by Tomy et al. (2004) indicated that PFOS biomagnified in an eastern Arctic marine food web (liver concentrations of PFOS were used for seabirds and marine mammals). Houde et al. (2006) showed PFOS biomagnification in the Atlantic ocean bottlenose dolphin food web.

A study by Bossi et al. (2005) further supports that biomagnification is taking place. In this study, a preliminary screening of PFOS and related compounds has been performed in liver samples of fish, birds and marine mammals from Greenland and the Faroe Islands. PFOS was the predominant fluorochemical in the biota analyzed, followed by perfluorooctane sulfonamide (PFOSA). The results from Greenland showed a biomagnification of PFOS along the marine food chain (shorthorn sculpin < ringed seal < polar bear).

The fact that PFOS binds to proteins leads to the relevant question -- at what concentrations of PFOS will the binding sites on these proteins be saturated? Serum albumin is most likely the binding pool of PFOS (Jones et al., 2003) and several studies have been carried out with regard to bioconcentration in plasma. In Ankley et al. (2005), the bioconcentration in fish was studied at concentrations of PFOS in water up to 1 mg/L; the concentration of PFOS in water and plasma followed an almost linear relationship in the doses tested up to 0.3 mg/l without any signs of saturation (1 mg/l was not tested due to mortality at that dose). This is far above environmentally relevant concentrations.

In a study by 3M (2003), the bioconcentration factor (BCF) in whole fish was determined to be approximately 2800 at a PFOS concentration of 86 µg/l, based on calculations of uptake and depuration of PFOS. Steady-state levels were attained after 49 days of exposure. Depuration occurred slowly and 50% clearance for whole fish tissues was estimated to be 152 days. Due to mortality, a BCF could not be calculated for the other concentration used, 870 µg/l. Thus, it is not likely that saturation of serum protein binding sites will limit the bioconcentration of PFOS in fish. We are not aware of similar data in mammals, but considering the high level of bioaccumulation observed in mammals, and that mammalian serum contains high concentrations of protein, one may speculate that saturation of binding sites are not likely to limit the bioaccumulation of PFOS in mammals either.

3. Long range environmental transport
The potassium salt of PFOS has a measured vapour pressure of 3.31 x 10^{-4} Pa (OECD, 2002). Due to this vapour pressure and a low air-water partition coefficient (< 2x10^{-6}), PFOS itself is not expected to volatilise significantly. It is therefore assumed to be transported in the atmosphere predominantly bound to particles, because of its surface-active properties, rather than in a gaseous state.

It should be noted that some of the PFOS-related substances have a considerably higher vapour pressure than PFOS itself, and are as a result more likely to be volatile. This may allow a wider transport of PFOS-related substances through air than is possible for PFOS itself. Examples of these are: EtFOSE alcohol, MeFOSE alcohol, MeFOSA, EtFOSA, and FOSA. These precursors to PFOS could evaporate into the atmosphere. Once in the atmosphere they can remain in gas phase, condense on particles present in the atmosphere and be carried or settle out with them, or be washed out with rain (3M, 2000). Martin et al. (2002) measured the air in Toronto and Long Point, Ontario for some precursors of PFOS. They found an average N-MeFOSE alcohol concentration of 101 pg/m^3 in Toronto and 35
The average concentrations of N-EtFOS alcohol were 205 pg/m$^3$ in Toronto and 76 pg/m$^3$ in Long Point.

PFOS has been detected in rainwater from an urban center in Canada with a concentration of 0.59 ng/L. Whether or not PFOS originates from precursors either being transported and subsequently wet deposited and degraded to PFOS, or atmospherically degraded and then wet deposited, is unclear. Measurements of potential precursors for PFOS were not performed in this study (Loewen et al., 2005).

The atmospheric half-life of PFOS is expected to be greater than two days. This statement, while not specifically tested, is based on the fact that PFOS has exhibited extreme resistance to degradation in all tests performed. However, an atmospheric half-life of 114 days has been calculated for PFOS using an AOP computer modeling program v1.91 (RER, 2004, Environment Agency).

The indirect photolytic half-life of PFOS at 25°C has been estimated to be more than 3.7 years (OECD, 2002).

PFOS has been measured in a wide range of biota in the Northern Hemisphere such as the Canadian Arctic, Sweden, the US and the Netherlands. In a study by Martin et al (2004), the levels of PFOS were measured in liver samples from biota in the Canadian Arctic and were found in the vast majority of the species examined. The presence of PFOS in Arctic biota, far from anthropogenic sources, demonstrates the potential of PFOS for long-range transport. The mechanisms of this transport are not known, but it could be due to the transport of volatile PFOS-related substances that eventually degrade to PFOS.

A recent study performed with rainbow trout (Onchorhynchus mykiss) liver microsomes has demonstrated that N-ethyl perfluorooctanesulfonamide (N-EtPFOSA) is a precursor of PFOS in fish (Tomy et al., 2004a). These findings combined with the recent measurements of concentrations up to 92.8 ± 41.9 ng/g wet weight of N-EtPFOSA in aquatic organisms from Arctic regions (Tomy et al., 2004b) strengthen the hypothesis that perfluorinated sulfonamides are one of the volatile precursors of PFOS transported over long distances to the Arctic. However, the hypothesis that these volatile precursors reach the Arctic latitudes by atmospheric transport has not yet been confirmed by atmospheric measurements (Bossi et al., 2005).

3. **Exposure**

**Measured environmental levels**

A screening study was assigned by the Swedish Environmental Protection Agency (Swedish EPA) and performed by ITM, Institute of Applied Environmental Research, on the levels of PFOS in the Swedish environment (Swedish EPA, 2004). The results showed highly elevated levels of PFOS in a wetland in the vicinity of a fire drill area with a declining gradient out in the adjacent bay (2.2 – 0.2 µg/L). Elevated levels were also detected outside sewage treatment plants (STPs) and landfills. Effluents from STPs contained levels of PFOS up to 0.020 µg/L and leachate levels from landfills were between 0.038 – 0.152 µg/L.

The occurrence of PFOS and other perfluoroalkyl sulfonate substances in open ocean waters such as the Atlantic and the Pacific Ocean have been investigated. The results showed that PFOS is present in central to western Pacific Ocean regions in concentrations ranging from 15 – 56 pg/L, comparable to the concentrations in the mid-Atlantic ocean. These values appear to be the background values for remote marine waters far from local sources (Taniyasu et al., 2004). PFOS was also detected in oceanic waters in several coastal seawaters from Asian countries (Japan, Hong Kong, China, and Korea) at concentrations ranging from 1.1 - 57 700 pg.L$^{-1}$ (Yamashita et al., 2005). PFOS was also observed in the North Sea (estuary of the river Elbe, German Bight, southern and eastern North Sea) (Caliebe et al., 2004).

Studies in the US have identified the presence of PFOS in surface water and sediment downstream of a production facility, as well as in wastewater treatment plant effluent, sewage sludge and landfill leachate at a number of urban centres in the US (3M Multi City study,
reviewed in OECD (2002) and 3M (2003). Four of the cities (Decatur (AL), Mobile, Columbus (GA), Pensacola) were cities that have manufacturing or industrial use of fluorochemicals; two of the cities (Cleveland (TN), Port St. Lucie) were control cities that do not have significant fluorochemical activities. The ranges of PFOS levels in these cities are provided in Table 5.

### Table 5. Environmental Levels of PFOS in Six US Urban Centres in the US (from OECD, 2002)

<table>
<thead>
<tr>
<th>Medium</th>
<th>Range of PFOS levels (µg/L or µg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Municipal wastewater treatment plant effluent</td>
<td>0.041 - 5.29</td>
</tr>
<tr>
<td>Municipal wastewater treatment plant sludge</td>
<td>0.2 - 3.120 (dry weight)</td>
</tr>
<tr>
<td>Drinking water</td>
<td>ND - 0.063</td>
</tr>
<tr>
<td>Sediment</td>
<td>ND - 53.1 (dry weight)</td>
</tr>
<tr>
<td>Surface water</td>
<td>ND - 0.138</td>
</tr>
<tr>
<td>‘Quiet’ water</td>
<td>ND - 2.93</td>
</tr>
</tbody>
</table>

**Note:** ND: not detected

The control cities’ samples generally inhabited the lower end of the above ranges, except for the municipal wastewater treatment plant effluent and sludge findings for one of the control cities (Cleveland), which were intermediate in their ranges, and the ‘quiet’ water samples at control city (Port St. Lucie), which were the highest. In Canada, suspended sediment samples were collected annually at Niagara-on-the-Lake in the Niagara River over a 22 year period (1980-2002). PFOS concentrations ranged from 5 to 1100 pg.g\(^{-1}\) (Furdui et al., 2005). Preliminary findings suggest that PFOS concentrations increased during the study period from < 400 pg.g\(^{-1}\) in the early 1980s to > 1000 pg.g\(^{-1}\) in 2002.

Samples of effluent from fifteen representative industry sectors have been analysed for PFOS (Hohenblum et al, 2003). The industry sectors were printing (1 site), electronics (3), leather, metals, paper (6), photographic and textiles (2). The PFOS levels ranged from 0-2.5 µg/L (2.5 µg/L for leather, 0.120 µg/l for metal, 0.140-1.2 µg/l at four paper sites, 1.2 µg/l for photographic, not found in textiles or electronics).

Groundwater from below an air force base in Michigan, US, has been sampled (Moody et al, 2003). Fire fighting foams containing PFOS had been used there in training exercises from the 1950s to 1993 when the base was decommissioned. The groundwater was found to contain PFOS, at levels from 4 - 110 µg/l.

Sixteen Great Lakes water samples (eight locations) were analysed for perfluorooctane surfactants. PFOS was present in all samples with a concentration range of 21-70 ng/L. Three PFOS precursors were also found in the water samples. N-EtFOSAA (4.2-11 ng/L) and sulfonamide (FOSA) (0.6 -1.3 ng/L) were present in nearly all samples while PFSOulfinate was identified at six out of eight locations (2.2-17 ng/L) (Boulanger et al, 2004). PFOS was detected in surface water as a result of a spill of fire-fighting foam from the Toronto International Airport into nearby Etobicoke Creek. Concentrations of PFOS ranging from <0.017 to 2210 µg.L\(^{-1}\) were detected in creek water samples over a 153-day sampling period. PFOS was not detected at the upstream sample site (Moody et al. 2002).

PFOS and related fluorochemicals have been detected in animals in a number of studies in a variety of locations around the globe. Generally, the highest concentrations are found in top predators in food chains containing fish.

Martin et al. (2004) measured the levels of PFOS in liver samples from biota in the Canadian Arctic. PFOS was found in the vast majority of the samples and higher levels were found in animals at the top of the food chain. The highest levels were found in polar bear, with a mean level of 3100 ng/g from seven animals (maximum value > 4000 ng/g). These levels of PFOS in the liver exceed the levels of all other known individual organohalogenes. High levels of PFOS were also found in the arctic fox. FOSA, a precursor to PFOS, was also found in most of the samples. The concentration of FOSA was higher than that of PFOS in fish, but not in mammals. This could indicate that FOSA has been metabolised to PFOS in mammals and
the high concentrations may be the result of both direct exposure to PFOS and metabolism from FOSA.

Kannan and Giesy (2002) have summarised results of analyses on archived tissue samples. The tissues analysed came from marine mammals, birds, fish, reptiles and amphibians from around the worlds, including the Arctic and Antarctic Oceans. Samples collected in the 1990s were used. Around 1700 samples were analysed, with concentrations in liver, egg yolk, muscle or blood plasma determined. The detection limit varied from 1 ng/g to 35 ng/g wet weight. A summary of the results is shown in Table 6.

**Table 6.** Maximum concentrations of PFOS in various species as well as frequency of detection. Based on Kannan and Giesy (2002)

<table>
<thead>
<tr>
<th>Species</th>
<th>Maximum concentration ng/g wet wt</th>
<th>Frequency of Detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marine mammals</td>
<td>1520</td>
<td>77%</td>
</tr>
<tr>
<td>Mink and otter</td>
<td>4900</td>
<td>100%</td>
</tr>
<tr>
<td>Birds</td>
<td>2570</td>
<td>60%</td>
</tr>
<tr>
<td>Fish</td>
<td>1000</td>
<td>38%</td>
</tr>
</tbody>
</table>

PFOS was detectable in most of the samples, including those from remote marine locations, at concentrations >1 ng/g. The authors compared the results from remote areas with those from more industrial locations and noted that PFOS is widely distributed in remote regions, including the Polar Regions, but that the levels found in more urban and industrial areas (e.g. the Baltic, Great Lakes) are several times higher. The tissues of fish-eating birds in Canada, Italy, Japan and Korea all contained detectable levels of PFOS, suggesting that they are exposed through the fish they consume. A summary of several studies is given in Table 7.

**Table 7.** Monitored Levels of PFOS in Animals (data from selected studies, based on OECD, 2002)

<table>
<thead>
<tr>
<th>Description</th>
<th>Reference</th>
<th>Reported Highest Concentrations (Max, Mean)</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global monitoring survey of marine mammals (Florida, California, Alaska, northern Baltic Sea, Mediterranean Sea, Arctic, Sable Island (Canada))</td>
<td>A</td>
<td>Bottlenose dolphin (liver, n = 26): Max: 1520 ng/g wet wt. Mean: 420 ng/g wet wt.</td>
<td>Florida</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ringed seal (liver, n = 81): Max: 1100 ng/g wet wt. Mean: 240 ng/g wet wt.</td>
<td>Northern Baltic Sea</td>
</tr>
<tr>
<td>Survey of mammals, birds and fish in the Canadian Arctic</td>
<td>B</td>
<td>Polar bear (liver, n = 7): Max: &gt; 4000 ng/g wet wt. Mean: 3100 ng/g wet wt.</td>
<td>Canadian Arctic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arctic fox (liver, n = 10): Max: 1400 ng/g wet wt. Mean: 250 ng/g wet wt.</td>
<td></td>
</tr>
<tr>
<td>Survey of fish (US, Europe, North Pacific Ocean, Antarctic)</td>
<td>C</td>
<td>Fish (muscle, n = 172): Max: 923 ng/g wet wt. Mean: 40 ng/g wet wt.</td>
<td>Belgian estuary</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carp (muscle, n = 10): Max: 296 ng/g wet wt. Mean: 120 ng/g wet wt.</td>
<td>US Great Lakes</td>
</tr>
<tr>
<td>Survey of fish-eating birds (US, Baltic Sea, Mediterranean Sea, Japanese coast, Korean coast)</td>
<td>D</td>
<td>Bald eagle (plasma, n = 42): Max: 2570 ng/mL Mean: 520 ng/mL</td>
<td>Midwest US</td>
</tr>
<tr>
<td>Description</td>
<td>Reference</td>
<td>Reported Highest Concentrations (Max, Mean)</td>
<td>Location</td>
</tr>
<tr>
<td>-------------</td>
<td>-----------</td>
<td>-------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Survey of mink and river otter in the US</td>
<td>E</td>
<td>Mink (liver, n = 77): Max: 4870 ng/g wet wt. Mean: 1220 ng/g wet wt.</td>
<td>US</td>
</tr>
<tr>
<td></td>
<td></td>
<td>River otter (liver, n = 5): Max: 994 ng/g wet wt. Mean: 330 ng/g wet wt.</td>
<td>US</td>
</tr>
<tr>
<td>Survey of oysters in the US (Chesapeake Bay &amp; Gulf of Mexico)</td>
<td>F</td>
<td>Oyster (Whole body, n = 77) Max: 100 ng/g wet wt. Mean: 60 ng/g wet wt.</td>
<td>US</td>
</tr>
<tr>
<td>Fish samples upstream and downstream of 3M facility in Decatur, Alabama, US</td>
<td>G</td>
<td>Fish (whole body): Mean (upstream): 59.1 µg/kg wet wt. Mean (downstream): 1332 µg/kg wet wt.</td>
<td>Decatur, US</td>
</tr>
<tr>
<td>Swedish urban and background fish samples</td>
<td>H</td>
<td>Perch: 3 - 8 ng/g (urban sites in the vicinity of municipal STPs); 20-44 ng/g in Lake Mälaren and near Stockholm</td>
<td>Sweden (Lake Mälaren)</td>
</tr>
</tbody>
</table>


Concentrations of PFOS in guillemot (*Uria aalge*) eggs from Stora Karlsö in the Baltic Sea have been measured retrospectively from 1968 to 2003 (Holmström et al, 2005). The results shown in Figure 2 display a trend of increasing concentrations since 1968 (17 – 623 ng/g).

![Temporal trend in Guillemot eggs, Stora Karlsö](image)

**Figure 2.** Measured concentrations of PFOS in Guillemot (*Uria aalge*) eggs sampled at Stora Karlsö in the Baltic Sea between the years 1968 – 2003. The graph is taken from the report “Screening av perfluorerade ämnen” by the Swedish EPA, Environmental Assessment Department (2004).

### 2.3.1 Bioavailability

Studies on fish have shown that PFOS has bioconcentrating properties. In studies on bluegill sunfish (*Lepomis macrochirus*) and rainbow trout (*Oncorhynchus mykiss*) bioconcentration factors (BCFs) have been estimated to be 2796 (whole fish) as well as 2900 (liver) and 3100
(plasma), respectively. The major route of uptake is believed to be through the gills (Martin et al., 2003)

Since PFOS is believed to be released to the environment mainly through water from STPs, one major route for PFOS into food chains could be through fish. PFOS has shown a high oral uptake (95%) within 24 hours in the gastro-intestinal (GI) tract in studies on rats (OECD, 2002). Taken together, this could constitute the basis of the highly elevated levels that have been observed in top predators in food chains containing fish.

This could also be confirmed by two separate human monitoring studies on the Swedish population where the levels of PFOS in whole blood was higher (27.2 ng/g, 3.0 – 67, n = 10) in females with a high consumption of fish (Berglund, 2004) compared to samples from females in the general population (17.8 (ng/g, 4.6 – 33, n = 26) (Kärnman et al., 2004).

In humans, the highest concentrations of PFOS have been detected in workers at 3M’s manufacturing plant for perfluorochemicals in Decatur, US, where the levels in serum in the last year of measurement (2000) ranged between 0.06 – 10.06 ug/g (OECD, 2002).

In a study of the general population, blood samples from families including three generations living in 12 European countries were tested for a large number of chemicals including PFOS and perfluorooctane sulfonamide (FOSA). PFOS was present in 37 of 38 samples with concentrations from 0.36 to 35.3 ng/g blood, while FOSA was present in 36 of 38 samples with concentrations from 0.15 to 2.04 ng/g blood (WWF, 2005).

4. Hazard assessment for endpoints of concern

1. Toxicity

Evidence of the toxicity of PFOS is available from acute, sub-chronic and chronic exposures to rats, sub-chronic exposures to monkeys, and a two-generation study on rats. Results are available from reproductive and teratogenicity studies on rats and rabbits. Details of these studies are not included here, they can be found in the assessment made by OECD (2002).

The most relevant data for this risk profile are:

- A 90-day study on rhesus monkeys exposed to PFOS potassium salt via gavage at the doses 0, 0.5, 1.5 and 4.5 mg/kg bw/day. At 4.5 mg/kg bw/day all monkeys (4) died or were sacrificed in moribound condition. No deaths were observed at 0.5 or 1.5 mg/kg bw/day, but there were signs of gastrointestinal toxicity. A NOAEL could not be established since the lowest dose was a LOAEL. The results of this test show that PFOS fulfils the EU criteria for classification as Toxic, with the risk phrase R48.

- A 90-day oral repeated dose toxicity study in rats that were fed diets containing 0, 30, 100, 300, 1000 and 3000 mg PFOS potassium salt per kg diet. All rats died when fed diets containing 300 mg/kg PFOS and above (equivalent to 18 mg/kg bw/day and above). At 100 mg/kg (6 mg/kg bw/day), 50% (5/10) of the animals died. All rats receiving diets containing 30 mg/kg PFOS (2.0 mg/kg/day) survived until the end of the study, but small changes in body and organ weights were reported. Since the lowest dose tested was a LOAEL, a NOAEL could not be established. Also in rats, a classification of chronic toxicity for PFOS (R 48 according to EU criteria) is warranted.

- A two-generation reproductive toxicity study on rats that were fed PFOS potassium salt via gavage at the doses 0.1, 0.4, 1.6,and 3.2 mg/kg bw/day. At the doses 1.6 and 3.2 mg/kg bw/day a significant reduction in the viability of the F1 generation was observed. In the 1.6 mg/kg bw/day group, 34% (86/254) of the F1 pups died within four days after birth. In the 3.2 mg/kg bw/day group, 45% (71/156), of the F1 pups died within one day after delivery. None of these pups survived beyond day 4. A new study by Luebker et al. (2005) supports these results.

Maternal toxicity at 1.6 and 3.2 mg/kg bw/day was manifested as reduced food consumption, body weight gain, and terminal bodyweight. Localised alopecia was also observed at 3.2 mg/kg bw/day. The LOAEL in this study was 0.4 mg/kg bw/day based on significant
reductions in pup weight gain in the F1 generation animals. The NOAEL was 0.1 mg/kg bw/day.

A study by Grasty et al. (2003) concluded that exposure to PFOS late in gestation is sufficient to induce 100% pup mortality and that the causative factor may be inhibition of lung maturation.

2.4.2 Ecotoxicity

Environmental toxicity data for PFOS is predominantly found for aquatic organisms such as fish, invertebrates and algae.

PFOS has shown moderate acute toxicity to fish. The lowest observed LC$_{50}$ (96h) was estimated to be 4.7 mg/l in a study where Fathead minnow (Pimephales promelas) were exposed to the lithium salt of PFOS. The lowest NOEC, 0.3 mg/l, has been observed in Pimephales promelas at prolonged exposure (42d) and was based on mortality (OECD, 2002). By this toxicity to fish PFOS fulfills the EU criteria for the classifications R 51 with the risk phrase “toxic to aquatic organisms” and R 53 “may cause long-term adverse effects in the aquatic environment.”

The lowest LC$_{50}$ (96h) for aquatic invertebrates has been observed in the Mysid shrimp (Mysidopsis bahia) and was estimated to be 3.6 mg/l. The lowest NOEC value has been observed in Mysidopsis bahia at 0.25 mg/l (OECD, 2002).

A study by Macdonald et al. (2004) reported a 10 day NOEC of 0.0491 mg/L for the growth and survival of the aquatic midge (Chironomous tentans).

The most sensitive algae appear to be the green algae Pseudokirchnerilla subcapitata with a LC$_{50}$ (96h, cell density) of 48.2 mg/L. The lowest NOEC value for algae was determined in the same study for Pseudokirchnerilla subcapitata, 5.3 mg/L (Boudreau et al., 2003).

Mallard and bobwhite quail were exposed to PFOS in feed for 21 weeks and a variety of endpoints examined including changes in adult body and organ weights, feed consumption rate, fertility, hatchability, and offspring survival. The LOEC of 10 ppm (10 mg/kg diet) PFOS for mallards (Anas platyrhyncu) included reduced testes size, decreased spermatogenesis and survivability of hatchlings (US EPA OPPT AR 226-1738). At this dose, the level of PFOS in serum and mallard liver were 87.3 µg/mL serum and 60.9 µg/g liver. (US EPA OPPT AR 226-1735). There is uncertainty in the study as no NOAEL was reported. For quail (Colinus virginianus), at 10 ppm (10 mg/kg) in diet, minor overt signs of toxicity were observed in adults, there was an increase in liver weight (females) an increase in the incidence of small testes size (males), and reduction in survivability in quail chicks as a percentage of eggs set. Concentrations in serum and liver of adult quail females was 84 µg.mL$^{-1}$ serum, 8.7 µg.mL$^{-1}$ and 4.9 µg.kg$^{-1}$ wet weight liver, respectively and in adult quail males 141 µg.mL$^{-1}$ and 88.5 µg.kg$^{-1}$, respectively (US EPA OPPT AR226-1831).

SYNTHESIS OF THE INFORMATION

Perfluorooctane sulfonate (PFOS) is a fully fluorinated anion, which is commonly used as a salt in some applications or incorporated into larger polymers. Due to its surface-active properties it has historically been used in a wide variety of applications, typically including fire fighting foams and surface resistance/repellency to oil, water, grease or soil. PFOS can be formed by degradation from a large group of related substances, referred to as PFOS-related substances (see definition on page 3). According to available data, PFOS meets the criteria for the potential for long-range transport. This is evident through monitoring data showing highly elevated levels of PFOS in various parts of the northern hemisphere. It is especially evident in the Arctic biota, far from anthropogenic sources. PFOS also fulfills the specific criteria for atmospheric half-life.
PFOS fulfills the criteria for toxicity. It has demonstrated toxicity towards mammals in sub-chronic repeated dose studies at low concentrations, as well as rat reproductive toxicity with mortality of pups occurring shortly after birth, probably caused by inhibition of lung maturation. PFOS is toxic to aquatic organisms with mysid shrimp (or Chironomus tentans- see Macdonald et al. 2004) being the most sensitive organism.

PFOS is extremely persistent. It has not showed any degradation in tests of hydrolysis, photolysis or biodegradation in any environmental condition tested. The only known condition whereby PFOS is degraded is through high temperature incineration.

With regard to bioaccumulation potential, PFOS meets the criterion given the highly elevated concentrations that have been found in top predators such as the polar bear, seal, bald eagle and mink. Based on the concentrations found in their prey, high BMFs have been estimated for these predators. BCF values in fish, although (rather) high do not in themselves meet the specific numeric criteria. However, due to the properties of PFOS, which binds preferentially to proteins in non-lipid tissues, application of numeric criteria for BCF or BAF, which are derived based on consideration of lipid-partitioning substances, may be inappropriate for PFOS. Most notable and alarming are the high concentrations of PFOS that have been found in Arctic animals, far from anthropogenic sources.

<table>
<thead>
<tr>
<th>Table 8. POP characteristics of PFOS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criterion</td>
</tr>
</tbody>
</table>
| Potential for Long-Range Environmental Transport | Yes | Vapour pressure = $3.31 \times 10^{-4}$ Pa  
Atmospheric half life > 2 days  
(estimated value based on photolytic half life > 3.7 years) |
| Toxicity                           | Yes | Sub-chronic exposure: Mortality in monkeys at 4.5 mg/kg bw/day.  
Reproductive toxicity: mortality in rat pups at 1.6 mg/kg bw/day.  
Acute toxicity to Mysid shrimp (Mysisopsis bahia): LC$_{50}$ (96h) = 3.6 mg/L  
Acute toxicity to fish, Fathead minnow (Pimephales promelas): LC$_{50}$ = 4.7 mg/L |
| Persistence                        | Yes | Extremely persistent. No degradation recorded in chemical or biological tests |
| Bioaccumulation                    | Yes | Found in highly elevated concentrations in top predators. Calculated hypothetical BMFs = 22 - 160.  
BCF in fish = 2796 - 3100. |

Due to their intrinsic properties, PFOS and its related substances have been used in a wide variety of applications. While historically, PFOS and PFOS-related substances have been used in eight different sectors as shown in Section 2.1.2. above, the present use in industrialized countries seems to be limited to five sectors, see 2.1.2. It is not known whether this also refers to the global use.

PFOS and PFOS-related substances can be released to the environment at their manufacture, during their use in industrial and consumer applications and from disposal of the chemicals or of products or articles containing them after their use.

The rate and the extent of the formation of PFOS from its related chemicals are largely unknown. Lack of data makes it very difficult to estimate the net contribution of the transformation of each of the PFOS-related substances to the environmental loadings of PFOS. However, based on its extreme stability it is expected that PFOS will be the final degradation product of all PFOS-related substances.
CONCLUDING STATEMENT

According to the available data, PFOS is extremely persistent in the environment. Due to its physical and chemical properties and considerably long half-life and based on findings in environmental samples in distant locations e.g. the Arctic, it can be assumed that PFOS/PFOS-related substances can be transported long distances in air or oceanic currents, far from its sources. Laboratory studies indicate PFOS is associated with serious harmful effects in mammals and aquatic organisms. Studies in the field have clearly indicated biomagnification in several food webs.

The voluntary phase out of PFOS production by the major producer in the USA has led to a significant reduction in the current use of PFOS-related substances. However, it can be assumed that it is still produced in some countries and there is evidence that it continues to be used in many countries. As PFOS-related substances can move to locations far from its sources, measures taken by single countries or groups of countries are not sufficient to prevent exposure that may lead to adverse effects. Regional action has already been considered necessary and PFOS is nominated under the CLRTAP Protocol on POPs. Due to the harmful POP properties and risks related to its possible continuing production and use, global action is warranted to eliminate the pollution caused by PFOS.
References:


1 Tomy, G. T.; Tittlemier, S. A.; Palace, V. P.; Budakowski, W. R.; Braekevelt, E.; Brinkworth, L.; Friesen, K. Biotransformation of N-ethyl perfluorooctanesulfonamide by rainbow trout (Onchorhynchus mykiss) liver microsomes. Environ. Sci. Technol. 2004a, 38, 758-762


RISK MANAGEMENT EVALUATION

Executive summary

PFOS was proposed as a POPs candidate by Sweden in 2005. The 2nd meeting of the POPs Review Committee decided that PFOS is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

As PFOS is both an intentionally produced substance and an unintended degradation product of related chemicals, under the Convention the most adequate control measures would be listing in Annex A. To allow for some critical uses of PFOS-related substances, which may ultimately degrade to PFOS, an acceptable purpose/specific exemption for use of PFOS and production of PFOS as an intermediate only as required to produce other chemical substances designated for these critical uses could be given together with a detailed description of the conditions for these uses in a new Part III to Annex A. Stockpiles and waste containing PFOS or PFOS-related substances would be subject to the provisions in Article 6.

1. Introduction

1.1 Chemical identity of the proposed substance

On July 14, 2005, the government of Sweden made a proposal for listing perfluorooctane sulfonate (PFOS) and 96 PFOS-related substances in Annex A of the Stockholm Convention on Persistent Organic Pollutants (POPs).

1.1.1. PFOS

Chemical name: Perfluorooctane Sulfonate (PFOS)

Molecular formula: C8F17SO3-

PFOS, as an anion, does not have a specific CAS number. The parent sulfonic acid has a recognised CAS number (CAS No. 1763-23-1). Some examples of its commercially important salts are listed below:

- Potassium salt (CAS No. 2795-39-3)
- Diethanolamine salt (CAS No. 70225-14-8)
- Ammonium salt (CAS No. 29081-56-9)
- Lithium salt (CAS No. 29457-72-5)

Structural formula:

![Figure 1. Structural formula of PFOS shown as its potassium salt](image)

1.1.2. Issues regarding PFOS-related substances

PFOS is a fully fluorinated anion, which is commonly used as a salt or incorporated into larger polymers. PFOS and its closely related compounds, which contain PFOS impurities or substances which can give rise to PFOS, are members of the large family of perfluoroalkyl sulfonate substances. In its regulatory measures on PFOS, the European Union (EU) has addressed all molecules having the following molecular formula: C8F17SO2Y, where Y = OH, metal or other salt, halide, amide and other derivatives including polymers (European Union, 2006).

The physical and chemical properties of the potassium salt of PFOS are listed in Table 2.

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance at normal temperature and pressure</td>
<td>White powder</td>
</tr>
</tbody>
</table>

(Data from OECD, 2002, unless otherwise noted)
Molecular weight 538 g/mol
Vapour Pressure 3.31 x 10^-4 Pa
Water solubility in pure water 519 mg/L (20 ± 0.5°C)
680 mg/L (24 - 25°C)
Melting point > 400 °C
Boiling point Not measurable
Log KOW Not measurable
Air-water partition coefficient < 2 x 10^-6 (3M, 2003a)
Henry’s Law Constant 3.09 x 10^-9 atm m3/mol pure water

PFOS can be formed (by environmental microbial degradation or by metabolism in larger organisms) from PFOS-related substances, i.e., molecules containing the PFOS-moiety depicted in Figure 1. Although the ultimate net contribution of individual PFOS-related substances to the environmental loadings of PFOS cannot be predicted readily, there is a potential that any molecule containing the PFOS moiety could be a precursor to PFOS. This is further supported by modelling the fate of perfluorinated chemicals (PFCs) in the environment. There was a trend towards more bioaccumulative and more toxic products. Perfluorooctanoic acid and perfluorooctane sulfonate were predicted to be the persistent biodegradation products of 17 and 27% of the perfluorinated sulphonic acid and carboxylic acid containing compounds, respectively. (Canada, 2007).

The majority of PFOS-related substances are polymers of high molecular weights in which PFOS is only a fraction of the polymer and final product (OECD, 2002). PFOS-related substances have been defined somewhat differently in different contexts and there are currently a number of lists of PFOS-related substances (Table 3). The lists contain varying numbers of PFOS-related substances that are thought to have the potential to break down to PFOS. The lists overlap to varying extents depending on the substances under consideration and the overlap between national lists of existing chemicals.

Table 3. Number of PFOS-related substances as proposed by UK – DEFRA, US – EPA, OECD, OSPAR, and Canada

<table>
<thead>
<tr>
<th>Source</th>
<th>Number of PFOS-related substances</th>
</tr>
</thead>
<tbody>
<tr>
<td>RPA and BRE (2004)</td>
<td>96</td>
</tr>
<tr>
<td>OECD (2002)</td>
<td>1721 (22 classes of perfluoroalkyl sulfonate substances)</td>
</tr>
<tr>
<td>OSPAR (2002)</td>
<td>48</td>
</tr>
<tr>
<td>Environment Canada (2006)</td>
<td>57</td>
</tr>
</tbody>
</table>

A large number of substances may give rise to PFOS and thus contribute to the contamination problem. DEFRA in the United Kingdom (RPA and BRE, 2004) has recently proposed a list of 96 PFOS-related substances. However, the properties of the 96 substances have not generally been determined. According to 3M (submission to the secretariat of Stockholm Convention (SC), 2006), they may have very different environmental characteristics such as solubility, stability and ability to be absorbed or metabolised. Nevertheless, the document by the United Kingdom infers that all of these substances would give rise to the final degradation product of PFOS (RPA and BRE, 2004). Environment Canada’s ecological risk assessment defines PFOS precursors as substances containing the perfluorooctylsulfonyl (C8F17SO2, C8F17SO3, or C8F17SO2N) moiety that have the potential to transform or degrade to PFOS (Environment Canada, 2006). The term “precursor” applies to, but is not limited to, some 51 substances identified in the ecological assessment. However, this list is not considered exhaustive, as there may be other perfluorinated alkyl compounds that are also PFOS precursors. This information was

70 Perfluorinated substances with different carbon chain lengths are included in the list.
compiled based on a survey to industry, expert judgement and CATABOL modelling, in which 256 perfluorinated alkyl compounds were examined to determine whether non-fluorinated components of each substance were expected to degrade chemically and/or biochemically and whether the final perfluorinated degradation product was predicted to be PFOS. While the assessment did not consider the additive effects of PFOS and its precursors, it is recognized that the precursors to PFOS contribute to the ultimate environmental loading of PFOS. Precursors may also play a key role in the long-range transport and subsequent degradation to PFOS in remote areas, such as the Canadian Arctic.

A preliminary substance flow analysis for Switzerland in 2005 based on the international literature estimated remaining PFOS-related substances in products after the retreat of 3M products to be approx. 230 kilogram /a. (Switzerland, 2007)

1.2 Conclusions of the Review Committee Annex E information
The POPs Review Committee has conducted and evaluated the risk profile for perfluorooctane sulfonate contained in document UNEP/POPS/POPRC/17/Add.5:in accordance with Annex E of the Convention and adopted the risk profile for perfluorooctane sulfonate. The Committee decided, in accordance with paragraph 7 (a) of Article 8 of the Convention, that perfluorooctane sulfonate is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects such that global action is warranted (Decision POPRC-2/5). The Committee also decided (item 3 of the Decision) that issues related to the inclusion of potential perfluorooctane sulfonate precursors should be dealt with in developing the draft risk management evaluation for perfluorooctane sulfonate.

1.3 Data sources
Data according to Annex F were submitted by the following Parties:
- Algeria
- Brazil
- Canada
- Czech Republic
- European Commission
- Germany
- Japan
- Mauritius
- Monaco
- Switzerland

and the following observers:
- European Photo And Imaging Association
- European Electronic Component Manufacturers Association
- International Imaging Industry Association
- European Semiconductor Industry Association (EECA-ESIA)
- International POPs Elimination Network (IPEN)
- Japan Electronics and Information Technology Industries Association –Japan Semiconductor Industry Association (JEITA-JSIA)
- Photo Sensitized Materials Manufacturers’ Association
- Semiconductor Industry Association (SIA)
- Semiconductor Equipment and Materials International (SEMI)
- United States of America

Canada, the EU and Germany have provided national and international management reports (see References)

Status of the chemical under international conventions
PFOS is subject to a risk management evaluation under the UNECE Convention on Long-Range Trans-Boundary Air Pollution (LRTAP) POPs Protocol. The POPs Task Force will meet in Vienna in June to discuss and agree, as appropriate, on a proposal for measures on
PFOS to be submitted to the Executive Body through the Working Group on Strategies and Review for consideration and possible adoption.

1.5 Any national or regional control actions taken
Australia has produced two Alerts concerning PFOS through its National Industrial Chemicals Notification and Assessment Scheme (NICNAS). The first Alert indicated the phasing-out of water, oil, soil and grease repellent products containing PFOS by September 2002. As well, the use of PFOS for leather products was to be phased out by March 2003. All other products containing PFOS, including fire fighting foams and industrial additives, were to be phased out in Australia by December 2003.

The second Alert makes recommendations regarding PFOS, perfluorosulfonates (PFAS) and perfluorooctanoic acid (PFOA). These recommendations include:
- that PFOS (and PFAS-based chemicals) be used only for essential uses for which there is no suitable alternative, such as certain class B fire fighting foams, but not for use in fire training exercises; and
- that caution be used in selecting PFOA as an alternative for PFOS since PFOA may show the same environmental and health concerns as PFOS.

Canada has proposed regulations to prohibit the production and use of PFOS and its salts and substances that contain on of the following groups: C8F17SO2, C8F17SO3 or C8F17SO2N (Canada Gazette, vol. 140, No 50, December 16, 2006).

The proposed regulations for PFOS would:
- prohibit the manufacture, use, sale, offer for sale and import of PFOS or products containing these substances;
- exempt the use of PFOS-based aqueous film-forming foam, sometimes also referred to as aqueous fire fighting foam (AFFF), manufactured or imported before the coming into force of the proposed Regulations for a period of five years after the coming into force of the proposed Regulations (but this AFFF may not be used for training or testing purposes);
- exempt the use of PFOS-based fume suppressants, and sale, offer for sale and import for that use, for a period of five years after the coming into force of the Regulations for chromium electroplating, chromium anodizing, reverse etching, electroless nickel-polytetrafluoroethylene plating and etching of plastic substrates prior to their metallization;
- exempt the use, sale, offer for sale and import of the following manufactured items: semiconductor or similar components of electronic or other miniaturized devices and photographic films, papers and printing plates;
- exempt the use, sale and offer for sale of manufactured items, that were manufactured or imported before the coming into force of the proposed Regulations; and
- provide standard exemptions for laboratories, scientific research and laboratory analytical standards.

Importers of PFOS-based fume suppressants will be required to submit annual reports detailing types, quantities, sales and end uses for the substances that are imported.

- PFOS and related substances will be banned as substances or constituents of preparations in concentrations equal to or higher than 0.005%, in semi-finished products and articles at a level of 0.1% except for textiles or coated materials in which the restricted amount of PFOS will be 1 µg/m2. Exemptions were provided for the following PFOS uses, as well as for the substances and preparations needed to produce them: photoresists or anti-reflective coatings for photolithography processes, industrial photographic coating, mist suppressants for chromium plating and other electroplating applications, as well as aviation hydraulic fluids;
- stocks of PFOS-based AFFF supplied on or before the date 12 months before the legislation comes into force may be used for a period of 54 months.
The United States Environmental Protection Agency (US EPA) has adopted federal Significant New Use Rules (SNURs) for 88 PFOS substances which apply to new manufacture and new uses of these substances. A proposed SNUR for 183 additional perfluoroalkyl sulfonate substances was posted in April 2006 for public consultations, and the publication of the final SNUR is expected in 2007. The SNURs require manufacturers and importers to notify the US EPA at least 90 days before manufacture or import of these substances for any use other than certain narrow, ongoing uses. This provides the US EPA with the necessary time to evaluate the intended new use and prohibit or limit the new activity, if necessary. While the SNURs did not require current manufacturers to stop manufacturing or selling the substances, the primary manufacturer in the United States voluntarily discontinued production between 2000 and 2002. Therefore, once the SNURs became effective, they essentially restricted all manufacture and importation of PFOS other than manufacture and importation other than for certain specific uses, excluded from the SNURs including

- use in aviation hydraulic fluids;
- as a component of a photoresist substance, including a photo acid generator (PAG) or surfactant, or as a component of an anti-reflective coating used in a photomicroelectronic fabrication process to produce semiconductors or similar components of electronic or other miniaturized devices;
- in coatings for surface tension, static discharge, and adhesion control for analogue or digital imaging films, papers and printing plates; and
- as an intermediate only to produce other chemical substances to be used solely for the uses listed above.

The US EPA also negotiated a phase-out of PFOS-related pesticide products containing sulfuramid, a substance that is manufactured using a PFOS derivative and will degrade to PFOS, or the lithium salt of PFOS (LPOS), concurrently with the 2002 publication of the final SNUR on 88 PFOS substances. Sulfuramid and LPOS were formulated in bait stations for the control of ants, roaches, termites, wasps, and hornets, and in one granular broadcast bait for control of leaf cutter ants in pine reforestation areas. The registrants associated with those products agreed to voluntarily cancel some of their products and to phase out the remaining ones under an agreed-upon timeline. The continuing products being phased out were produced using stocks of sulfuramid produced before the completion of the PFOS production phase-out in the US in 2002.

2. Summary information relevant to the risk management evaluation

2.1 Identification of possible control measures

The objective of the Stockholm Convention (Article 1) is to protect human health and the environment from persistent organic pollutants. When assessing control measures under the Convention, consideration should be given to the potential for all PFOS-related substances to degrade to PFOS and thus contribute to the total environmental load. When assessing whether specific exemptions would be needed, factors such as low exposure; small amounts; and high societal costs and the ubiquitous contamination of humans, the environment and future generations as well as the principles of Polluter Pays and Intergenerational Equity should be considered.

Under the convention this may be achieved in different ways.

- PFOS and/or PFOS-related substances may be listed in Annex A, with or without specific exemptions, or accompanied with a new Part III that details actions for each or groups of PFOS-related substances or uses of such substances; or
- PFOS and/or PFOS-related substances may be listed in Annex B, with acceptable purposes/species exemptions or a Part III of Annex B that details actions for each or groups of PFOS-related substances or uses of such substances; or
- PFOS may be listed in Annex C as an unintentional POP to capture all future uses of presently unknown PFOS-related substances that may give rise to PFOS when released into the environment; or
- PFOS may be listed in Annex A or B, as described above, and at the same time also be listed in Annex C.
In the Annex F process, some critical uses have been identified by Parties and observers. They may be grouped into two subgroups.

A. Uses for which at present, according to responses received, it is claimed that no technically feasible alternatives are available. These uses are:
- Photo imaging
- Photo mask
- Semi-conductor
- Aviation hydraulic fluids
- Manufacture of ant baits for leaf-cutting ants

B. Uses for which alternative substances or technologies are or may be available but would need to be phased in. These uses are:
- Metal plating
- Fire fighting foam

These uses and the potential substitutes will be further described in section 2.3 below.

2.2 Efficacy and efficiency of possible control measures in meeting risk reduction goals

The phase-out and regulation in the US successfully reduced the volume of these chemicals produced and/or used in the U.S. from approximately 2,900 tonnes in 2000 to less than 8 tonnes in 2006.

Canada has provided a cost-benefit analysis for the proposed Canadian regulation of PFOS and PFOS-related substances. The key assumptions used in the cost-benefit analysis included:
- Time frame: The proposed control measures could come into force in 2009, with the exemption for AFFF and the metal plating sector expiring 5 years later in 2012;
- Time span for analysis: A time frame of 25 years is selected to account for the life span of PFOS containing AFFF as well as the service life of metal plating equipment. Thus, the analysis time frame is 2008 to 2032;
- Cost and benefit perspective: costs and benefits which directly or indirectly affect human health and the environment are included in the analysis to the extent possible;
- Discount rate: A discount rate of 5.5%, and all monetized costs and benefits are expressed in 2006 € or US$.

For Canada the net benefits of the proposed regulations were estimated at US$337,000. It should be noted that this does not include benefits to the ecosystem as these could not be quantified due to data limitations and uncertainties (Canada, 2007).

There are also some cost calculations in the RPA report on risk reduction strategy (2002) and estimates of development costs provided by SIA that are used in the following.

2.3 Information on alternatives (products and processes), where relevant

The POPRC has agreed that PFOS is a POP. The target or aim of any risk reduction strategy for PFOS should be to reduce or eliminate emissions and releases taking into consideration technical feasibility and risk and benefits of the substances and their continued production and use. In considering any strategy for a reduction in such risks, it is important to consider the availability of substitutes in the sectors of concern. In this regard, the replacement of a PFOS-related substance by another chemical or an alternative system needs to take account of:
- the existence and cost of the substitute or alternative system;
- the technical suitability of the substitute or alternative system;
- the environmental and human health effects of the substitute or alternative system; and
- the capability of the substitute or alternative system to meet the required safety standards.

71 Not relevant for unintentionally produced POPs
A discussion of the availability and suitability of substitutes for the ‘continuing uses’ of PFOS-related substances is provided below against the factors noted above. The discussion focuses on continuing uses (rather than historical uses) as substitution is considered to already have taken place in the other sectors.

A significant proportion of previous users of PFOS-related substances have moved to other fluorochemical products (telomers and related products). Telomers cannot degrade to PFOS but under certain circumstances may degrade to perfluoroctanoic acid (PFOA) or related perfluorinated carboxylic acids. It is important to note that, while there is little information currently available to assess the environmental and health impacts of telomers, extensive work is currently on-going in the US and other countries where there is some concern over the fate and behaviour of these substances. Until these and other studies are concluded, it will not be possible to draw any firm conclusions concerning the environmental/human health advantages of telomers and related products over the PFOS-related substances that they have substituted.

A. Uses for which at present, according to responses received, no technically feasible alternatives are available

2.3.1 Photo imaging
Chemicals or classes of chemicals that may be considered alternatives to PFOS-related substances on an industry-wide basis (or even a company-wide basis) are reported as not currently being available for the photographic industry.

Successful alternatives to PFOS materials have included non-perfluorinated chemicals such as hydrocarbon surfactants, chemicals with short perfluorinated chains (C3 - C4), silicones, telomers, and in very few cases it has been possible to reformulate coatings so that they are inherently less sensitive to static build-up.

The imaging products/applications where there are currently no identified alternatives to PFOS-related substances and which represent critical uses are as follows:

- surfactants for mixtures used in coatings applied to films, papers, and printing plates; The ability to control surface tension in imaging materials is a critical aspect of the use of PFOS substances as coating aids. Imaging materials must be coated with multiple (up to 18) layers of light sensitive materials at high speed to prevent the drying of materials as they are laid down. PFOS chemicals are critical for creating coatings of high complexity in a highly consistent manner, thus avoiding the creation of large amounts of waste due to irregularities in coating thickness;

- electrostatic charge control agents for mixtures used in coatings applied to films, papers, and printing plates. PFOS coating aids also have unique properties at low concentrations for controlling static charge during the manufacture and use of imaging materials. This is particularly important for imaging materials that have a high sensitivity to light (i.e., high speed), as these products are unusually sensitive to light produced by static discharge during transport of imaging materials. Coating aids must not be photoactive; otherwise, unacceptable fogging or speed effects may occur in the coatings;

- friction control and dirt repellent agents for mixtures used in coatings applied to films, papers, and printing plates. Excessive friction during the transport of imaging materials and contamination of imaging materials by dirt or clogging of magnetic strip readers with debris can lead to significant waste of imaging materials during manufacturing and use; and

- adhesion control agents for mixtures used in coatings. Adhesion control is a property imparted to film coatings as a result of the use of PFOS materials as coating aids. Control of adhesion of various tapes to imaging materials is important because tape is the primary way in which imaging materials are attached to spools and to each other during processing. The strength of the bond between the tape and the imaging materials must be controlled so that imaging devices (e.g., cameras, photo processors) and imaging materials are not damaged during transport (i.e., the adhesive bond between the tape and the imaging material must be broken by a force that will not damage devices or materials being transported).
Industry estimates that the releases from the photo imaging industry are 1.02 kilogram into waste water and 0.051 kilogram into air from the manufacturing uses by the photo imaging industry in the EU and by extrapolation, less than 2 kilogram worldwide. Occupational exposure to PFOS-related substances from photo imaging products is minimal. Personal and area monitoring in the workplace show that airborne concentrations are below the level of detection (< 0.013 mg/m3). Most consumer and professional imaging papers do not contain PFOS-related substances. For papers that do contain the substances, the coatings contain concentrations in the range of 0.1-0.8 µg/cm2. Most of this material will not be on the surface of the coating as the PFOS-related substance is contained within a matrix and is bound to coating matrices.

The cost, so far, for replacement of PFOS materials is estimated to be in the range of € 20-40 M for the full range of imaging products. These costs are based on the estimated cost of achieving the current reduction of 83% in the use of PFOS-related substances. The cost to be incurred from further work on replacements (for the remaining 17%) is expected to be significantly higher than the above figure as the replacement work is increasingly more difficult.

Based on previous cost estimates of US$20-40 M for reduction that took place between 2000 and 2004, i.e. a reduction of roughly 15 tonnes, the average cost is US$2 M per tonne. Further reductions are estimated to cost more than twice as much, up to US$5 M per tonne. The cost of substituting the remaining 10 tonnes would be US$50 M. Since only 2 kilogram is estimated to be released into the environment the cost of reducing the release to zero, using these estimates would be US$25 M per kilogram. This calculation indicates the level of magnitude of the costs of reducing the release. The estimate is more likely to be an overestimation than the opposite but would in any case be very high.

2.3.2. Photoresist and Semi-conductor

The operation of PFOS based PAGs is critical to the semiconductor industry in the photolithography process. PAGs contain PFOS-related substances due to the resultant optical characteristics (uniform exposure), sensitivity, speed, low acid volatility, resolution and depth of focus and high yield (low incidence of contamination or defects), all of which allow semiconductors to be manufactured with more accurately defined features, which reduces the risk of semiconductor failure. ESIA, JSIA, SIA and SEMI indicate that there are currently no substitutes known that give the same level of critical functionality to cause effective, efficient transformation in leading edge photoresists and which can be used in volume manufacturing.

For anti-reflective coatings used in combination with photoresists, ESIA indicates that there is also no alternative available which fulfils the critical technical requirements necessary (ESIA, 2003). The industry is also evaluating one additional specialized application for which PFOS use may have no current substitute -- use in liquid etchant in the photo mask rendering process.

The semiconductor industry indicates that the industry and its suppliers continue to search for alternatives for these critical uses. If alternatives to PFOS are eventually identified at the fundamental research stage, the nature of semiconductor production is such that critical adjustment to the chemistry of inputs such as PFOS use in the photolithography process will trigger far-reaching adjustments throughout the manufacturing process and supply chain to ensure that the chemical processes throughout the production process remain aligned. Thus, it could take an additional ten years to design, operationalize and integrate the new technology, once it has been identified, into the semiconductor manufacturing process. The delay is a necessary function of the semiconductor technology development cycle: technological innovations generally require 10 years of further development before they can be reflected in high volume manufacturing. (ESIA, JSIA, SIA, SEMI 2007).

It should also be noted that during the chemical formulation of photolithography products, worker exposure potential is very low. Chemical formulation of photolithography products occurs under highly automated, largely closed system conditions. The same process for
electronics fabrication is similarly automated, with a low volume of PFOS used, and use of protective equipment. Chemical isolation is also an intrinsic part of quality control procedures.

Environmental release potentials are deemed to be low. Due to the low vapour pressure of PFOS, and the nature of the process, no emissions to the air are expected. Waste products, including 93% of the resist formulation (PAGs and surfactants) are incinerated. Releases to water are also considered to be negligible. Furthermore, there is no residual PFOS compound present in manufactured microprocessors and therefore no consumer exposure or concern about releases from electronic waste disposal or recycling.

PFOS releases from photolithography uses are small compared with PFOS use in other industry sectors. In 2002 for the whole of Europe, an estimated 43 kilogram of PFOS were released in the effluent from photolithography uses, in the order of only 0.45 percent of all PFOS releases at that time in Europe. Mass balance data for Europe in 2004 indicates an estimated 54 kilogram of these releases. It has been estimated that a similarly small proportion of releases in the United States and Japan is attributable to the photolithography uses, based on recent past use patterns.

It is difficult to quantify the costs that will ultimately be involved in replacing PFOS with alternative substances, given that such alternatives are not currently available. The requirements for innovation and the limits of technical feasibility are the main factors that currently limit access to alternatives. If those hurdles can be overcome there will be substantial costs associated with the transition to the use of alternative substances in the photolithography process. In addition, there could be extensive introduction costs associated with bringing a new system into high volume production, including requalification costs and possible loss of revenues associated with much lower yield as new systems are brought on line. Many resists are specifically tailored to one individual company’s process, which means that a valid replacement for one cannot necessarily be applied industry-wide.

Given those uncertainties the estimate below is only an indication of the order of magnitude of the costs involved.

Replacing existing resists systems would require extensive R&D followed by a time-consuming manufacturing process requalification. The development cost of a completely new photoresists system - one resist system - for the industry has been estimated at US$192M for 193nm resist, US$287M for 157nm, and US$218M for EUV resist. The cost for 157nm resist development is the highest, because it has more novel requirements than either 193nm or EUV resists.

Development costs of a new photoresist system thus add up to US$700M. Assuming that variable costs are the same as in the present system, it takes 5 years to develop the new system and the time span for the analysis is 25 years. This would imply that the reduction in release of PFOS related substances is equal to 20 years of releases (50 kilogram per year), i.e. a total of 1000 kilogram. Costs would be US$0.7M per kilogram PFOS. This calculation indicates the level of magnitude of the costs of reducing the release.

The semiconductor industry recently signed an agreement to curtail the use of PFOS-based chemicals at the global level. Under the agreement, members of the World Semiconductor Council, which comprises the trade associations representing the microchip industries of most of the world’s leading semiconductor-producing countries (including SIA, ESIA and trade associations in Asia), and SEMI have committed to the following actions: (i) ending non-critical uses for PFOS by specific dates; (ii) working to identify substitutes for PFOS in critical uses for which no other materials are presently available; (iii) destroying solvent wastes from critical uses; and (iv) taking other steps to mitigate the potential environmental impacts of PFOS use in these critical applications.

2.3.3 Photo masks in the Semiconductor and Liquid Crystal Display (LCD) Industries

Photo masks are an essential part of the photolithography process of semiconductor and LCD production. Photo mask production is mainly outsourced from semiconductor or LCD producers to other companies. The production process of photo masks is as follows:

1. Deposit a metal chromium layer on glass a substrate by sputtering
2. Coat photoresist on the Chromium layer.
(3) Draw wiring pattern on the photoresist.
(4) Develop the photoresist.
(5) Remove un-covered Chromium layer by etchant containing PFOS.
(6) Remove hardened photoresist by rinsing fluid.
(8) Dry the photo mask.

Three major photo mask producers in Japan report that this wet process is used in the production of most photo masks. PFOS and PFOS-related substances are contained in etchants for semiconductor and TFT panels, because these products require very fine patterning. In the case of photo masks for semiconductors, a dry process is also used for some specific cases but the major process is a wet process using etchants containing PFOS and PFOS-related substances. All TFT photo masks are produced using a wet process because of their large size.

PFOS is used to enhance surface wettability of etchants by its low surface tension. Sufficient capability to lower surface tension is essential in order to apply a very fine pattern on photo mask blanks without any defects.

The total amount of PFOS (including PFOS moiety in PFOS-related substance) use for this purpose in Japan is estimated at approximately 70 kilogram per year. It is estimated that Japanese companies play a major role in photo mask production, and have more than a 70% share of the worldwide market. Thus it is estimated that total use of PFOS and PFOS-related substances for this use in the world is approximately 100 kilogram.

The use of PFOS and PFOS-related substances in etchants was not recognized by the photo mask industry until recently owing to this small amount of use and low concentration. Because of strong acid of etchants, non-fluoro surfactant is not stable in etchants, thus it is not applicable for this process. Furthermore, other fluoro surfactants such as shorter chain PFAS are not suitable because their ability to lower surface tension is not sufficient.

A dry etching process is applied to high-end ultra-fine patterns of semiconductor photo masks. However, the yield and productivity of the dry etching process is much (15 to 20 times) lower than the wet process. Furthermore, the dry process is not useable for LCD panels because of their large size (more than 1m by 1m).

2.3.4. Aviation hydraulic fluids
According to information received from one of the major producers of hydraulic fluids, there are no alternatives to the PFOS substances currently being used in aircraft systems. There is also no known alternative chemistry which will provide adequate protection to aircraft.

It has been suggested that the process of qualifying a new fluid for use in commercial aircraft has historically taken about 10 years from concept to actual commercial manufacture. As there are no current alternatives to PFOS substances currently being used in aircraft systems, there is no information on costs or environmental/human health attributes of alternatives.

2.3.5. Use of PFOS derivative in production of ant baits for control of leaf-cutting ants
Sulfuramid (1-octanesulphonamide-N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro; CAS: 4151-50-2), is manufactured using a PFOS derivative (perfluorooctyl sulfonyl fluoride, CAS. No. 307-35-7). Sulfuramid is the active ingredient in the manufacture of ant baits in ready-to-use formulations. and is known to degrade to PFOS. It is estimated that the production of sulfuramid in Brazil is about 30 metric tonnes per annum. Sulfuramid is used at a 0.3% concentration, resulting in a production of around 10,000 metric tonnes of ant baits/year. In 2006, around 400 metric tonnes of ant baits (sulfuramid 0.3%) were exported to 13 countries in South and Central America. Sulfuramid cannot be manufactured without the use of PFOS derivatives.

Several mechanical, cultural, biological and chemical methods, including different formulations, have been studied for controlling leaf-cutting ants. Granulated baits represent the most widely used method for leaf-cutting ant control, consisting of a mixture of an attractant (usually orange pulp and vegetable oil) and an active ingredient (insecticide),
presented in the form of pellets. This method features some significant advantages over other methods. It is a low-cost method, delivering high efficiency with reduced health hazards to humans and the environment during application and being specific to the pest target. Its formulation is developed with low concentrations of active ingredients, and its localized application does not require application equipment. The utilization of ready-to-use formulations should reduce or impede releases to humans and to the environment when using the formulated product.

Currently, the active ingredients used in ant baits are: sulfluramid, fipronil and chlorpyrifos. Fipronil and chlorpyrifos are more acutely toxic to mammals, water organisms, fish and bees than sulfluramid. Comparative studies demonstrate low efficiency of ant baits with chlorpyrifos and fipronil. It has been claimed that sulfluramid cannot be efficiently replaced in Brazil by other registered products commercialized for the same purpose.

B. Uses for which alternative substances or technologies may be available but would need to be phased in.

2.3.6. Metal plating

PFOS-related substances are used in the following main applications:

• decorative chromium plating; and

• hard chromium plating.

Information received indicates that there are currently no known effective alternative chemical mist suppressants to PFOS-related substance for these applications.

However, information received from a number of industry and regulatory authorities indicates that the substitution of Cr (VI) with the less hazardous Cr (III) in decorative plating applications would eliminate the need for the use of PFOS-related substances in this application. Such substitution has potentially significant cost savings and health and safety and environmental benefits for the metal plating sector. The higher costs of using Cr (III) are more than offset by the savings from reduced waste treatment costs, reduced air monitoring costs, record keeping, and the reduced reject rate. The major benefit, however, relates to the significantly reduced risk of employee ill health induced by working with hexavalent chromium. However, the progress of substitution is different due to the quality requirements of the different markets e.g. in Japan only 40-50 of about 1000 companies have changed their process.

For hard chromium plating, information received indicates that the direct substitution of Cr (VI) is not currently a viable option and there are currently no known effective alternative chemical mist suppressants to PFOS-related substance for this use.

In Japan alternatives for other uses than hard chromium plating are not yet identified partially because of the requirements for high reliability e.g. for automobile pumping parts.

The cost of improved ventilation with extraction, which is the recommended substitute for PFOS-based mist suppressants, has been calculated to be €3400 per year in each production unit where the investment period is 15 years (RPA 2004). Assuming a few hundred units in the EU the total cost would be one or two million euros. In Japan it has been estimated that the cost would be US$40 000 per each 1000 litre bath (Japan, 2007).

The anticipated costs of the proposed Canadian regulations by firm size are US$0.65 M for 34 small firms, US$2.6 M for 52 medium firms and US$0.68 M for 14 large firms. The total estimated compliance costs for facilities using PFOS fume suppressants to comply with the proposed regulations is approximately US$3.9 M (discounted at 5.5% over 25 years). This would result in a reduction in PFOS releases of approximately 86 tonnes over the 2013 to 2032 period (Canada, 2007)). Based on these Canadian calculations the cost of reduction is US$46 per kilogram of PFOS reduced.

2.3. 7. Fire fighting foam

A number of alternatives to the use of PFOS-based fluorosurfactants in fire fighting foams are now available/under development. These alternatives include:
• non-PFOS-based fluorosurfactants;  
• silicone based surfactants;  
• hydrocarbon based surfactants;  
• fluorine-free fire fighting foams; and  
• other developing fire fighting foam technologies that avoid the use of fluorine.

Fluorine-free foams are approximately 5-10% more expensive than the fluorosurfactant-based foams (including those PFOS-based foams marketed previously). The manufacturers, however, indicate that prices for fluorine-free foams would reduce if the market size increased. It is, therefore, assumed that prices are broadly comparable.

As the transition from PFOS-based products has already taken place for most uses in many countries, there are only limited developmental or operational costs associated with the substitution of PFOS based foams by foam manufacturers or users. The main costs for phasing out PFOS-based foams are related to managing stockpiles and waste containing such foams.

With regard to the toxicological and ecotoxicological suitability of non-PFOS based fluorosurfactants, data are limited. Whether telomers represent a significant concern for human health and the environment is under review elsewhere and conclusions are awaited.

With regard to fluorine-free foams, current information indicates that compared to PFOS based foams, they do not persist or bio-accumulate in the environment (due to the absence of fluorine). With regard to acute toxicity, fluorine-free foams appear to have a slightly lower acute toxicity, although the information provided to date is not conclusive.

For Canada, it is estimated that the proposed regulations would reduce the release of PFOS based AFFF into the environment in the order of 2.83 tonnes over the 2008 to 2032 period. The present value of the disposal and replacement costs experienced by airports, military facilities and refineries would be in the order of approximately US$0.64 M (in 2006 $) discounted at 5.5% over the 25-year time period. (Canada, 2007) Based on these Canadian calculations the cost of reduction is US$226 per kilogram of PFOS reduced.

For the EU, costs of replacement and destruction of foam have been estimated at €6000 per tonne. The stocks in the EU are 122 tonnes (RPA 2004). Based on the RPA:s calculations, the cost of reduction is €6 per kilogram of PFOS reduced. Once the foam has been renewed, the cost of destruction may be as low as €1 per kilogram.

In Japan, it has been estimated that 86 tonnes of PFOS equivalent exist in AFFF products on the market. Based on this information, the estimated total amount of PFOS in the market is less than 200 tonnes in fire fighting foam concentrate. PFOS fire fighting foam concentrate is very stable and stored for emergency cases. Therefore manufactured PFOS fire fighting foam concentrate is basically stored on a long-term basis. It means some 20,000 tonnes of PFOS fire fighting foam concentrate is stored in the Japanese market. The majority of the market stock is fire fighting foam for water immiscible liquid and non-PFOS alternatives are marketed for this use. Fire fighting foam for water miscible liquid which is required to fulfil government standards is not yet developed due to technical difficulties and the technical feasibility is still uncertain. Furthermore, fire fighting foam containing PFOS is also stored at airports. (Japan, 2007)

2.4 Summary of information on impacts on society of implementing possible control measures

2.4.1 Health, including public, environmental and occupational health
A positive impact on human health and on the environment can be expected from reduction or elimination control measures on PFOS on a global scale. The establishment of further control measures for those uses of PFOS for which no substitution is yet possible, will presumably contribute positively to human health and the environment, especially concerning reprotoxicity and blood values.
The positive impact may be greatest for vulnerable groups such as pregnant women, embryos and infants due to the reproductive toxicity of PFOS. If PFOS production and use is not eliminated, then levels in the environment including humans and animals will continue to rise, even in locations distant from production and use.

It is claimed that no negative impact is anticipated to result from the ongoing small number of critical uses e.g. the imaging industry and the semi-conductor industry.

2.4.2 Agriculture, including aquaculture and forestry

The elimination of PFOS could adversely affect Brazilian agriculture through its impact on the production of sulfluramid ant baits claimed to be indispensable for the control of leaf-cutting ants in agricultural or forest undertakings. Specific exemptions or listing of acceptable purposes could permit the continued use of PFOS in the production of sulfluramid ant baits, which would protect agricultural interests.

2.4.3 Biota (biodiversity)

As the persistent, bioaccumulative and toxic properties of PFOS were shown under the POPs-Protocol and under the Stockholm Convention, a positive impact on biota from a ban/restriction of the substance can be expected.

The scientific literature has identified that at current exposure levels, PFOS could harm certain wildlife organisms (e.g. polar bear, fish-eating birds), including those found in remote locations such as in the Canadian Arctic. The effects include growth inhibition of birds and aquatic invertebrates; liver and thyroid effects in mammals; lethality to fish and saltwater invertebrates; and changes in biodiversity. While PFOS is generally acknowledged to have the potential to cause serious, irreversible impacts (related to bioaccumulation and persistence), the current science is unable to accurately predict the ecological effects of these substances. The absence of specific impacts on the environment on which to model the economic value of reductions in current releases makes it difficult to quantify and monetize the benefits from the proposed Regulations. Although difficult to quantify, these benefits should be considered qualitatively in the assessment.

Environmental sampling studies conducted since the 2002 PFOS phase-out and already submitted to the committee by Canada suggest that environmental exposures to PFOS have decreased significantly as a result of the phase-out. (USA, 2007)

2.4.4 Economic aspects, including costs and benefits for producers and consumers and the distribution of costs and benefits

The potential benefit from avoided alternate water supply expenditure attributable to the proposed Canadian regulations (prohibiting production, marketing and use of PFOS and PFOS-related substances) is estimated to result in an average annual net benefit of US$0.49 M per year. It is recognized that this benefit is uncertain; however, the value can be used to approximate the benefits to be derived as a result of the proposed regulations. Total benefits to Canadians are estimated to be approximately $557 M million. (Canada, 2007) It is recognized that this benefit is uncertain and that only a fraction of the benefits have been monetarized.

Photo-imaging

According to industry, restrictions on the remaining uses of PFOS-related substances would have a severe impact on the photo imaging industry’s ability to manufacture a number of imaging products, including diagnostic medical products, industrial X-ray (non destructive testing), graphic printing (printing mask) and would impose a significant cost by requiring substantial investment in research and development.

Photoresists and semi-conductors

2005 global sales were US$228 billion, with initial estimates for 2006 well above US$260 billion. The semiconductor industry employed 226,000 people in the United States and 87,000 in Europe. Global employment in the industry was approximately 500,000 in 2003, but this figure has certainly increased.
The semi-conductor industry considers that implementation of control measures that effectively precluded the use of PFOS in critical applications for semiconductor manufacturing would likely shut down high volume production semiconductor manufacturing for a considerable time. This action could have a drastic effect on the global economy.

**Metal plating**
The cost of improved ventilation with extraction, which is the recommended substitute for PFOS-based mist suppressants, has been calculated to be €3400 per year in each production unit where the investment period is 15 years (RPA 2004). Assuming a few hundred units in the EU the total cost would be one or two million euros. In Japan it has been estimated that the cost would be US$40 000 per each 1000 litre bath (Japan, 2007). Based on the Canadian calculations the cost of reduction is US$46 per kilogram of PFOS reduced.

**Fire fighting foam**
For the EU costs of replacement and destruction of foam have been estimated at €6000 per tonne. The stocks in the EU are 122 tonnes (RPA 2004). Based on the RPA:s calculations the cost of reduction is €6 per kilogram of PFOS reduced. Once the foam has been renewed the cost of destruction may be as low as €1 per kilogram. Based on the Canadian calculations the cost of reduction is US$226 per kilogram of PFOS reduced.

**Cost comparisons**
Rough calculations based on limited existing data and estimations indicate that the differences in costs for reduction of one unit of release of PFOS-related substances are very large. The cost per kilogram has been estimated at US$25 M (€ 18.6 M) for photo imaging, US$0.7 M (€ 0.52 M) for semi-conductors, US$184 (€137) for destruction of fire foam and US$46 (€ 40) for metal plating. Lack of data has made it impossible to do similar estimates for photo masks, aviation hydraulic fluids and ant baits. These uses can be judged to have costs between the two uses that are expensive to reduce and the two that are relatively cheap to reduce.

In conclusion, no substitutes seem to exist today for the following uses/sectors: photo imaging, photo masks, semi-conductor, aviation hydraulic fluids and baits for leaf-cutting ants. For photo imaging and semi-conductors the costs for alternatives have been found to be very high and for aviation hydraulic fluids, photo masks and baits for leaf-cutting ants there is no data for analyses of economic costs of alternatives.

2.4.5 Movement towards sustainable development

As the persistent, bioaccumulative and toxic properties of PFOS as well as its potential for a long-range transboundary transport were shown under the LRTAP Convention POPs-Protocol and in the risk profile agreed by the POPRC of the Stockholm Convention, a positive impact on a globally sustainable development from a ban/restriction of the substance is be expected.

**2.4.6 Other impacts**
Although PFOS is not used for the manufacturing of fire fighting foams anymore, there are still stocks of ca. 122 tonnes in the EU (Germany, 2007).

The large use of PFOS in consumer products has implications for municipal waste and disposal along with attention to production stockpiles. A listing of PFOS in Annex A or B would subject wastes, products or articles containing the substance to Article 6 of the Stockholm Convention and require that they be disposed, “…in a safe, efficient and environmentally sound manner.

2.5 Other considerations

2.5.1 Access to information and public education
For more information on industry innovations regarding PFOS alternatives, please see the following websites:
SIA: [http://www.sia-online.org/home.cfm](http://www.sia-online.org/home.cfm)
EECA-ESIA: [http://www.eeca.org/esia.htm](http://www.eeca.org/esia.htm)


(Semi-conductor)

Access to information on the U.S. regulation of these chemicals and the industry response is available through the online dockets for the U.S. EPA rulemaking proceedings. Information on those proceedings and dockets is available on the EPA website at [http://www.epa.gov/opptintr/pfoa/pubs/related.htm](http://www.epa.gov/opptintr/pfoa/pubs/related.htm).

Additional material on PFOS and related perfluorinated compounds is publicly available in a non-regulatory data repository maintained by the EPA Docket Office as Administrative Record AR-226. AR-226 is not available online, but an index to the documents can be requested by email from oppt.ncic@epa.gov, and documents in AR-226 are available on CD-ROM. (USA, 2007)

### 2.5.2 Status of control and monitoring capacity

The semiconductor industry will collect and make available aggregated industry information every 2 years to provide a transparent communication of industry progress, including:

a) the results of PFOS wastewater treatment evaluations including any wastewater measurement data;

b) a description of the current relevant research and development activities and any conclusions including the results of collaboration with equipment and chemical suppliers;

c) industry phase-out schedules that are known for critical uses in semiconductor manufacturing and processing; and

d) the results of the PFOS mass balance model. (SIA, 2007)

### 3. Synthesis of information

#### 3.1 Summary of risk profile information

Perfluorooctane sulfonate (PFOS) is a fully fluorinated anion, which is commonly used as a salt in some applications or incorporated into larger polymers. Due to its surface-active properties, it has historically been used in a wide variety of applications, typically including fire fighting foams and surface resistance/repellency to oil, water, grease or soil. PFOS can be formed by degradation from a large group of related substances, referred to as PFOS-related substances (see definition on page 4).

Due to their intrinsic properties, PFOS and its related substances have been used in a wide variety of applications. The present uses seem to be limited to the sectors described above. It is not known whether this reflects the total global use.

PFOS and PFOS-related substances can be released to the environment at their manufacture, during their use in industrial and consumer applications, and from disposal of the chemicals or of products or articles containing them after their use.

The rate and the extent of the formation of PFOS from its related chemicals are largely unknown and may differ between individual substances. Lack of data makes it very difficult to estimate the net contribution of the transformation of each of the PFOS-related substances to the environmental loadings of PFOS. However, based on its extreme stability, it is expected that PFOS is likely to be the final degradation product of all PFOS-related substances.

PFOS is extremely persistent. It has not shown any degradation in tests of hydrolysis, photolysis or biodegradation in any environmental condition tested. The only known condition whereby PFOS is degraded is through high temperature incineration.

With regard to bioaccumulation potential, PFOS meets the Annex D criteria given the highly elevated concentrations that have been found in top predators such as the polar bear, seal, bald eagle and mink. Based on the concentrations found in their prey, high BMFs have been estimated for these predators. BCF values in fish, although (rather) high do not in themselves meet the specific numeric criteria. However, due to the properties of PFOS, which binds preferentially to proteins in non-lipid tissues, application of numeric criteria for BCF or BAF, which are derived based on consideration of lipid-partitioning substances, may be inappropriate for PFOS. Most notable and alarming are the high concentrations of PFOS that
have been found in Arctic animals, far from anthropogenic sources. PFOS has been detected in higher trophic level biota and predators such as fish, piscivorous birds, mink, and Arctic biota. Also, predator species, such as eagles, have been shown to accumulate higher PFOS concentrations than birds from lower trophic levels. Even with reductions in manufacturing of PFOS by some manufacturers, wildlife, such as birds, can continue to be exposed to persistent and bioaccumulative substances such as PFOS simply by virtue of its persistence and long-term accumulation.

According to available data, PFOS meets the criteria for the potential for long-range transport. This is evident through monitoring data showing highly elevated levels of PFOS in various parts of the northern hemisphere. It is especially evident in the Arctic biota, far from anthropogenic sources. PFOS also fulfills the specific criteria for atmospheric half-life.

PFOS fulfills the criteria for adverse effects. It has demonstrated toxicity towards mammals in sub-chronic repeated dose studies at low concentrations, as well as rat reproductive toxicity with mortality of pups occurring shortly after birth. PFOS is toxic to aquatic organisms with mysid shrimp and *Chironomus tentans* being the most sensitive organisms.

The voluntary phase out of PFOS production by the major producer in the USA has led to a reduction in the current use of PFOS-related substances. However, PFOS or PFOS-related substances are still produced in some countries and it continues to be used in many countries. Given the inherent properties of PFOS, together with demonstrated or potential environmental concentrations that may exceed the effect levels for certain higher trophic level biota such as piscivorous birds and mammals; and given the widespread occurrence of PFOS in biota, including in remote areas; and given that PFOS precursors may contribute to the overall presence of PFOS in the environment, it is concluded that PFOS is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

### 3.2 Suggested risk management measures

As a POP, PFOS should be managed with the objective of achieving the lowest feasible level of releases to the environment taking into account the socioeconomic considerations in Annex F. Consideration should also be given to the potential for all PFOS-related substances to degrade to PFOS and thus contribute to the total environmental load.

Listing of PFOS under the Convention is able to address various aspects of substance life-cycles, including manufacture, use, sale, offer for sale, import and export as well as prescribing emissions measures e.g. BAT/BEP or others to reduce releases with the aim of eliminating them. Listing of PFOS under the Convention would also make it subject to the provisions on stockpiles and waste in Article 6.

**Listing of PFOS in Annex A** of the Convention would prohibit the manufacture, use, sale, offer for sale and import and export of PFOS and could be linked with specific exemptions that specify deadlines for the eventual elimination of remaining PFOS use. Such listing could also be coupled with a Part III of Annex A that would describe in more detail the critical uses of PFOS and/or PFOS-related substances and appropriate conditions for their use including timelines.

**Listing of PFOS in Annex B** of the Convention would prohibit the manufacture, use, sale, offer for sale and import and export of PFOS except for specified acceptable purposes/specific exemptions such as those mentioned above for which at present no alternatives are available. The listing could be accompanied by a Part III to Annex B, which would describe in more detail the critical uses of PFOS and/or PFOS-related substances and appropriate conditions for their use, including timelines for review and revision, as appropriate.

**Listing of PFOS in Annex C** of the Convention to address the unintentional production of PFOS as a transformation or degradation product of PFOS-related chemicals, would eventually eliminate all releases of PFOS and could potentially reduce or eliminate the
manufacture and use of all PFOS-related substances that are degraded to PFOS to any degree.

The suggested options for control measures for PFOS are as follows:

1. PFOS may be listed in Annex A, with or without specific exemptions, and accompanied with a new Part III of Annex A that details actions for each or groups of PFOS-related substances or uses of such substances; or

2. PFOS may be listed in Annex B, with specified acceptable purposes, and accompanied with a new Part III of Annex B that details actions for each or groups of PFOS-related substances or uses of such substances

3. PFOS may be listed in Annex C as an unintentional POP to capture all present and future uses of all, also presently unknown, PFOS-related substances that may give rise to PFOS when released into the environment; or

4. PFOS may be listed in Annex A or B, as described above, and at the same time also be listed in Annex C.

These options are further described below.

Option 1. Listing of PFOS in Annex A.
Listing of PFOS in Annex A would be consistent with the POPs properties of this intentionally produced substance. This would send a clear signal that production and use of PFOS should eventually be phased out. To allow for medium-term use of PFOS and PFOS-related substances in critical applications, an exemption for production and use could be given e.g. “as required to produce other chemicals substances to be used solely in accordance with Part III of this Annex”. Specific exemptions for certain critical uses could be difficult to develop or apply, however, given the general time constraint of five years, with a possible extension applicable to specific exemptions, among other reasons.

This option could be exercised by all Parties, in which case they would not need to register the exemption. This would also imply that any restrictions with regard to time would appear in the new Part III of Annex A. The information that has been supplied indicates that for some uses, such deadlines could be difficult to determine at present.

Option 2. Listing of PFOS in Annex B.
Listing of PFOS in Annex B would be consistent with the POPs properties of this intentionally produced substance. While allowing for some specified acceptable purposes/specific exemptions, it would still send a signal that production and use of PFOS should eventually be phased out. To allow for the medium-term use of PFOS-related substances in critical applications, an acceptable purpose for production of PFOS could be given e.g. “as required to produce other chemical substances to be used solely in accordance with Part III of this Annex”;

Option 3. Listing of PFOS in Annex C.
This option regards PFOS as an unintentionally produced POP that results from the degradation of PFOS-related substances. The argument behind this would be that PFOS is not any longer produced except as an intermediate for the production of PFOS-related substances to be used in the critical uses. Releases of PFOS to man and the environment would then only occur as a result of degradation of the PFOS-related substances. The weakness of the argument is that production of PFOS itself, which fulfils the POPs criteria and has been concluded by the Committee to “lead to significant adverse effects such that global action is warranted” would not be prohibited, but it would depend on voluntary commitments not to produce PFOS except as an intermediate. All uses of any PFOS-related substance that degraded to PFOS would be affected by the listing, in that all measures should be taken to reduce with the aim to eliminate the releases of PFOS as a result of that use. The responsibility to identify whether a specific use of a PFOS-related substance was degraded to PFOS would rest with the user.

However, Article 5 of the Treaty does not envision “unintentional production” to include substances that are the result of non-anthropogenic transformation processes. Accordingly,
listing PFOS in Annex C on the basis of it resulting from the degradation of other intentionally produced chemicals may not be an appropriate exercise.

In addition, listing PFOS in Annex C alone would not be appropriate because PFOS is and will continue to be produced intentionally for some years at least, and will not occur solely as the unintended result of transformation or degradation from other chemicals.

**Option 4. Listing of PFOS in Annex A or B and Annex C.**
This option could combine the listing of PFOS in Annex A or B, with the acceptable purposes/specific exemptions as noted above, with its listing as an unintentional POP in Annex C to capture existing and future uses of all substances that could potentially degrade to PFOS. This option would not add anything to either of the two options above.

**Conclusions**

In comparing options 1 and 2 with option 3 and 4, it seems most logical to regulate PFOS under the Convention as an intentionally produced POP, which should eventually be phased out. It is therefore proposed to list PFOS in Annex A or B of the Convention. Given the toxicity and extreme persistence of PFOS the goal should be elimination of emissions of PFOS. It is therefore suggested to list PFOS in Annex A of the Convention.

There is, however, a need for some remaining critical uses over the foreseeable future. To allow for this, one could introduce specific exemptions for production as required only to produce other chemical substances designated for these critical uses and to describe the conditions for the use of PFOS-related substances further in a new Part III to Annex A. A suggested outline of Part III can be found in the Appendix.

**4. Concluding statement**

In accordance with paragraph 9 of Article 8 of the Convention, the Committee recommends the Conference of the Parties to the Stockholm Convention to consider listing and specifying the related control measures of PFOS in Annex A as described above.
Part III.

PFOS

1. The manufacture, import, and use of PFOS-related substances shall be eliminated by all Parties except for the uses described below and except for the production of PFOS as an intermediate to produce other chemical substances for the uses described below.

A) Uses for which alternatives substances or technologies are available but need to be phased in.
   1. Metal plating.
   2. Fire Fighting Foam

B) Uses for which at present, according to responses received, no technically feasible alternatives are available
   1. Photoresists or anti reflective coatings for photolithography processes
   2. Photo mask rendering process
   3. Photo imaging
   4. Hydraulic fluids in aviation
   5. Ant baits for control of leaf-cutting ants

[For each of the uses, Part III could specify the conditions for use, e.g. closed systems whenever possible, no or negligible emissions/releases, recovery of spent PFOS-containing products, labelling of PFOS-containing articles, time limits for stockpiles (FFF), time limits for technology transfer (metal plating).]
### B.11. Short-chained chlorinated paraffins (SCCPs) – SUMMARY

**SUMMARY**

**11. SCCPs**

Draft Risk Profile May 2007


<table>
<thead>
<tr>
<th>Composition</th>
<th>SCCPs cover carbon chain lengths of 10 – 13 with 1 – 13 chlorine atoms. Production yields extremely complex mixtures, owing to the many possible positions for the chlorine atoms, and standard analytical methods do not permit their separation and identification. Thus the commercial mixture is proposed for listing.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uses</td>
<td>SCCPs are used primarily in metalworking applications. Other uses include uses as flame retardants or plasticizers in PVC, paints, adhesives, sealants in buildings, PCB substitutes in gaskets, leather fat liquors, and flame retardants in rubber, car carpets, textiles, and other polymers. SCCPs used as flame retardants are added to rubber in a proportion of 1–10%.</td>
</tr>
<tr>
<td>Releases</td>
<td>Anthropogenic releases of CPs into the environment may occur during production, storage, transportation, industrial and consumer usage of CP-containing products, disposal and burning of waste, and land filling of products such as PVC, textiles, painted materials, paint cans and cutting oils.</td>
</tr>
<tr>
<td>Fate</td>
<td>SCCPs are not expected to degrade significantly by hydrolysis in water, and dated sediment cores indicate that they persist in sediment longer than 1 year. SCCPs have atmospheric half-lives ranging from 0.81 to 10.5 days, indicating that they are also relatively persistent in air. SCCPs have been detected in a diverse array of environmental samples (air, sediment, water, wastewater, fish and marine mammals) and in remote areas such as the Arctic (which is additional evidence of long range transport). Evidence for the bioaccumulation of SCCPs is further supported by the high concentrations of SCCPs measured in marine mammals and aquatic freshwater biota (e.g., beluga whales, ringed seals and various fish).</td>
</tr>
<tr>
<td>Effects</td>
<td>SCCPs can harm sensitive aquatic organisms at relatively low concentrations (i.e. below threshold criteria of 1 mg/L used to categorize substances on Canada’s Domestic Substances List). SCCPs affect the liver, kidney and thyroid in rats including increased liver, weight, altered liver enzymes, and enlarged thyroid. Rodent studies showed dose related increases in adenomas and carcinomas in the liver, thyroid, and kidney. There continues to be contention over the mechanisms of these tumors and whether they are relevant for human health. SCCPs were classified as a group 2B carcinogen (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC). There are no data on fertility or developmental effects for humans.</td>
</tr>
<tr>
<td>Exposure</td>
<td>SCCPs have been found in breast milk from Inuit women in Northern Quebec in the UK. They have been measured in cow’s milk and in butter from Denmark, Wales, Normandy, Bavaria, Ireland and southern and northern Italy. SCCPs have been measured in seabird eggs, Arctic char, cod, blue mussel, spart, redfish, herring, halibut, sardine, trout, dab,</td>
</tr>
</tbody>
</table>
Flounder, beluga whale, walrus, ringed seal, reindeer, and osprey. SCCPs were measured in air in several countries including Canada, the United Kingdom (U.K.) and Norway. SCCPs were detected in all eight sewage treatment plant final effluents sampled from southern Ontario, Canada. Total SCCPs (dissolved and particulate C_{10-13}) ranged from 59 to 448 ng/L. The highest concentrations were found in samples from treatment plants in industrialized areas, including Hamilton, St. Catharine’s and Galt. SCCPs were detected in surface waters in Ontario and Manitoba, Canada. SCCPs were detected in sediments around the Great Lakes in Canada, Germany, Czech Republic and the United Kingdom. They have also been detected in Arctic sediment.

| Status       | In December 2006, the Parties to the UNECE POPs Protocol agreed that SCCPs should be considered as a POP as defined under the Protocol, and requested that the Task Force continue with the Track B reviews of the substances and explore management strategies for them. In 1995, OSPAR Commission for the Protection of Marine Environment of the North-East Atlantic adopted a decision on SCCPs (Decision 95/1). This established a ban on the use of SCCPs in all areas of application by the end of 1999 with exemptions for use of SCCPs in dam sealants and underground conveyor belts until 2004. Similar to OSPAR, the Baltic Marine Environment Protection Commission (HELCOM) has included SCCPs on their list of harmful substances. SCCPs have been identified as priority hazardous substances in the field of water policy under the Water Framework Directive (Directive 2000/60/EC of 23 October 2000) and are listed in the draft amendment of Directive 2000/60/EC, which defines water quality standards for European surface waters. Substances listed in this Directive will be subject to cessation or phasing out of discharges, emissions and losses with in an appropriate time table that shall not exceed 20 years (EC, 2005). The most important uses (metal working fluids and leather fat liquors) in the EU were restricted in directive 2002/45/EC. SCCP in plastics is a major use in Europe that was not covered by directive 2002/45/EC. |
| Alternatives | Will be discussed in Annex F evaluation if SCCPs advance. |
Short-chained chlorinated paraffins (SCCPs) – DETAILED PROFILE

Short-chained chlorinated paraffins (SCCPs)
Introduction

Short-chained chlorinated paraffins (SCCPs) are a group of synthetic compounds mainly used in metal working fluids, sealants, as flame retardants in rubbers and textiles, in leather processing and in paints and coatings.

The available data from remote areas clearly show contamination of biota and air by SCCPs. SCCPs are highly toxic to aquatic organisms. They do not break down naturally and tend to accumulate in biota. Their persistence, bioaccumulation, potential for long-range environmental transport and toxicity mean that they may have damaging environmental effects at a global level.

Data Sources


1. Identification of the chemical

SCCPs are n-paraffins that have carbon chain lengths of between 10 and 13 carbon atoms and a degree of chlorination of more than 48% by weight. There is a range of commercially available C10-13 chlorinated paraffins and they are usually mixtures of different carbon chain lengths and different degrees of chlorination, although all have a common structure in that no secondary carbon atom carries more than one chlorine.

Two other groups of chlorinated paraffins are made commercially. These are known as “mid, medium or intermediate chain length” (typically C14-17) and “long chain length” (typically C20-30). This dossier, however, concerns only the short chain length (C10-13) chlorinated paraffins.

1.1 Chemical Identity of the Proposed Substance

IUPAC\textsuperscript{72} Name: Alkanes, C\textsubscript{10-13}, chloro
Synonyms: alkanes, chlorinated; alkanes (C\textsubscript{10-13}), chloro-(50-70%); alkanes (C\textsubscript{10-13}), chloro-(60%); chlorinated alkanes; chlorinated paraffins; chloroalkanes; chlorocarbons; polychlorinated alkanes; paraffins chlorinated.

CAS\textsuperscript{73} No: 85535-84-8
EINECS\textsuperscript{74} No: 287-476-5

\textsuperscript{72} International Union of Pure and Applied Chemistry.
\textsuperscript{73} Chemical Abstracts Service.
\textsuperscript{74} European Inventory of Existing Chemical Substances.
1.2 Structure

Short chain chlorinated paraffins (SCCPs) are chlorinated derivatives of n-alkanes, having carbon chain lengths ranging from 10 to 13 and 1-13 chlorine atoms (Molecular formula: $C_xH_{2x-2y+2}Cl_y$, where $x=10-13$ and $y=1-13$). Chlorination of the n-alkane feedstock yields extremely complex mixtures, owing to the many possible positions for the chlorine atoms, and standard analytical methods do not permit their separation and identification. Thus the commercial mixture would fall under the proposed identity for SCCPs specified here.

The structure of two example SCCP compounds (C$_{10}$H$_{17}$Cl$_5$ and C$_{13}$H$_{22}$Cl$_6$) is shown below.

![Structure of SCCP compounds](image)

Molecular weight: 320-500

2. Persistence

Atmospheric half-lives of 1.9-7.2 days have been estimated for SCCPs (European Commission, 2000). Nevertheless, in the aqueous phase, rates of hydrolysis, photolysis with visible or near UV radiation, oxidation and volatilization are insignificant under ambient temperatures (Government of Canada, 1993). Photolytical degradation in aqueous media may take place, but at a very slow rate (Koh and Thiemann, 2001; El-Morsi et al. 2002).

SCCPs are not readily or inherently biodegradable in standard tests. It can be concluded from the simulation tests that SCCPs with low chlorine content (e.g. <50% wt Cl) may biodegrade slowly in the environment in the presence of adapted microorganisms. An additional simulation study on degradation of SCCPs in the marine environment has been required recently within the EU and the results of the study should be available by the end of 2006.

In its opinion given in 2003 (CSTEE 2003), the EU Scientific Committee on Toxicity, Ecotoxicity and the Environment came to the conclusion that SCCPs are potentially persistent (P) and possibly very persistent (vP). The Committee emphasised the evidence that SCCPs are occurring in remote areas and were of the view that this is particularly important evidence that gives further support to the P/vP classification. Weight of evidence indicates that the half-life of SCCPs in sediment is greater than 1 year.

The available information seems to indicate that SCCPs have long half-lives in the environment.

3. Bioaccumulation

Reported log K$_{ow}$ of different SCCPs range from 4.39 to 8.69, indicating a high potential for bioaccumulation (European Commission, 2000).
High bioconcentration factors (BCFs) in fish have been reported in the scientific literature (European Commission, 2000). In one of the key studies, whole body BCFs of 173-7 816 were determined based on radioactivity measurements in the fish and BCFs of 574-7 273 were determined based on the parent compound analysis (Madeley and Maddock 1983). In addition to these experimental values from laboratory studies, BCF values have been estimated in situ for Lake Trout (Salvelinus namaycush) in western Lake Ontario, i.e., the overall BCF for C10-13 SCCPs was 36 500 (Filyk et al. 2003). Bioconcentration in mussels has also been assessed with reported whole body BCFs ranging from 5 785 to 40 900 (European Commission 2000).

SCCP have high reported values of both log Kow and BCF for different aquatic species.

4. Potential for long-range environmental transport

Drouillard et al. (1998a) have determined vapour pressures for a range of SCCPs. In the EU Risk Assessment Report (European Commission, 2000), an assumed vapour pressure of an SCCP with chlorine content of approximately 50% of 0.0213 Pa at 40°C is used. Henry's Law constants range from 0.7 to 18 Pa m³/mol, near the values of some acknowledged POPs (Drouillard et al. 1998a). This constant and vapour pressure are the most important chemical characteristic to determine whether a substance may undergo long-range environmental transport in the atmosphere. As shown in table 1, Henry's Law constants are in the range of those for currently listed POPs. Therefore, based on their chemical properties, SCCPs are likely to undergo long-range environmental transport in the atmosphere. Moreover, atmospheric half-lives exceeding the screening criteria of 2 days (1.9-7.2 days) have been estimated for SCCPs (European Commission, 2000).

Table 1: Water solubility (WS), vapour pressure (VP) and Henry’s Law Constant (HLC) (at 25 °C) for SCCPs and currently listed POPs

<table>
<thead>
<tr>
<th>Substance</th>
<th>WS mg/L</th>
<th>VP Pa</th>
<th>HLC Pa m³/mol</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCCP-min</td>
<td>0.0224**</td>
<td>2.8 x 10⁻⁷*</td>
<td>0.7 *</td>
</tr>
<tr>
<td>SCCP-max</td>
<td>0.994**</td>
<td>2.5*</td>
<td>18*</td>
</tr>
<tr>
<td>POP-min</td>
<td>0.0012 (DDT)</td>
<td>2.5 x 10⁻⁵ (DDT)</td>
<td>0.04 (endrin)</td>
</tr>
<tr>
<td>POP-max</td>
<td>3.0 (toxaphene)</td>
<td>27 (toxaphene)</td>
<td>3726 (toxaphene)</td>
</tr>
<tr>
<td>POP-2nd max</td>
<td>0.5 (dieldrin)</td>
<td>0.04 (heptachlor)</td>
<td>267 (heptachlor)</td>
</tr>
</tbody>
</table>

* Drouillard et al. 1998a
** Drouillard et al. 1998b

SCCPs have been found in air samples from a remote area in the Canadian Arctic (Peters et al., 1998). The mean total concentrations (vapour + particulate phase) found were 20 ±32 pg/m³ at the remote site. Tomy (1997a; as reported in Tomy, 1998) found that SCCPs were present in air from Egbert, Canada, at a concentration of 65-924 pg/m³. Muir et al. (2001) reported SCCPs to be present at a concentration of 249 pg/m³ in air overlying the west basin of Lake Ontario. The levels of SCCPs in air from the Arctic have also been reported by Bidleman et al. (2001). The levels found ranged from 1.07 to 7.25 pg/m³ and were dominated by the contributions from chlorodecanes (C10 fractions).

The concentrations of SCCPs (vapour + particulate phase) reported in the Arctic environment ranged from 1.07 to 7.25 pg/m³ (Borgen et al., 2000) and from 1.8 to 10.6 ng/m³ (Bidleman et al., 2001). Tomy et al. (1997 and 1999) reported SCCPs in surface sediments from the Canadian mid-latitude and
Arctic regions, and attributed these to long-range transport. Stern (2003, as reported in Filyk, 2003) found levels of SCCPs in a lake sediment core taken from a very remote lake in Arctic Canada (75°34’N; 89°19’W) which provides evidence for transport to, and deposition in, the Arctic (Filyk et al. 2003).

SCCPs have been reported in marine mammals from various regions of the Arctic (Stern et al., 1997; Tomy et al., 1998). There is also evidence of SCCP accumulation in fish species from Lake Ontario (Muir et al., 2001).

The study of Stern et al. (1997) also detected SCCPs in three samples of breast milk taken from Inuit women living in settlements along the Hudson Strait of Canada. A study performed by Thomas et al. (2006) found similar SCCP concentrations in breast milk of women from the United Kingdom.

The ubiquity of SCCPs, the vapour pressures reported, and the Henry's Law Constant values (similar to those of acknowledged POPs), indicate that SCCPs are transported long range.

5. **Adverse effects**

According to EU Risk Assessment Report (European Commission, 2000) SCCPs are of low acute toxicity to fish, with 48 and 96-hour LC50 values in excess of the water solubility of the substance. Chronic toxicity values include a 60-day LC50 of 0.34 mg/L for rainbow trout and no observed effect concentrations of <0.040 and 0.28 mg/L for rainbow trout and sheepshead minnows respectively.

For aquatic invertebrates, SCCPs are of high toxicity with 24-hour EC50 values with daphnids ranging from 0.3 to 11.1 mg/L and with acute NOECs ranging from 0.06 to 2 mg/L. In 21-day tests with daphnids, EC50 values ranged from 0.101 to 0.228 mg/L and NOECs ranged from 0.005-0.05 mg/L. For algae, 96-hour EC50 values ranged from 0.012 to 3.7 mg/L, depending on the species.

Information available from acute studies and skin irritation studies in animals indicates that the intensity and nature of effects for these endpoints are independent of chain length and degree of chlorination. Assessment of the available data clearly indicates that SCCPs are of low acute toxicity in animals. In rodent carcinogenicity studies, dose-related increases in the incidence of adenomas and carcinomas were observed in the liver, thyroid and kidney. Other cancers seen were dismissed as not significant. Consideration of the likely underlying mechanisms for these tumours suggests that they are not relevant to human health.

There are no data available on the effects of SCCPs on fertility in humans or animals. An SCCP produced developmental effects in rats at a dose which also caused maternal toxicity (2 000 mg/kg body weight). No developmental effects were observed in a study in rabbits, although maternally toxic doses were not tested. NOAELs for general toxicity of 100 and 1 000 mg/kg/day were identified in rats and mice respectively.

The relevance of the finding that medium-chain length chlorinated paraffins can cause a severe effect (internal haemorrhaging leading to deaths) in suckling rat pups has been recently discussed (European Commission, 2005).

The current EU hazard classification for SCCPs is: Carc. Cat. 3; R40 - N; R50-53 (Risk Phrases: R40: Limited evidence of a carcinogenic effect; R50/53: Very toxic to aquatic organisms; may cause long-term adverse effects in the aquatic environment.). Also the International Agency for Research on Cancer (IARC) has designated SCCPs (as a group) as possible carcinogens.
In summary, SCCPs are of high aquatic toxicity to a variety of species, and its terrestrial toxicity may be an additional cause of concern. SCCPs are also possible carcinogens.

REASONS FOR CONCERN

SCCPs are highly toxic to aquatic organisms and it is considered as a possible carcinogen. SCCPs do not break down naturally and tend to accumulate to biota. The available data from remote areas clearly show contamination of the environment and biota by SCCPs. Their persistence, bioaccumulation and toxicity mean that they may have damaging environmental effects at a global level. Overall, it can be considered that SCCPs meet the screening criteria for persistence, potential to cause adverse effects, bioaccumulation and potential for long range environmental transport.

Placing on the market and use of SCCPs have been restricted over the last years in the European Union but no total prohibition has yet been foreseen. On the other hand, production and use of SCCPs continues unrestricted in many other countries. As SCCPs can move in the atmosphere far from [their] sources, single countries or groups of countries alone cannot abate the pollution caused by [them]. Due to the harmful POP properties and risks related to its widespread production and use, international action is warranted to control this pollution."
RISK PROFILE

Executive Summary

Releases of short-chain chlorinated paraffins (SCCPs) can occur during production, storage, transportation, and use of SCCPs. Facility wash-down and spent metalworking / metal cutting fluids are sources to aquatic ecosystems. Although data are limited, the major sources of release of SCCPs are likely the formulation and manufacturing of products containing SCCPs, such as polyvinyl chloride (PVC) plastics, and use in metalworking fluids. While historical use of SCCPs was high in several countries, major reductions have been noted in recent years.

SCCPs are not expected to degrade significantly by hydrolysis in water, and dated sediment cores indicate that they persist in sediment longer than 1 year. SCCPs have atmospheric half-lives ranging from 0.81 to 10.5 days, indicating that they are also relatively persistent in air. The Henry’s law constant indicates that there may be substantial partitioning from water to air under certain conditions, thus facilitating atmospheric partitioning and transport. SCCPs have been detected in a diverse array of environmental samples (air, sediment, water, wastewater, fish and marine mammals) and in remote areas such as the Arctic (which is additional evidence of long range transport). In addition, Arctic Contamination Potential (ACP) modeling and OECD LRTP screening tools suggests that SCCPs have moderate ACP when emitted to air and have properties similar to known POPs that undergo long range transport.

Bioaccumulation factors (BAFs) of 16 440–25 650 wet weight (wet wt.) in trout from Lake Ontario indicate that SCCPs can bioaccumulate to a high degree in aquatic biota. This is supported by modeling data for log Kow and bioaccumulation factors which indicate that SCCPs bioaccumulate. In addition, biomagnification factors for some SCCPs have been found to be greater than 1. High concentrations of SCCPs in upper trophic level organisms is additional evidence of bioaccumulation. Evidence for the bioaccumulation of SCCPs is further supported by the high concentrations of SCCPs measured in marine mammals and aquatic freshwater biota (e.g., beluga whales, ringed seals and various fish). High concentrations of SCCPs have also been measured in the breast milk of Inuit women in Northern Quebec.

There is evidence that SCCPs are toxic to sensitive aquatic organisms at relatively low concentrations. The most sensitive organism, Daphnia magna, has chronic NOECs of 5 µg/L.

The weight of evidence supports the conclusion that SCCPs are persistent, bioaccumulative, inherently toxic to some species, and undergo long range transport to remote areas. Concentrations of SCCPs currently measured in the environment are generally below those associated with effects. However, because of their widespread distribution, persistence and accumulation, they continue to have potential for long-term harmful effects.

The increasing regulation of SCCPs in a few geographical areas have resulted in a decrease in SCCPs currently in use and released into the environment. However, evidence suggests that significant amounts are still in use and being released in several countries. The available empirical and modeled data strongly indicate that SCCPs are persistent, bioaccumulative, and toxic to aquatic organisms at low concentrations, and undergo long range environmental transport. In December 2006, the Parties to the UNECE POPs Protocol agreed that SCCPs should be considered as a POP as defined under the Protocol.

Concentrations currently measured in the environment are generally below levels that have been associated with effects in laboratory studies. In some cases, concentrations are approaching those that may be of concern, for example in secondary consumers, and elevated levels have been measured in human breast milk, including in remote communities. Particularly in view of SCCPs persistence, bioaccumulation and their inherent toxicity to a range of organisms, it is considered that SCCPs are likely to cause significant adverse effects as a result of long range transport.
Based on the available evidence, it is thus likely that SCCPs can, as result of long range environmental transport, cause significant adverse effects on human health and/or the environment, such that global action is warranted.

**Data Sources**

The risk profile for SCCPs builds on information gathered by the EU in its proposal of SCCPs to the POPRC (UNEP/POPS/POPRC.2/INF/6). The risk profile also incorporates information collected from risk assessment documents prepared by Canada (Environment Canada) and the United Kingdom (DEFRA). Annex E information submissions from several POPRC parties and observers were also reviewed and any additional information incorporated as appropriate. Some additional information from peer reviewed scientific literature (as of February 1, 2007) is also included.

1. **STATUS OF THE CHEMICAL UNDER INTERNATIONAL CONVENTIONS**

In August, 2005, the European Community proposed SCCPs to be added to the UNECE Convention on Long Range Transboundary Air Pollution, Protocol on Persistent Organic Pollutants (LRTAP). SCCPs were proposed to meet the criteria of decision 1998/2 of the Executive Body for persistence, potential to cause adverse effects, bioaccumulation and potential for long range transport. At the 24th session of the Executive Body in December 2006, the Parties to the UNECE POPs Protocol agreed that SCCPs should be considered as a POP as defined under the Protocol, and requested that the Task Force continue with the Track B reviews of the substances and explore management strategies for them.

In 1989, as a result of laboratory testing in animals, SCCPs were classified as a group 2B carcinogen by the International Agency (IA) for Research on Cancer (IARC).

In 1995, OSPAR Commission for the Protection of Marine Environment of the North-East Atlantic adopted a decision on SCCPs (Decision 95/1). This established a ban on the use of SCCPs in all areas of application. Under this decision, all sale and use of SCCPs should be prohibited by the end of 1999. Exemptions will allow the use of SCCPs in dam sealants and underground conveyor belts until 2004. Similar to OSPAR, the Baltic Marine Environment Protection Commission (HELCOM) has included SCCPs on their list of harmful substances (no recommendations have yet been taken).

SCCPs have been identified as priority hazardous substances in the field of water policy under the Water Framework Directive (Directive 2000/60/EC of 23 October 2000) and are listed in the draft amendment of Directive 2000/60/EC, which defines water quality standards for European surface waters. Substances listed in this Directive will be subject to cessation or phasing out of discharges, emissions and losses with in an appropriate time table that shall not exceed 20 years (EC, 2005).

The most important uses (metal working fluids and leather fat liquors) in the EU were restricted in directive 2002/45/EC. SCCP in plastics is a major use in Europe that was not covered by directive 2002/45/EC.

2. **Summary information relevant to the risk profile**

2.1 **Sources**

2.1.1 **Production**

Total reported annual usage of all chlorinated paraffins (CPs) in Canada (production + imports – exports) was approximately 3000 tonnes in 2000 and 2001 (Environment Canada 2003a). The Canadian sales pattern for SCCPs (as a proportion of total usage of chlorinated paraffins) is similar to the...
European sales pattern, rather than the North American sales pattern, which is dominated by the United States (Table 1).

Whether these sales patterns are the same at present is not known. North American demand for total CPs fluctuates depending on the strength of the economy (Camford Information Services 2001). Overall, SCCP uses have declined within the EU, in part owing to the phasing out of production and use in Germany [Stolzenberg 1999; OSPAR 2001] and the EU marketing and Use Directive.

Table 1: Sales of CPs in the EU and North America during the 1990s

<table>
<thead>
<tr>
<th>CP group</th>
<th>EU¹</th>
<th>North America²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Year (tonnes/year)</td>
<td>% total CPs sales</td>
</tr>
<tr>
<td>SCCPs</td>
<td>1994 13200</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1997 7370</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1998 4080 6.4</td>
<td></td>
</tr>
</tbody>
</table>

¹ OSPAR (2001).
² CPIA (2000).

In addition to production in the US and the EU, it should be noted that there are CPs (of various chain length) producers in Russia, India, Taiwan, China and Japan. In some cases, total CPs are produced in Asia under licence to the European manufacturer. It is unclear to what extent imports from these countries are accounted for in the information provided by industry associations such as Eurochlor and CPIA (see Table 1). There is no production of SCCPs in Canada (Camford Information Services 2001).

Information submitted under Annex E of the Stockholm convention indicated that SCCPs were produced in the former Czechoslovakia (Novaky, Slovakia), though quantities are not known. Japan also noted that there is a possibility of 1% in medium chained chlorinated paraffin production. Germany noted that there has been no production in Germany since 1995. Prior to 1995, Clariant, Hoechst, and Huels produced SCCPs in Germany. Hoechst produced between 9300 - 19300 tonnes/year in Germany between the years 1993 and 1995.

As noted in the Annex E information submitted by the USA, some chlorinated paraffins are on the Toxic Substances Control Act (TSCA) inventory and are subject to the Environmental Protection Agency's (EPA's) TSCA inventory update reporting rule under which production and import information is collected. The CAS numbers used in the United States are not specific to SCCPs, hence the information collected includes other chain-length chlorinated paraffins. For 2002, the production and import volumes reported for CAS# 63449-39-8 (paraffin waxes and hydrocarbon waxes, chloro) were in the range of >50 million – 100 million pounds (>23 million – 45 million kg), and for CAS # 61788-76-9 (alkanes, chloro; chloroparaffins) in the range of >50 million – 100 million pounds (>23 million – 45 million kg). In 1994, for CAS # 68920-70-7, (alkanes, C₆-18, choro) production and import volume in the range of >1 million – 10 million pounds (>0.45 million – 23 million kg) were reported.

Annex E information submitted by Brazil indicates that 150 tons/year of SCCPs are produced in Brazil.

2.1.2 Uses
Nearly all reported usage of SCCPs in Canada was for metalworking applications. Minor uses included use as a flame retardant in plastics and
rubber. European use pattern data for SCCPs from the years 1994 and 1998 are given in Table 2.
Table 2: Applications of SCCPs in Europe

<table>
<thead>
<tr>
<th>Application</th>
<th>1994 data</th>
<th>1998 data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>tonnes/year</td>
<td>% of total use</td>
</tr>
<tr>
<td>Metalworking lubricants</td>
<td>9,380</td>
<td>71.02</td>
</tr>
<tr>
<td>PVC plasticizers</td>
<td>Note 3</td>
<td>Note 3</td>
</tr>
<tr>
<td>Paints, adhesives and sealants</td>
<td>1,845</td>
<td>13.97</td>
</tr>
<tr>
<td>Leather fat liquors</td>
<td>390</td>
<td>2.95</td>
</tr>
<tr>
<td>Rubber/flame retardants/textiles/polymers (other than PVC)</td>
<td>1,493</td>
<td>11.31</td>
</tr>
<tr>
<td>Other</td>
<td>100</td>
<td>0.75</td>
</tr>
<tr>
<td>Total</td>
<td>13,208</td>
<td>100</td>
</tr>
</tbody>
</table>

1 Data from Euro Chlor (1995).
2 Data from OSPAR (2001) from Western Europe.
3 The given data did not include information specifically on usage in PVC.

The use of SCCPs in the EU in metalworking (and also in fat liquoring of leather) is now subject to marketing and use restrictions. EU Directive 2002/45/EC, which was adopted in June 2002, restricts the concentration of SCCPs in metalworking and leather fat liquoring preparations to 1% or less. The use of SCCPs in these applications has decreased significantly since the release estimates initially used in the European risk assessment of SCCPs (EC 2000) were obtained (U.K. Environment Agency 2003c).

As noted in Annex E submissions, 70 tonnes of SCCPs were used in Switzerland in 1994 and while newer data does not exist, it is estimated that uses have reduced by 80%. The most widespread use of SCCPs in Switzerland was in joint sealants where it was often used instead of PCBs in buildings. Canton Basel-Town (2001) found that 15 out of 44 joint sealants used in schools and kindergartens contained SCCPs with a content of 2-34%. Canton Argau (2003) found that 18 out of 54 joint sealants sampled (years 1960-1976) and 7 out of 29 joint sealants sampled (years 1974-2002) contained SCCPs (Kantonales Laboratorium Basel-Stadt., 2001; 2003).

As noted in Annex E submission by Germany, the most important uses (74% of the total) of SCCPs were banned by the EU directive 2002/45/EC. SCCPs have been used as a PCB substitute in gaskets (e.g. splices, in buildings) and this may be a source when buildings are renovated.

Annex E information submitted by Brazil indicates that 300 tons/year is used in Brazil for the purposes of flame retardant in rubber, car carpet and accessories. It was noted that leather processing and use in paints was not relevant.

Use of SCCPs in Australia decreased by 80% between 2001 to 2003 to approximately 25 tonnes per annum of SCCPs in the metal working industry (NICNAS, 2004).

**Plastics and Rubber**

SCCPs are not used in PVC in the EU (U.K. Environment Agency 2003a). CPs with high chlorine contents (e.g., 70% by weight) can be used as flame retardants in natural and synthetic rubbers (Zitko and Arsenault 1974). All chain lengths of CPs appear to be used in rubber where they have a plasticizing and flame retarding function. An important use for flame retarded rubber appears to be in conveyor belts for mining applications, but the rubber is also used in other applications. In Canada, 8% of CP usage is as a flame retardant in heavy-duty rubber (Government of Canada 1993b). The amount of
CP added is generally in the range 1–4% by weight (Zitko and Arsenault 1974), but can be up to 15% by weight for some applications (BUA 1992).

The results of a survey for the British Rubber Manufacturers’ Association was carried out (BRMA 2001) and found that 10.1 – 16.8% of CPs in conveyor belting rubber was in the form of SCCP with approximately 48-51 tonnes/year being used at the site. Other unidentified CPs (probably SCCPs) included 6.5% (6 tonnes/year) used in shoe soles, and 13% (1.2 tonnes/year) used in industrial sheeting (U.K. Environment Agency, 2001).

**Adhesives/sealants**

Various CPs, including SCCPs are used as plasticizers/flame retardants in adhesives and sealants. Examples include polysulphide, polyurethane, acrylic and butyl sealants used in building and construction and in sealants for double- and triple-glazed windows. The CPs are typically added at amounts of 10–15% by weight of the final sealant, but could be added at amounts up to 20% by weight of the final sealant in exceptional cases.

**Paints**

CPs are used as plasticizers, binders and flame retardants in paints. The concentrations used are usually in the range 5–15% by weight. They are reported to be used in marine paints based on chlorinated rubber. Such paints may contain CPs with 70% chlorine by weight as binder and CPs with 40% chlorine by weight as plasticizer (Zitko and Arsenault 1974). For paints and coatings, there is a general move away from CP-containing products to higher solids/lower volatile organic compound alternative coatings such as epoxies as a result of increased controls on emissions of volatile organic compounds (U.K. Environment Agency 2001).

### 2.1.3 Releases to the environment

There is currently no evidence of any significant natural source of CPs (U.K. Environment Agency 2003b). Anthropogenic releases of CPs into the environment may occur during production, storage, transportation, industrial and consumer usage of CP-containing products, disposal and burning of waste, and land filling of products such as PVC, textiles, painted materials, paint cans and cutting oils (Tomy et al. 1998a). The possible sources of releases to water from manufacturing include spills, facility wash-down and storm water runoff. CPs in metalworking/metal cutting fluids may also be released into aquatic environments from drum disposal, carry-off and spent bath use (Government of Canada 1993a). These releases are collected in sewer systems and ultimately end up in the effluents of sewage treatment plants.

The major source of releases of SCCPs in the EU was from metalworking applications (EC 2000). Another significant source of release of CPs to the environment is from losses during the service life of products containing CP polymers (PVC, other plastics, paints, sealants, etc.) (EC 2000; U.K. Environment Agency 2003b). These releases are predicted to be mainly to urban/industrial soil and to wastewater.

Data since 1999 reported to Canada’s National Pollutant Release Inventory (NPRI) found that very small amounts of CPs (short, medium and long chain) are being released to the Canadian environment by companies that meet the NPRI reporting requirements (Environment Canada, 2005). In 2001-2002, the NPRI found 1.45 tonnes CPs for disposal to landfill and 1.94 tonnes recycling by recovery of organics from two companies in Ontario. Both of these
companies use SCCPs as a formulation component in the manufacture of wires and cables and of paints and coatings, respectively. In 2005, NPRI found that one company in Ontario disposed 0.023 tonnes of Alkanes 10-13, chloro (CAS# 85535-84-8) off-site and 2.967 tonnes were recycled off-site.

In the USA, SCCPs are subject to the Toxic Release Inventory (TRI) reporting as part of a broader category of polychlorinated alkanes (C10-13). As described in the USA Annex E submission, a total of 62023 lb (28133 kg) was reported for on and off site disposal or other releases, 4220 lb (1914 kg) of which were releases to air and water (TRI, 2004 data for all US industries in the polychlorinated alkanes category, data release April 12, 2006).

**Releases from production**

Releases from production sites are thought to be low. Default release estimates from production can be obtained using the emission factors contained in Appendix 1 of the EU (2003) Technical Guidance Document (TGD). These are carried out for a typical production site, assuming production of around 10 000–20 000 tonnes/year. The default emission factors (Table A1.1 of Appendix 1 of the TGD: Main Category 1c; VP <1 Pa) are 0 to air and 0.003 (0.3%) to wastewater.

*There are no producers of SCCPs in Canada and there was previously only one producer of MCCPs and LCCPs in Canada (Pioneer Chemicals Inc. - PCI, Canada). While PCI's production capacity in the year 2000 was 8500 tonnes (Camford Information Services 2001), it is currently not producing any chlorinated paraffins.*

**Releases from formulation of metalworking fluids**

Losses of CPs could occur during blending of metalworking fluids. It has been estimated that the likely loss of lubricant at a formulation site would typically be in the region of 1%, with a maximum of 2% (EC, 2000). Most of these losses would be controlled losses, such as off-specification material that could not be reused, and would be collected and sent for disposal. The largest consumer of CPs in Sweden (1400 tonnes/year) has estimated its emissions as 0.06 g/kg CP consumed (KEMI 1991). The European assessment (EC, 2000) estimated that the loss of SCCPs was 23 tonnes/year in Europe in the mid-1990s. According to the EU TGD (EU 2003), default emission factors for all CPs for the formulation of metalworking fluids are 0.005% to air and 0.25% to wastewater before any on-site treatment, which small blending businesses may not have.

**Releases from production of rubber**

SCCPs is used as a flame retardant, softener or process oil in rubber. SCCPs used as flame retardants are added to rubber in a proportion of 1–10%. The U.K. draft risk assessment (U.K. Environment Agency 2003b) discussed the release estimates in the TGD (EU 2003) and Use Category Document (BRE 1998) for plastics additive substances used in the polymer industry, such as MCCPs: the release factors for flame retardants (Use Category 22) during the polymer processing step for thermoplastics are 0.1% to air (boiling point <300°C/unknown; VP <1 Pa) and 0.05% to wastewater (Table A.3.11 of Appendix 1 of the TGD). For thermosetting resins, the release factor to air is 0 and the release factor to wastewater is 0.0005 (0.05%). The EU updated risk assessment of SCCPs (U.K. Environment Agency 2003a) also uses release factors for SCCPs from rubber production from the Use Category Document (BRE 1998). The release factors used are not specified in the assessment, but are assumed to be the same as or similar to those for MCCPs.
Releases from production of textiles and polymeric materials
SCCPs are used in fire proofing of cellulosic textiles and in other polymers, representing about 17% of SCCP use in 1998 (EC 2000). In some applications (e.g., waterproofing fabrics) small amounts of CPs could be applied directly to the textile in an emulsion, which may cause releases to wastewater (EC 2000; U.K. Environment Agency 2001). Releases could occur from washing of these textiles, which are generally used in furniture and other interior decorations.

Releases from production of paints and sealants
Some SCCPs are used in paints to a small extent. Losses to air and wastewater from formulation of SCCP-containing paints and coatings are estimated to be insignificant (U.K. Environment Agency 2003a). Losses to wastewater during the manufacture of sealants are reported to be low or zero. Scrap material and machine cleaning can account for up to 5% solid waste.

Releases from use of CP-containing products

Releases from PVC, paints, adhesives and sealants
Losses from the use of SCCPs in paint and sealants are generally regarded as much lower than those from metalworking. The updated SCCP assessment (U.K. Environment Agency 2003a) discusses these losses, but does not give percent losses per unit volume or weight of paint. A considerable amount of the CP-containing paints may end up as waste during the application process and therefore be disposed of in landfill sites. Immediate losses of CPs to air and wastewater during paint application should be minimal due to the low VPs and water solubilities of CPs. However, there will be gradual losses to air and water after application. The draft U.K. assessments of MCCPs and LCCPs (U.K. Environment Agency 2001, 2003b) examined this issue in more detail, as discussed below, and its conclusions should be applicable to SCCPs, given their structural similarities.

Losses from volatilization

Rubber
SCCPs are reported to be used in rubber, with applications mostly in high-density conveyor belts (EC 2000). Releases from use of SCCPs in polymers such as rubber or PVC may also occur via volatilization or from loss of polymeric material as particles during wear and abrasion of the products. Belts may also be recycled into other products at the end of their life. Volatilization of 0.05% of the MCCPs during the lifetime of the product was assumed, and it is reasonable to assume that this would be the case for SCCPs as well.

Losses from leaching

Paints and varnishes
For the industrial application of paints containing MCCPs and LCCPs, the U.K. risk assessments used default emission factors taken from the Use Category Document (BRE 1998), which are 0 to air and 0.1% to wastewater (U.K. Environment Agency 2003b). The same emission factors apply to domestic use of paints. The domestic usage of this type of paint is unknown but likely to be very limited (U.K. Environment Agency 2001, 2003b).
Leaching of MCCPs and LCCPs following application of paints and sealants could be relatively high. The U.K. draft risk assessment assumed a release factor for MCCPs to water for outdoor use in paints and sealants of 0.15% per year over 5–7 years and the same fractional release over the 20- to 30-year lifetime of sealants. No estimate of leaching loss from paints was available in the EU assessment of SCCPs (EC 2000); however, it can be assumed to be similar to that for MCCPs/LCCPs.

**Releases from use of metalworking fluids**

Losses of SCCPs due to carry-off from workpieces were estimated to be 2.5 kg/site per year for a small user (100-L capacity) and 2500 kg/site per year for a large user (95 000-L capacity) based on the early 1990s (Government of Canada 1993a). The estimated annual losses of CPs from cutting fluid, based on the replacement rates, are thought to be 48% for a large machine shop, 75% for a medium-sized machine shop and 100% for a small machine shop (EC 2000). Not all of this loss is to wastewater. Releases of SCPPs from use in metalworking fluids to wastewater streams in the EU were estimated to be 18% of use (or 733 tonnes/year in 1998) (EC 2000). In addition, the EU assessment estimated that about 3% of metalworking use would be disposed of in landfill. A breakdown of releases of CPs from small and large machine shops is provided in Table 3.

**Table 3:** Total losses of CPs for a large and small machine shop using oil-based cutting fluids (from EC 2000; U.K. Environment Agency 2001, 2003b)

<table>
<thead>
<tr>
<th></th>
<th>Large facility with swarf reprocessing</th>
<th>Small facility with no swarf reprocessing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Misting/evaporation</td>
<td>2% to air</td>
<td>2% to air</td>
</tr>
<tr>
<td>Overalls</td>
<td>1% to water</td>
<td>2% to water</td>
</tr>
<tr>
<td>Leaks</td>
<td>1% to water*</td>
<td>3% to water*</td>
</tr>
<tr>
<td>Dragout/swarf</td>
<td>27% incinerated</td>
<td>81% incinerated</td>
</tr>
<tr>
<td></td>
<td>3% to landfill</td>
<td>9% to landfill</td>
</tr>
<tr>
<td>Dragout/workpiece</td>
<td>1% to water</td>
<td>1% to water</td>
</tr>
<tr>
<td></td>
<td>2% chemical waste</td>
<td>2% chemical waste</td>
</tr>
<tr>
<td>Internal reprocessing</td>
<td>1% to water*</td>
<td></td>
</tr>
<tr>
<td>External reprocessing</td>
<td>10% reused/discarded as waste oil</td>
<td></td>
</tr>
<tr>
<td>Total losses</td>
<td>48%</td>
<td>100%</td>
</tr>
</tbody>
</table>

*These losses may be further minimized by collecting the cutting fluid for reuse.

The U.S. EPA (1993) assessment estimated that approximately 90% of CPs used in water-based metalworking fluids were removed via pre-discharge separation or treatment, so that only 10% of CPs in water-based metalworking fluids were assumed to be discharged to wastewater. Assuming removal from degradation and sorption to solids, actual releases from WWTPs were much less than estimated in the U.K. Environment Agency (2001, 2003b) draft assessments. In a critique of the EU assessment, CPIA (2000) indicated that estimates developed by the U.S. EPA are more relevant and appropriate for use in the USA than the estimates derived by the United Kingdom for the EU.

The EU estimates that 80% of SCCPs are used in oil-based metalworking fluids and only 20% in water-based applications (EC 2000).
U.K. Environment Agency (2003b) estimated releases for metal cutting/metalworking fluid additives using release factors from Appendix 1 of the TGD (EU 2003) to be 18.5% from oil-based fluids and 31.6% from water-based (emulsifiable) fluids. Emissions to air are estimated at 0.02% from both types of fluids. These release estimates from the TGD are worst-case, default estimates.

**Releases from leather fat liquid and carbonless copy paper**

According to industry data, there are no uses of CPs in North America in leather fat liquid and carbonless copy paper. The use of SCCPs in leather fat liquors has been restricted in the EU. Beginning January 6, 2004, EU Directive 2002/45/EC restricts the concentration of SCCPs in leather fat liquor preparations to 1% or less. The updated risk assessment of SCCPs (U.K. Environment Agency 2003a) assumes no use of SCCPs in leather fat liquor.

**Releases from other uses**

CPIA (2002), in a review of the Canadian SCCP United Nations Economic Commission for Europe (UNECE) information dossier (Environment Canada 2003b), has indicated that releases from gear oil packages, fluids used in hard rock mining and equipment used in other types of mining, fluids and equipment used in oil and gas exploration, manufacture of seamless pipe, metalworking and operation of turbines on ships may explain the presence of SCCPs in remote environments. However, there is currently insufficient information to assess the relative importance of these activities to the presence of SCCPs in the Arctic.

**Releases during disposal**

Landfilling is a major disposal route for polymeric products in Canada. CPs would be expected to remain stabilized in these products, with minor losses to washoff from percolating water. Leaching from landfill sites is likely to be negligible owing to strong binding of CPs to soils. Minor emissions of these products, which are effectively dissolved in polymers, could occur for centuries after disposal (IPCS 1996).

The releases and bioavailability of CPs from polymers that are landfilled or from losses of polymeric material as particles during wear and abrasion of flooring, rubber products, etc., are unknown. These releases could be sources of input of CPs to air and soils in urban and industrial areas (U.K. Environment Agency 2001, 2003a,b). Polymer-incorporated CPs could also be released during recycling of plastics, which may involve processes such as chopping, grinding and washing. If released as dust from these operations, the CPs would be adsorbed to particles because of high sorption and octanol–air partition coefficients.

**Overall emissions in Europe and estimates for North America**

Fractional losses of SCCPs to wastewater and surface waters have been estimated based on EU data (EC 2000) and are summarized in Table 4. Behaviour similar to that of MCCPs (U.K. Environment Agency 2003b) is assumed. Overall most releases of SCCPs are expected to be associated with metal working operations, however there is potential for widespread release in small amounts associated with uses in products (e.g. paints, textiles, rubber).
Table 4: Estimated fractional losses of SCCPs in the EU to wastewaters, surface waters and the terrestrial environment

<table>
<thead>
<tr>
<th>Application</th>
<th>Release to each compartment</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wastewater</td>
<td>Surface water</td>
<td>Terrestrial</td>
</tr>
<tr>
<td>Metalworking lubricants</td>
<td>18%</td>
<td>1.4%</td>
<td>17.8%</td>
</tr>
<tr>
<td>Paints and sealants</td>
<td>0.1%</td>
<td>0.015%</td>
<td>Unknown</td>
</tr>
<tr>
<td>Unknown — Landfilling of used material</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rubber/flame retardants/textiles/polymers (other than PVC)</td>
<td>0.1%</td>
<td>0.05–0.4%</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Landfilling of used material</td>
</tr>
</tbody>
</table>

1 Wastewater during use (metalworking fluids) or product formulation (paints/polymers).
2 For metalworking fluids, surface water = 0.08 × wastewater. For PVC and paints/adhesives/sealants, direct losses to surface water are included.
3 Terrestrial = soil + landfilling/burial, assuming landfilling or sludge spreading, except for PVC and paints/adhesives/sealants, where direct losses to urban/industrial soils need to be considered.

2.2 Environmental Fate

2.2.1. Persistence

**Persistence in Air**

SCCPs have VPs (2.8 $\times 10^{-7}$ to 0.028 Pa) and HLCs (0.68–18 Pa·m$^3$/mol for C$_{10-12}$ congeners) that are in the range of VPs and HLCs for some persistent organic pollutants that are known to undergo long-range atmospheric transport under the 1979 UNECE Convention on Long Range Transboundary Air Pollution (e.g., hexachlorocyclohexane [lindane], heptachlor, mirex). In general, the HLC values reported imply partitioning from water to air or from moist soils to air.

SCCPs were detected in four individual samples of air collected at Alert at the northern tip of Ellesmere Island in the high Arctic. Concentrations ranged from <1 to 8.5 pg/m$^3$ in gas-phase samples (see Table 11). The occurrence of SCCPs at a remote site like Alert indicates that the substance is subject to long range transport. The occurrence of long range transport is one line of evidence suggesting that SCCPs are persistent chemicals.

Tomy et al. (2000) reported SCCPs in the blubber of ringed seal from Eureka, southwest Ellesmere Island, beluga whales from northwest Greenland and the Mackenzie Delta and walrus from northwest Greenland at concentrations ranging from 199 to 626 ng/g wet wt. Tomy et al. (2000) also observed that the concentration profiles for the Arctic marine mammals show a predominance of the shorter carbon chain length congeners, i.e., the C$_{10}$ and C$_{11}$ formula groups. Drouillard et al. (1998a) showed that these congeners are the more volatile components of SCCP mixtures, which show a trend of decreasing VPs with increasing carbon chain length and degree of chlorination. SCCPs have also been detected in sediments from Hazen Lake and Lake DV09 in the Arctic, at levels of 7 and 17 ng/g dry wt., respectively (Tomy et al. 1998a; Stern and Evans 2003).

Although CPs do not degrade by direct photolysis in air, they theoretically would be subject to attack via hydroxyl radicals in the troposphere (Bunce 1993). Estimated atmospheric half-lives for SCCPs based on reaction with hydroxyl radicals are shown in Table 5 and range from 0.81 to 10.5 days, using the default atmospheric hydroxyl radical concentration of $1.5 \times 10^{6}$ molecules/cm$^3$ during sunlight hours in AOPWIN (v. 1.86) computer program.

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75 The VP of lindane is $4.3 \times 10^{-3}$ Pa (IPCS 1991), the VP of heptachlor is $3.0 \times 10^{-6}$ Pa (IPCS 1984a) and the VP of mirex is $2.3 \times 10^{-9}$ Pa (IPCS 1984b). The HLCs of lindane and heptachlor are 0.13 and 0.02 Pa·m$^3$/mol, respectively.
In the risk assessment methodology used in the EU, a lower hydroxyl radical concentration of $5 \times 10^5$ molecules/cm$^3$ is generally used as a daily (24-hour) average, which is typically found in relatively unpolluted air. Use of the latter hydroxyl radical concentration results in atmospheric half-lives ranging from 1.2 to 15.7 days (Table 5). It should be noted that hydroxyl radical reaction rates vary temporally with average daily sunlight, and $5 \times 10^5$ molecules/cm$^3$ may not be typical of northern latitudes since hydroxyl radical concentrations decline with latitude. In addition, the high adsorption of CPs to atmospheric particles at low temperatures, typical of Canadian conditions, may limit the atmospheric oxidation pathway.

Table 5: Estimated atmospheric half-lives for SCCPs calculated using the Syracuse Research Corporation AOPWIN computer program

<table>
<thead>
<tr>
<th>Example structure</th>
<th>Chlorine content (% by weight)</th>
<th>Estimated k$_{OH}$ (cm$^3$/molecule per second)</th>
<th>Estimated atmospheric half-life (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>[OH]$^{-1}$ = $1.5 \times 10^5$ molecules/cm$^3$</td>
<td>[OH]$^{-1}$ = $5 \times 10^5$ molecules/cm$^3$</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{17}$Cl</td>
<td>20.1</td>
<td>$9.75 \times 10^{-12}$</td>
<td>1.1</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{20}$Cl$_2$</td>
<td>33.6</td>
<td>$8.16 \times 10^{-12}$</td>
<td>1.3</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{18}$Cl$_3$</td>
<td>43.4</td>
<td>$6.57 \times 10^{-12}$</td>
<td>1.6</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{17}$Cl$_4$</td>
<td>50.7</td>
<td>$5.17 \times 10^{-12}$</td>
<td>2.1</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{17}$Cl$_5$</td>
<td>56.4</td>
<td>$5.22 \times 10^{-12}$</td>
<td>2.0</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{16}$Cl$_6$</td>
<td>61.0</td>
<td>$3.77 \times 10^{-12}$</td>
<td>2.8</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{15}$Cl$_7$</td>
<td>64.8</td>
<td>$3.68 \times 10^{-12}$</td>
<td>2.9</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{14}$Cl$_8$</td>
<td>68.0</td>
<td>$2.63 \times 10^{-12}$</td>
<td>4.1</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{13}$Cl$_9$</td>
<td>70.6</td>
<td>$1.59 \times 10^{-12}$</td>
<td>6.7</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{12}$Cl$_{10}$</td>
<td>72.9</td>
<td>$1.02 \times 10^{-12}$</td>
<td>10.5</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{27}$Cl$_1$</td>
<td>16.2</td>
<td>$13.2 \times 10^{-12}$</td>
<td>0.81</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{26}$Cl$_2$</td>
<td>28.0</td>
<td>$10.9 \times 10^{-12}$</td>
<td>0.98</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{25}$Cl$_3$</td>
<td>37.0</td>
<td>$9.35 \times 10^{-12}$</td>
<td>1.1</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{24}$Cl$_4$</td>
<td>44.1</td>
<td>$7.76 \times 10^{-12}$</td>
<td>1.4</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{23}$Cl$_5$</td>
<td>49.8</td>
<td>$7.11 \times 10^{-12}$</td>
<td>1.5</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{22}$Cl$_6$</td>
<td>54.5</td>
<td>$5.94 \times 10^{-12}$</td>
<td>1.8</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{21}$Cl$_7$</td>
<td>58.4</td>
<td>$4.96 \times 10^{-12}$</td>
<td>2.2</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{20}$Cl$_8$</td>
<td>61.7</td>
<td>$4.87 \times 10^{-12}$</td>
<td>2.2</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{19}$Cl$_9$</td>
<td>64.6</td>
<td>$4.39 \times 10^{-12}$</td>
<td>2.4</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{18}$Cl$_{10}$</td>
<td>67.1</td>
<td>$3.34 \times 10^{-12}$</td>
<td>3.2</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{17}$Cl$_{11}$</td>
<td>69.3</td>
<td>$2.30 \times 10^{-12}$</td>
<td>4.7</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{16}$Cl$_{12}$</td>
<td>71.3</td>
<td>$1.40 \times 10^{-12}$</td>
<td>7.7</td>
</tr>
<tr>
<td>C$<em>{13}$H$</em>{15}$Cl$_{13}$</td>
<td>73.0</td>
<td>$1.31 \times 10^{-12}$</td>
<td>8.2</td>
</tr>
</tbody>
</table>

*Assumes 12 hours of sunlight per day.

The major SCCP structures observed in environmental samples, such as in Great Lakes and Arctic air and biota — C$_{10}$H$_{17}$Cl$_5$, C$_{10}$H$_{16}$Cl$_6$, C$_{10}$H$_{15}$Cl$_7$, C$_{11}$H$_{18}$Cl$_6$, C$_{11}$H$_{17}$Cl$_7$, C$_{12}$H$_{20}$Cl$_6$, C$_{12}$H$_{19}$Cl$_7$ — all have estimated atmospheric half-lives greater than 2 days. Furthermore, these structures predominate in SCCP products analyzed by Tomy (1997).

Van Pul et al. (1998) modeled the atmospheric transport of SCCPs and other semivolatile organics. Taking into account wet and dry deposition processes and using a relatively long atmospheric photochemical degradation half-life of 96 hours, they predicted atmospheric half-lives for SCCPs of 23 hours over land and 27 hours over the sea. Unfortunately, the physical property data used by van Pul et al. (1998) for SCCPs are not provided, and their source is unclear. Their study predates the publication of VPs by Drouillard et al. (1998a) as well as other physical properties that apply to individual formula groups.
Persistence in water
SCCPs are not expected to degrade significantly by abiotic processes such as hydrolysis (IPCS 1996; U.K. Environment Agency 2003a,b). However, additional information submitted by the Japanese government suggest that one SCCP congener (1-chlorooctadecane; C = 12, Cl = 1) is readily biodegradable. Furthermore, Koh and Thiemann (2001) showed that SCCP mixtures underwent rapid photolysis in acetone–water under ultraviolet light (mercury arc lamp; >254 nm) with half-lives of 0.7–5.2 hours. The half-life of a 52% chlorine by weight SCCP product in pure water under the same conditions was 12.8 hours. Photoproducts included n-alkanes. The use of a 254-nm wavelength irradiation source in these studies may have produced far shorter half-lives than under natural light conditions. These results suggest that sunlight photolysis may be a significant degradation pathway for some SCCPs.

Persistence in soil and sediment
SCCP residues were found in the surficial sediments of the following remote Arctic lakes (reported in ng/g dry wt.): Yaya Lake (1.6), Hazen Lake (4.5) and Lake DV-09 (17.6) (Table 16). Concentration profiles of SCCP residues in sediments from Lake Winnipeg, Manitoba, and Fox Lake, Yukon, indicated that residues were present in the slices dated at 1947 in the sediments from both of these lakes. SCCP residues in sediments were observed from the west basin of Lake Ontario dating back to 1949. The highest concentration (800 ng/g dry wt.) was observed in the slice dated at 1971 (Figure 6).

In the absence of information on loading for any of the years at any of these locations, it is not possible to calculate a discrete half-life value from these data. However, the fact that SCCP residues were detected in sediment cores dating back to the 1940s at these locations is convincing evidence that SCCPs can persist for long periods in sediment. Residues observed in slices dated earlier can be explained by contamination as an artefact of core sampling (Muir 2000). Environment Canada (2004) used first order decay equations in a back calculation method to determine that SCCPs have a half-life in sediments longer than 1 year, which is the criterion for persistence in sediment in the Persistence and Bioaccumulation Regulations of CEPA 1999. While the back calculation method for determining half lives does not provide a discrete value for the half life of a chemical it can provide an answer as to whether a chemical’s half life is significantly greater than a specified timeframe.

The European risk assessment of SCCPs (EC 2000) concluded that slow biodegradation in the environment may occur, but assumes that SCCPs are not readily biodegradable, because it was not possible to estimate a reliable biodegradation rate with the available data.

Several government assessments and published reviews have concluded that slow biodegradation in the environment may be expected to occur, particularly in the presence of adapted micro-organisms (Government of Canada 1993a,b; Tomy et al. 1998a; EC 2000). However, no information is currently available on anaerobic biodegradation. There has been relatively little progress in development of information on degradation of SCCPs in sediments since the mid-1990s.

Madeley and Birtley (1980) used 25-day biochemical oxygen demand (BOD) tests to examine the biodegradation of a range of CPs with different carbon chain lengths and chlorine contents. Acclimatized micro-organisms showed a greater ability to degrade CPs than did organisms normally used for treating domestic sewage, and increasing chlorination inhibited biodegradation. An SCCP with 49% chlorine appeared to be rapidly and completely degraded by
acclimatized micro-organisms after 25 days. No significant oxygen uptake was observed in tests using the highly chlorinated CPs, which included two SCCPs (60% and 70% chlorine) and one MCCP (58% chlorine).

Fisk et al. (1998a) found that two $^{14}$C-labelled C$_{12}$ chloro-n-alkanes (56% and 69% chlorine) were degraded at 12°C in aerobic sediments used for a study of the bioavailability of SCCPs to oligochaetes. The biodegradation in sediments was estimated by comparing the amount of toluene-extractable and toluene-non-extractable $^{14}$C after 18 and 32 days of incubation. It was hypothesized that the toluene-non-extractable $^{14}$C fraction represented a polar degradation product of the CPs, which was tightly bound to the sediments and therefore non-extractable using toluene. Half-lives in sediment were 12 ± 3.6 days and 30 ± 2.6 days for the 56% and 69% chlorine products, respectively. No degradation product identification was performed. This study, therefore, would not provide any indication of the mineralization or ultimate degradation half-life of SCCPs.

Allpress and Gowland (1999) isolated a strain of Rhodococcus bacteria that could utilize SCCP with 49.5% chlorine as the sole carbon source with the release of chloride ion. They inoculated 100 mL of growth medium with the bacteria and solutions of the CP formulations at 1% v/v. The test lasted 71 days. The greatest release of chloride ion (49%) occurred with the SCCP with 49.5% chlorine, compared with MCCP and LCCP formulations containing 42–47.5% chlorine by weight. The isolate was unable to utilize a more highly chlorinated SCCP (63.5% chlorine) or MCCP (58.5% chlorine) formulation.

Omori et al. (1987) studied the CP dechlorination potential of a series of soil bacterial strains acting on C$_{12}$H$_{18}$Cl$_8$ with 63% chlorine. Although they could not isolate a bacterial strain that could use CP as a sole carbon source, they did find that different strains pre-treated with n-hexadecane had different dechlorination abilities. A mixed culture (four bacterial strains) released 21% of the chlorine after 48 hours. The bacterial strain HK-3, acting alone, released 35% of the chlorine after 48 hours. Omori et al. (1987) suggested that the mechanism for the aerobic degradation was a $\beta$-oxidation enzyme system, which would first oxidize the terminal methyl group to produce chlorinated fatty acids and then break down the acids to 2- or 3- chlorinated fatty acids.

Concentration profiles of SCCPs in sediments from Lake Winnipeg, Fox Lake in the Yukon, the west basin of Lake Ontario (see Figure 6) and Lake DV09 (see Figure 8) indicate that SCCP residues were present in the 1940s (Muir et al. 1999; Tomy et al. 1999). Some transformation of SCCP chain length and formula groups may have occurred over time (i.e., with depth) in these cores, judging by the appearance of greater proportions of shorter chain lengths and lower chlorinated alkanes at shallower depths (Tomy et al. 1999). Tomy et al. (1999) hypothesized that this may be a result of rapid aerobic degradation occurring at these depths. However, these shifts may also be due to changing product formulations at upstream manufacturing and waste treatment sources. In the absence of information on loading for any of the years at any of these locations, it is not possible to calculate specific half-lives from these data. However, the fact that SCCP residues were detected in sediment cores at these dates is convincing evidence that SCCPs can persist for more than 50 years in subsurface anaerobic sediments. Nicholls et al. (2001) were unable to detect SCCPs (<0.1 µg/g) in farm soils in the United Kingdom on which several applications of sewage sludge had been applied. However, the study did not specifically follow the fate of SCCPs over time following sludge application.

2.2.2. Bioaccumulation
2.2.2.1 Modeled Log K\textsubscript{OW} and Bioaccumulation Factors

It was calculated that log K\textsubscript{OW} ranged between 4.8 and 7.6 for all possible SCCP congeners using the equation developed by Sijm and Sinnige (1995). QSAR based models (e.g. EPI KowWin) were used to compare to this empirical model but the predictions (ranging 5.4 – 21.6) are unreliable due to the results exceeding the limits of the training set. However the large amounts of high Kow readings suggest a large potential for bioaccumulation. The few experimental data available also fit with the modelled range. Fisk et al. (1998b) determined the octanol-water partition coefficients of two \textsuperscript{14}C-labelled short-chain chlorinated paraffins of single carbon chain length (C\textsubscript{12}). The two compounds used were C\textsubscript{12}H\textsubscript{20.1}Cl\textsubscript{5.9}, 55.9% wt. Cl and C\textsubscript{12}H\textsubscript{16.2}Cl\textsubscript{9.8}, 68.5% wt. Cl. The mean log Kow values determined by a HPLC method were reported to be 6.2 for the 55.9% wt. Cl substance (range of log Kow was 5.0 to 7.1 for the main components of this substance) and 6.6 for the 68.5% wt. Cl substance (range of log Kow was 5.0 to 7.4). Using the empirical Kow data in the Gobas BAF model with an assumption of no metabolism finds that all possible SCCP congeners have a BAF > 5000. As such it is likely they are bioaccumulative.

2.2.2.1 Bioconcentration

Reported bioconcentration factors (BCFs) calculated from laboratory studies for SCCPs have been reviewed in Government of Canada (1993b) and vary dramatically among different species. Relatively low BCF values have been determined in freshwater and marine algae (<1–7.6). BCF values of up to 7816 wet wt. have been measured in rainbow trout (\textit{Oncorhynchus mykiss}) (Madeley and Maddock 1983a,b) and 5785–138 000 wet wt. in the common mussel (\textit{Mytilus edulis}) (Madeley et al. 1983b, Madeley and Thompson 1983d, Renberg et al. 1986). Fisk et al. (1999) measured BCFs in Japanese medaka (\textit{Oryzias latipes}) embryos exposed to a range of concentrations of \textsuperscript{14}C-labelled C\textsubscript{10} and C\textsubscript{12} SCCPs for 20 days. Highest BCFs (1400–2700) were found for C\textsubscript{10}H\textsubscript{15}Cl\textsubscript{7}, compared with C\textsubscript{12}H\textsubscript{20}Cl\textsubscript{7} (740–1700). These BCF values were similar to previous measurements for fish, although it should be noted that the test organisms in this case were embryos and therefore accumulating SCCPs solely by respiration. No other recent laboratory BCF studies have been identified.

Information submitted by Japan on the measured BCF test results for C=11, Cl = 7-10 suggested that these chemicals are bioaccumulative. In addition the BCF of chlorinated paraffins (C=13-17, Cl = 6-12) were observed to be far low. The test results find that BCFs for some C=13 congeners are lower than BCFs for some congeners of C=11.

Table 6  BCF test results for some SCCP congeners (C=11, Cl=7-10)

<table>
<thead>
<tr>
<th>Cl</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12 days</td>
<td>24 days</td>
<td>38 days</td>
<td>47 days</td>
<td>60 days</td>
<td>12 days</td>
<td>24 days</td>
<td>38 days</td>
</tr>
<tr>
<td>Cl=7</td>
<td>4600</td>
<td>4900</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
</tr>
<tr>
<td>Cl=8</td>
<td>2900</td>
<td>2499</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
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<tr>
<td>Cl=9</td>
<td>4400</td>
<td>4400</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
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<tr>
<td>Cl=10</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
</tr>
</tbody>
</table>

Table 6  BCF test results for some SCCP congeners (C=11, Cl=7-10)

<table>
<thead>
<tr>
<th>Cl</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
<th>1µg/L</th>
<th>0.1µg/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12 days</td>
<td>24 days</td>
<td>38 days</td>
<td>47 days</td>
<td>60 days</td>
<td>12 days</td>
<td>24 days</td>
<td>38 days</td>
</tr>
<tr>
<td>Cl=7</td>
<td>4600</td>
<td>4900</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
</tr>
<tr>
<td>Cl=8</td>
<td>2900</td>
<td>2499</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
</tr>
<tr>
<td>Cl=9</td>
<td>4400</td>
<td>4400</td>
<td>2900</td>
<td>2499</td>
<td>4900</td>
<td>5300</td>
<td>3200</td>
<td>2500</td>
</tr>
<tr>
<td>Cl=10</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
<td>4500</td>
</tr>
</tbody>
</table>
Table 7  BCF test results for some SCCP congeners (C=13-17; Cl = 6-12)

<table>
<thead>
<tr>
<th></th>
<th>2 week</th>
<th>4 week</th>
<th>6 week</th>
<th>8 week</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.36ppm</td>
<td>0.9</td>
<td>0.9</td>
<td>1.1</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>0.9</td>
<td>1.1</td>
<td>0.7</td>
<td>1.0</td>
</tr>
<tr>
<td>0.036ppm</td>
<td>3.2</td>
<td>4.5</td>
<td>3.0</td>
<td>3.9</td>
</tr>
<tr>
<td></td>
<td>3.6</td>
<td>3.3</td>
<td>3.6</td>
<td>3.4</td>
</tr>
</tbody>
</table>

2.2.2.2 Laboratory studies of bioaccumulation, biomagnification and biotransformation

SCCPs are readily accumulated from food by fish in laboratory experiments. Dietary accumulation is influenced by carbon chain length and chlorine content (Fisk et al. 1996, 1998b, 2000). SCCPs with greater than 60% chlorine by weight were found to have equilibrium biomagnification factors (BMFs) greater than 1. Compounds with BMFs greater than 1 will theoretically be biomagnified in food webs. Equilibrium BMFs were predicted from the equation $\text{BMF} = \alpha \cdot \frac{F}{k_d}$, where $\alpha$, the assimilation efficiency, was assumed to be 0.5, $F$ is the feeding rate (lipid corrected) and $k_d$ is the depuration rate (Fisk et al. 1998b). Half-lives in fish ranged from 7 to about 53 days in laboratory studies with juvenile rainbow trout, which implies a potential to biomagnify in aquatic food chains (Fisk et al. 1998b). When compared with the half-lives of 40–60 days in rainbow trout for non-metabolizable organochlorine compounds of similar $\log K_{ow}$ values of 6 or greater (i.e., PCBs, mirex, hexachlorobenzene), it appeared that SCCPs with lower chlorination were being metabolized by the fish, but biotransformation products were not measured. Metabolism was inferred from lower amounts of toluene-extractable $^{14}$C residues after 40 days of depuration for some SCCPs. However, the half-lives for two chlorododecanes, C$_{12}$H$_{18}$Cl$_4$ and C$_{12}$H$_{20}$Cl$_6$, were similar to those of recalcitrant organochlorines. These compounds had long half-lives (Table 8) and essentially no biotransformation (biotransformation half-life >1000 days) based on this method of calculation. The lack of biotransformation was probably related to the particular chlorine substitution pattern on the n-alkanes; however, only the molecular formulas of these SCCPs were known.

Table 8: Half-lives and estimated biotransformation rates of SCCPs in rainbow trout (from Fisk et al. 2000)

<table>
<thead>
<tr>
<th>SCCP</th>
<th>Depuration half-life (days)$^1$</th>
<th>Biotransformation half-life (days)$^2$</th>
<th>% depuration associated with biotransformation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>C$<em>{10}$H$</em>{16}$Cl$_4$</td>
<td>8.3 ± 1.5</td>
<td>10</td>
<td>81</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{17}$Cl$_5$</td>
<td>7.8 ± 1.3</td>
<td>9</td>
<td>85</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{17}$Cl$_5$</td>
<td>7.1 ± 1.2</td>
<td>8</td>
<td>87</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{16}$Cl$_6$</td>
<td>10.2 ± 0.9</td>
<td>12</td>
<td>84</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{16}$Cl$_6$</td>
<td>10.0 ± 0.9</td>
<td>12</td>
<td>84</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$ H$</em>{16}$Cl$_6$</td>
<td>20.4 ± 5.4</td>
<td>30</td>
<td>68</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{15.5}$Cl$_{6.7}$ (low)</td>
<td>43.3 ± 5.7</td>
<td>124</td>
<td>35</td>
<td>Fisk et al. (2000)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{15.5}$Cl$_{6.7}$ (high)</td>
<td>25.7 ± 1.7</td>
<td>41</td>
<td>63</td>
<td>Fisk et al. (2000)</td>
</tr>
<tr>
<td>C$<em>{10}$H$</em>{15}$Cl$_7$</td>
<td>14.7 ± 1.6</td>
<td>19</td>
<td>79</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>SCCP</td>
<td>Depuration half-life (days)</td>
<td>Biotransformation half-life (days)</td>
<td>% depuration associated with biotransformation</td>
<td>Reference</td>
</tr>
<tr>
<td>-----------</td>
<td>----------------------------</td>
<td>-----------------------------------</td>
<td>------------------------------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>C_{10}H_{15}Cl_{7}</td>
<td>8.6 ± 0.5</td>
<td>10</td>
<td>88</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{10}H_{14}Cl_{8}</td>
<td>30.1 ± 5.2</td>
<td>50</td>
<td>61</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{10}H_{14}Cl_{8}</td>
<td>13.9 ± 1.1</td>
<td>17</td>
<td>82</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{11}H_{20}Cl_{4}</td>
<td>10.8 ± 1.0</td>
<td>14</td>
<td>80</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{11}H_{19}Cl_{5}</td>
<td>9.0 ± 1.4</td>
<td>11</td>
<td>86</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{11}H_{18}Cl_{6}</td>
<td>16.9 ± 2.5</td>
<td>22</td>
<td>76</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{11}H_{16}Cl_{8}</td>
<td>36.5 ± 9.6</td>
<td>69</td>
<td>53</td>
<td>Fisk et al. (1998b)</td>
</tr>
<tr>
<td>C_{12}H_{19.3}Cl_{6.7} (low)</td>
<td>38.5 ± 4.3</td>
<td>81</td>
<td>48</td>
<td>Fisk et al. (1996)</td>
</tr>
<tr>
<td>C_{12}H_{19.3}Cl_{6.7} (high)</td>
<td>77.0 ± 8.6</td>
<td>&gt;1000</td>
<td>0</td>
<td>Fisk et al. (1996)</td>
</tr>
<tr>
<td>C_{12}H_{16.2}Cl_{9.8} (low)</td>
<td>86.6 ± 10.8</td>
<td>&gt;1000</td>
<td>0</td>
<td>Fisk et al. (1996)</td>
</tr>
<tr>
<td>C_{12}H_{16.2}Cl_{9.8} (high)</td>
<td>77.0 ± 8.6</td>
<td>&gt;1000</td>
<td>8</td>
<td>Fisk et al. (1996)</td>
</tr>
</tbody>
</table>

1 Measured depuration rates (± standard error) converted to half-lives using the equation \( t_{1/2} \) (days) = 0.693/rate (day\(^{-1}\)).

2 Biotransformation rate = measured depuration rate – minimum depuration rate derived from a polynomial relationship of half-life with log \( K_{OW} \) developed by Fisk et al. (2000). Biotranformation rate converted to a half-life using the equation \( t_{1/2} \) (days) = 0.693/rate (day\(^{-1}\)).

Table 8 compares the measured depuration half-lives and estimated biotransformation half-lives of \( C_{10-12} \) SCCPs. The similar depuration and biotransformation half-lives for some SCCPs, particularly the decanes, suggest that the depuration is primarily due to biotransformation.

Fisk et al. (2000) showed that BMFs for 35 chlorinated n-alkanes (combined data for SCCPs and MCCPs; some were single homologues and others were homologue mixtures of single carbon chains) were significantly related to the number of carbon plus chlorine atoms per compound and to log \( K_{OW} \) (Figure 2). Biotransformation half-lives in trout ranged from 8 to >1000 days (Table 8).
Bengtsson and Baumann-Ofstad (1982) studied the uptake and elimination of two commercial SCCPs — a 49% chlorine product and a 71% chlorine product — in bleak (*Alburnus alburnus*). The SCCPs were administered in food at concentrations ranging from 590 to 5800 µg/g food. There was a 91-day uptake period followed by a 316-day depuration period. Although the uptake efficiency of the 71% chlorine product was low (6%, or half that of the 49% chlorine product), a remarkably high retention was observed. This formulation remained in the fish tissues at a steady level until the experiment was terminated after the 316-day elimination period.

Fisk et al. (1998a) studied the bioaccumulation and depuration of sediment-sorbed $^{14}$C-labelled C$_{12}$H$_{20}$Cl$_{6}$ (56% chlorine by weight) and C$_{12}$H$_{16}$Cl$_{10}$ (69% chlorine by weight) by oligochaetes (*Lumbriculus variegatus*). The experiment consisted of a 14- or 21-day uptake period followed by a 42-day depuration period. Organic carbon normalized biota–sediment accumulation factors (BSAF) calculated from the rates of uptake and depuration ranged from 1.9 for C$_{12}$H$_{16}$Cl$_{10}$ to 6.8 for C$_{12}$H$_{20}$Cl$_{6}$. Half-lives of the two SCCPs were similar, but uptake of the more highly chlorinated dodecane was significantly slower than that of the less chlorinated dodecane. There was evidence for biotransformation of the SCCPs by *Lumbriculus*; however, the degradation products may also have been formed in the sediments and accumulated by the oligochaetes.

### 2.2.2.3 Field studies of bioaccumulation

Bioaccumulation factors (BAFs) for SCCP chain length groups in western Lake Ontario lake trout (*Salvelinus namaycush*) were calculated based on concentrations in whole fish (wet weight or lipid weight) and dissolved water concentrations (Muir et al. 2001). BAFs ranged from 88 000 to 137 600 in lake trout on a lipid weight basis, or from 16 440 to 25 650 on a wet weight basis (Table 9). Chlorinated dodecanes (C$_{12}$) were the most prominent SCCPs in lake water and fish. The highest BAFs were for the chlorinated...
tridecanes (C\textsubscript{13}) because of their low water concentrations. BMFs for the SCCPs based on an alewife (Alosa pseudoharengus)/smelt (Osmerus mordax) diet ranged from 0.33 to 0.94 and were highest for the tridecanes. These BMF values suggest that SCCPs, especially the chlorinated decanes and dodecanes, are not biomagnifying in the pelagic food web of Lake Ontario. By contrast, PCBs have been shown to biomagnify in the same food web. Relatively high concentrations of SCCPs in sculpin (Cottus cognatus) and diporeia (Diporeia sp.) imply that sediments are an important source of SCCPs for bottom feeders (Muir et al. 2002).

Table 9: BAFs and BMFs for SCCPs in lake trout and forage fish from western Lake Ontario (from data of Muir et al. 2002)

<table>
<thead>
<tr>
<th>HOMOLOGUE</th>
<th>Concentration in water (ng/L)\textsuperscript{1}</th>
<th>BAF\textsubscript{ww}\textsuperscript{2} in lake trout</th>
<th>BAF\textsubscript{lw}\textsuperscript{3} in lake trout</th>
<th>BMF, lake trout to forage fish\textsuperscript{4}</th>
</tr>
</thead>
<tbody>
<tr>
<td>C\textsubscript{10}</td>
<td>0.12</td>
<td>20 690</td>
<td>110 990</td>
<td>0.33</td>
</tr>
<tr>
<td>C\textsubscript{11}</td>
<td>0.36</td>
<td>25 650</td>
<td>137 590</td>
<td>0.51</td>
</tr>
<tr>
<td>C\textsubscript{12}</td>
<td>0.73</td>
<td>16 440</td>
<td>88 180</td>
<td>0.74</td>
</tr>
<tr>
<td>C\textsubscript{13}</td>
<td>0.07</td>
<td>19 950</td>
<td>107 000</td>
<td>0.94</td>
</tr>
<tr>
<td>(\Sigma\text{C}_{10}^{13})</td>
<td>1.27</td>
<td>17 210</td>
<td>105 160</td>
<td>0.58</td>
</tr>
</tbody>
</table>

\textsuperscript{1} Water concentrations from large volume (90 L) of water samples collected in October 2000.

\textsuperscript{2} Bioaccumulation factor (wet weight).

\textsuperscript{3} Bioaccumulation factor (lipid weight). Average lipid content of (whole) lake trout samples = 16% in samples from July 2001.

\textsuperscript{4} Assumes 50% alewife and 50% rainbow smelt diet. BMF = ng/g lw in whole trout ÷ ng/g lw in forage fish.

SCCPs have been found in all components of the food chain in Lake Ontario, including 2630 ng/g wet wt. in carp (refer to Table 17). SCCPs have also been found in high concentrations in marine mammals, including beluga (Delphinapterus leucas), ringed seal (Phoca hispida) and walrus (Odobenus rosmarus) from the Arctic (95–626 ng/g wet wt.) (see Table 18).

Jansson et al. (1993) reported concentrations of SCCPs in rabbit, moose, reindeer and osprey from various regions in Sweden to be 2.9, 4.4, 0.14 and 0.53 µg/g lipid weight (lipid wt.), respectively. Thus, some herbivores had higher concentrations than a fish-eating bird (osprey).

Biomagnification of MCCPs and SCCPs in the Lake Ontario food web was examined. MCCPs and C\textsubscript{14–17} chain length groups had very low BMFs between the three forage fish species and lake trout compared with the SCCPs (Table 10).

Table 10: BMFs\textsuperscript{1} for MCCPs and SCCPs in the Lake Ontario food web (from Muir et al. 2003)

<table>
<thead>
<tr>
<th>Group</th>
<th>Alewife–lake trout</th>
<th>Smelt–lake trout</th>
<th>Sculpin–lake trout</th>
<th>Diporeia–sculpin</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCCPs</td>
<td>0.91</td>
<td>0.43</td>
<td>0.27</td>
<td>1.4</td>
</tr>
<tr>
<td>C\textsubscript{10}</td>
<td>0.43</td>
<td>0.27</td>
<td>0.17</td>
<td>1.3</td>
</tr>
<tr>
<td>C\textsubscript{11}</td>
<td>0.94</td>
<td>0.35</td>
<td>0.22</td>
<td>1.6</td>
</tr>
<tr>
<td>C\textsubscript{12}</td>
<td>1.1</td>
<td>0.56</td>
<td>0.37</td>
<td>1.2</td>
</tr>
<tr>
<td>C\textsubscript{13}</td>
<td>1.5</td>
<td>0.68</td>
<td>0.35</td>
<td>1.1</td>
</tr>
<tr>
<td>MCCPs</td>
<td>0.09</td>
<td>0.05</td>
<td>0.05</td>
<td>5.2</td>
</tr>
<tr>
<td>C\textsubscript{14}</td>
<td>0.27</td>
<td>0.16</td>
<td>0.10</td>
<td>2.7</td>
</tr>
<tr>
<td>C\textsubscript{15}</td>
<td>0.04</td>
<td>0.02</td>
<td>0.03</td>
<td>9.56</td>
</tr>
<tr>
<td>C\textsubscript{16}</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>14.5</td>
</tr>
<tr>
<td>C\textsubscript{17}</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>11.5</td>
</tr>
</tbody>
</table>

\textsuperscript{1} BMF = lipid weight concentration in predator / lipid weight concentration in prey.
The SCCPs, by comparison, had much higher BMFs, especially for C\textsubscript{12} and C\textsubscript{13} SCCPs in the same food web (Table 10). With SCCPs, the alewife to lake trout BMF exceeded 1, as did the BMF for diporeia to sculpin.

Bioaccumulation Summary

BAFs for SCCP homologue groups in western Lake Ontario trout range from 16 440 to 25 650 wet wt. (Table 9). Very high bioconcentration factors (BCFs) have been measured for SCCPs in rainbow trout (Oncorhynchus mykiss) (up to 7816 wet wt.) (Madeley and Maddock 1983a,b) and in mussels (Mytilus edulis) (5785−138 000 wet wt.) (Madeley and Thompson 1983d, Madeley et al. 1983b). Estimated and measured log K\textsubscript{OW} values for SCCPs range from 4.39 to 8.69. Based on measured Log K\textsubscript{OW} values in the range of 4.48 to 8.69 the Gobas model predicts a log BAF ranging from 3.47 to 6.2 with the large majority of the predictions having a BAF > 5000.

High SCCP concentrations in marine mammals (ringed seal, beluga whales and walrus) and carp (Tables 17 and 18) show that SCCPs are likely to bioaccumulate in aquatic biota. Bengtsson and Baumann-Ofstad (1982) found evidence of very high retention of a highly chlorinated (71% chlorine by weight) SCCP formulation in bleak during a 316-day elimination period of an uptake/elimination study.

Tomy (1997) found that SCCPs (around 60–70% chlorine by weight) were present at a concentration of 11–17 µg/kg lipid (mean concentration 13 µg/kg lipid) in human breast milk from Inuit women living on the Hudson Strait in northern Quebec, Canada. Similarly, SCCPs were found at concentrations of 4.5–110 µg/kg lipid in a study in the United Kingdom (Thomas and Jones 2002). These findings are indicative of bioaccumulation through the food chain, especially in northern Quebec, since food would be the major or only source of environmental exposure for the Inuit.

While biomagnifications factors (BMF) are not a criterion considered in the regulations for bioaccumulation, BMFs are important supplemental information. If a substance has a BMF greater than 1, it is likely to have high BCF/BAF values. SCCPs have been shown to have BMF values approaching 1 (ranging from 0.33-0.94) between lake trout and forage fish in western Lake Ontario (Environment Canada, 2004) (Table 9).

It is therefore concluded that short-chain chlorinated paraffins are bioaccumulative substances according to the criteria stipulated in the Persistence and Bioaccumulation Regulations of CEPA 1999 (Government of Canada 2000).

2.2.3. Potential for Long Range Transport

OECD (1999) performed fugacity modeling of SCCPs using a Level II fugacity model. Among their input data, they used a half-life in air of 7.2 days and a VP of 0.0213 Pa (at 40°C). In the 100% release to air and 100% release to water scenarios, 0.11% and 0.05%, respectively, of the SCCPs remained in air, and 0.02% and 1.16%, respectively, remained in water at steady state, indicating slight mobility of SCCPs in the environment.

Van de Meent et al. (2000) ran a global-scale multimedia model (similar to GloboPOPs) assuming no degradation except in air, water and soil. This method shows that SCCPs are multimedia chemicals found in all three compartments, except for some highly chlorinated compounds such as C\textsubscript{13}H\textsubscript{16}Cl\textsubscript{12}.

Arctic accumulation potential

Arctic contamination potential (ACP) is the amount of chemical in Arctic surface media divided by the amount of chemical in the global environment estimated from the GloboPOPs model (Wania 2003; Wania and Mackay 2000). The ACP results were generated from the GloboPOPs model for a
hypothetical series of chemicals of varying $K_{OA}$ and $K_{AW}$ assuming 10 years of emission into air, no atmospheric degradation and emission distribution by population density (refer to Canadian Annex E submission for diagram and more details). High ACP values are >3 while low ACP values are <1.0. Results suggest that SCCPs have moderate ACP when released to air, similar to tetra- to heptachlorobiphenyls. Only chemicals such as chlorobenzenes and mono/di/trichlorobiphenyls with log $K_{OA}$ of 3–7 and log $K_{AW}$ of –1 to –2 have high ACP. Similar assessment using emissions to water or soil (in which the ACP values in the two-dimensional [$K_{OA}$ vs. $K_{AW}$] ACP “space” are smaller than for air emissions) also shows that SCCPs have low ACP when emitted to water.

This data is in agreement with Reth et al. (2005) who found that the SCCP congener pattern measured in the Baltic Sea is similar to that of commercial SCCP mixtures and low molecular weight (C$_{10}$) congeners increased in the mixtures as samples were taken further away in the North Sea. In addition, lower molecular weight congener mixtures were reported by Tomy et al. (2000) to be present in marine mammals, consistent with long rage transport of SCCPs.

Annex E information submitted by Switzerland for this risk profile outlines a recent study by Wegmann et al. (2007) which used OECD Pov and LRTP screening tools to examine the long range transport of several POPs candidate substances including SCCPs. The authors included Monte Carlo calculations to demonstrate the influence of uncertain chemical properties. Although there are considerable uncertainties in the chemical properties, the results indicated that SCCPs have Pov and LRTP properties similar to those of several known POPs.

The EU (2005) dossier prepared for the UNECE LRTAP Convention found that the available data from remote areas (e.g. see section 2.3) clearly show the contamination of the environment and biota by SCCPs.

### 2.3 Exposure

**Atmospheric concentrations**

SCCPs were detected in four individual samples of air collected at Alert at the northern tip of Ellesmere Island in the high Arctic (Table 11). Concentrations ranged from non-detectable to 8.5 pg/m$^3$ in gas-phase samples. Blank samples from the air sampling program at Alert also contained low levels of SCCPs, but at concentrations lower than those in air samples. Heptachloroundecane was the major component of the SCCPs, and undecanes (C$_{11}$) were the predominant alkane group. Tomy (1997) had previously analyzed air samples collected at Egbert, Ontario, in 1990 for SCCPs and found similar profiles, with chlorodecanes (C$_{10}$) and chloroundecanes (C$_{11}$) predominating. The higher SCCP concentrations at Egbert (Table 11) are consistent with its location near population centers of southern Ontario.

<table>
<thead>
<tr>
<th>Location</th>
<th>Date</th>
<th>Total SCCP concentration (pg/m$^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert, Nunavut</td>
<td>September 14, 1992</td>
<td>5.7</td>
</tr>
<tr>
<td>Alert, Nunavut</td>
<td>September 21, 1992</td>
<td>non-detectable</td>
</tr>
<tr>
<td>Alert, Nunavut</td>
<td>September 28, 1992</td>
<td>2.1</td>
</tr>
<tr>
<td>Alert, Nunavut</td>
<td>December 28, 1992</td>
<td>8.5</td>
</tr>
<tr>
<td>Alert, Nunavut</td>
<td>August (weeks 29–32) 1994</td>
<td>7.25</td>
</tr>
<tr>
<td>Alert, Nunavut</td>
<td>September (weeks 33–36) 1994</td>
<td>6.14</td>
</tr>
</tbody>
</table>
Comparison of SCCPs in the atmosphere in the United Kingdom, Canada and Norway

In the United Kingdom, Peters et al. (2000) reported a mean SCCP concentration of 99 pg/m$^3$ in air collected from a semi rural site in Lancaster. SCCP concentrations in air were ranked such that Egbert > Lancaster > Alert and most likely reflect the proximity of Egbert and Lancaster to local source areas. A recent paper by Barber et al. (2005) found that concentrations in the U.K. atmosphere ranged between <185 to 3430 pg/m$^3$ (mean of 1130 pg/m$^3$) and were higher than 1997 levels at the same site. Barber et al. (2005) also calculated an average concentration of 600 pg/m$^3$ of SCCPs for the UK atmosphere. The profiles of SCCPs at Alert resemble those measured in Lancaster but are quite different from those at Egbert and over western Lake Ontario. This is illustrated in Figures 3 and 4. In Figure 3, the individual “formula groups” for each carbon chain length within the SCCP mixture are plotted for Egbert and Lancaster (Tomy et al. 1998a; Peters et al. 2000). From this figure, it is clear that Egbert air samples have higher proportions of penta-, hexa- and heptachlorodecanes and undecanes compared with Lancaster, whereas Lancaster air has higher proportions of hexa-, hepta- and octadecanones and tridecanes than Egbert air. Both locations are north of major urban areas (Lancaster is about 70 km north of the Manchester–Liverpool area, and Egbert is about 50 km north of Toronto), so the reasons for the differences in homologue patterns between Lancaster and Egbert are not clear.
In Figure 4, the proportions of SCCP chain length groups are compared at four locations. It can be seen that the profile of SCCP components in Egbert air in summer 1990 resembles that of air over western Lake Ontario collected in July 1999 (Muir et al. 2001), in that both have high proportions of chlorodecanes and chloroundecanes. The profile in Alert air resembles that at Lancaster but differs from that of the southern Ontario sites by having higher proportions of chlorododecanes. These differences appear to be consistent over time. Analysis of air samples collected in October 2000 over western Lake Ontario showed the same profile as in July (i.e., higher chlorodecanes and chloroundecanes), despite much lower air temperatures (D.C.G. Muir, unpublished data). The reasons for these differences are not clear. The Alert site is very remote from urban areas and receives air predominantly from sources in Europe and Asia because of its unique geographic location. However, this air flow from Europe/Asia is more pronounced in winter than in summer months (Halsall et al. 1998). The three other sites are outside of, but within 100 km of, urban areas.

Borgen et al. (2000) measured SCCPs in Arctic air samples taken at Mt. Zeppelin, Svalbard, Norway, during the period March to May 1999, using HR-ECNIMS. Concentrations ranging from 9.0 to 57 pg/m$^3$ were found, which are higher than those found at Alert. Borgen et al. (2002) found much higher SCCP concentrations in air at Bear Island, a small isolated island between Svalbard and mainland Norway. Total SCCP concentrations (air + filter) ranged from 1800 to 10 600 pg/m$^3$. These high air concentrations are unusual and may be related to the transport of persistent organic
pollutants to terrestrial and freshwater environments on the island via seabird guano (Borgen et al. 2002).

SFT (2002) measured SCCP concentrations in 3 moss samples from Norway (Valvil, Molde, and Narbuvoll). Samples were taken in forest areas at a minimum distance of 300m from roads and buildings and 10 km from towns. Concentrations of 3 – 100 µg/kg wet weight were measured, suggesting the presence of SCCPs in the atmosphere.

**Wastewater treatment effluents**

SCCPs were detected in all eight sewage treatment plant final effluents from southern Ontario (Canada) at ng/L concentrations. The highest concentrations were found in samples from treatment plants in industrialized areas, including Hamilton, St. Catharine’s and Galt, compared with lower concentrations in samples from treatment plants in non-industrial towns, such as Niagara-on-the-Lake and Niagara Falls (Table 12).

<table>
<thead>
<tr>
<th>Sewage treatment plant</th>
<th>Concentration (ng/L)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woodward Avenue, Hamilton, Ontario</td>
<td>128 155 153 11.5</td>
<td>448</td>
</tr>
<tr>
<td>Halton Skyway, Burlington, Ontario</td>
<td>38 19 12 &lt;1</td>
<td>69</td>
</tr>
<tr>
<td>Stanford, Niagara Falls, Ontario</td>
<td>11 34 36 1</td>
<td>82</td>
</tr>
<tr>
<td>Port Dalhousie, St. Catharines, Ontario</td>
<td>19 39 47 5</td>
<td>110</td>
</tr>
<tr>
<td>Port Weller, St. Catharines, Ontario</td>
<td>22 27 28 4</td>
<td>81</td>
</tr>
<tr>
<td>Niagara-on-the-Lake, Ontario</td>
<td>13 18 27 1</td>
<td>59</td>
</tr>
<tr>
<td>Galt, Ontario</td>
<td>82 85 86 11</td>
<td>264</td>
</tr>
<tr>
<td>Guelph, Ontario</td>
<td>23 32 34 4</td>
<td>93</td>
</tr>
</tbody>
</table>

Table 12: SCCP concentrations\(^1\) in final effluent of sewage treatment plants in southern Ontario based on samples collected in 1996 (from Muir et al. 2001)

Reiger and Ballschmiter (1995) reported C\(_{10–13}\), 62% chlorine SCCP concentrations of 80 ± 12 ng/L in water upstream and 73 ± 10 ng/L in water downstream of a sewage treatment plant in Germany. The concentration of SCCPs in the effluents was 115 ng/L. In the United States, Murray et al. (1988) reported C\(_{10–13}\), 60% chlorine SCCP concentrations of <150–3300 ng/L in water from an impoundment drainage ditch that received effluent from a CPs production plant in Dover, Ohio.

**Sewage sludge and soils**

Nicholls et al. (2001) found total CP (SCCP + MCCP) concentrations in digested sewage sludge ranging from 1.8 to 93.1 µg/g dry wt. Nicholls et al. (2001) did not detect CPs in farm soils on which sewage sludge had been applied (<0.1 µg/g); however, SCCPs/MCCPs were detected in earthworms (<0.1–1.7 µg/g wet wt.) from four of nine farms receiving urban sewage sludge.

Stevens et al. (2002) found SCCP concentrations ranging from 6.9 to 200 µg/g dry wt. in sewage sludge from 14 WWTPs in the United Kingdom. They found that SCCP concentrations were not related to the population equivalent (wastewater volume/population) of WWTPs, whereas other contaminants, such as PCBs, were strongly correlated. However, highest concentrations of SCCPs were in sludge from industrial catchments. A rural catchment with zero industrial effluent had significant levels (590 µg/g) of total SCCPs/MCCPs in sludge (Stevens et al. 2002).
In comparison to river and lake sediments, concentrations of SCCPs/MCCPs in sewage sludge are much higher, especially from WWTPs serving industrial areas (Table 13). The fate of CPs in biosolids that are then applied to farmland, a common practice in Europe and a growing practice in North America, has not been thoroughly investigated. The work of Stevens et al. (2002) and Nicholls et al. (2001) points to agricultural soils as potentially a major reservoir of CPs (SCCPs and MCCPs) due to sewage sludge application. This source needs further investigation to determine if it is short term due to rapid biodegradation or represents a long-term reservoir for further environmental distribution.

Table 13: Concentrations of MCCPs in sewage sludges

<table>
<thead>
<tr>
<th>CP</th>
<th>Location</th>
<th>Country</th>
<th>Concentration (µg/g dry weight)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>C_{14-18}, 52% Cl</td>
<td>Zürich, urban area</td>
<td>Switzerland</td>
<td>30</td>
<td>Schmid and Müller (1985)</td>
</tr>
<tr>
<td>C_{14-17}, 52% Cl</td>
<td>Manufacturing site</td>
<td>Ohio, U.S.A.</td>
<td>0.76–50</td>
<td>Murray et al. (1988)</td>
</tr>
<tr>
<td>SCCP/MCCP</td>
<td>Urban and rural</td>
<td>U.K.</td>
<td>1.8–93.1</td>
<td>Nicholls et al. (2001)</td>
</tr>
</tbody>
</table>

Surface waters

Large-volume water samples (100 L) collected in mid-Lake Ontario in July 1999 and October 2000 showed low levels of SCCPs (Table 14; Figure 5). In 1999, the highest dissolved SCCP concentration (1.8 ng/L) was observed in the western basin and probably resulted from sewage treatment plant sources from large urbanized areas, such as Toronto and Hamilton. A similar gradient was observed in October 2000; however, concentrations were lower. Notwithstanding the high dilution factor that would be involved in Lake Ontario, the fact that these concentrations were observed is an indication that considerable inputs of SCCPs are occurring.

Table 14: Concentrations of SCCPs in filtered surface waters from western Lake Ontario (from Muir et al. 2001)

<table>
<thead>
<tr>
<th>Carbon chain group</th>
<th>Concentrations (pg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>July 1999</td>
</tr>
<tr>
<td>C_{10}</td>
<td>168</td>
</tr>
<tr>
<td>C_{11}</td>
<td>490</td>
</tr>
<tr>
<td>C_{12}</td>
<td>1000</td>
</tr>
<tr>
<td>C_{13}</td>
<td>94</td>
</tr>
<tr>
<td>ΣSCCP</td>
<td>1750</td>
</tr>
<tr>
<td>ΣPCBs</td>
<td>620</td>
</tr>
</tbody>
</table>

1 Average of duplicate samples of water at site 1007 in the west basin of Lake Ontario. Collected with XAD-2 resin after filtration through glass fibre filters.
SCCP concentrations of 30 ± 14 ng/L were measured in the Red River in Selkirk, Manitoba, over a 6-month period in 1995 (Tomy 1997). Tomy et al. (1999) attributed the SCCPs in the water to a local source, possibly a metal machining/recycling plant in the town of Selkirk, because of the similarity of the formula group abundance profile to that of PCA-60, a commercial SCCP was used as the external standard.

The Ministry of the Environment (2006) in Japan, monitored SCCPs in 6 surface water samples from across the country and did not find any concentrations above the detection limits (which varied from 0.0055 to 0.023 between chain lengths).

**Sediments**

Tomy et al. (1997) measured SCCPs at concentrations around 245 μg/kg dry weight in sediment from the mouth of the Detroit River at Lake Erie and Middle Sister Island in western Lake Erie (sampled August 1995).

SCCPs were detected in all surface sediment samples from harbour areas along Lake Ontario at concentrations ranging from 5.9 to 290 ng/g dry wt. (Table 15). The highest concentrations were found at the most industrialized site (Windermere Basin, Hamilton Harbour), which has well-documented heavy metal, PAH and PCB contamination. Surface sediments from cores at more remote lakes in northern Ontario, Manitoba, and the Canadian Arctic had concentrations ranging from 1.6 to 257 ng/g dry wt. (see Table 16 below).

**Table 15:** SCCP concentrations in surface sediment grab samples collected in 1996 from Lake Ontario harbours (from Muir et al. 2001)

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration (ng/g dry wt.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C₁₀</td>
</tr>
<tr>
<td>Toronto Harbour: inner harbour</td>
<td>0.7</td>
</tr>
<tr>
<td>Toronto Harbour: inner harbour</td>
<td>0.6</td>
</tr>
<tr>
<td>Port Credit Harbour</td>
<td>0.9</td>
</tr>
<tr>
<td>Hamilton Harbour</td>
<td>1.4</td>
</tr>
</tbody>
</table>

* Dots under each bar indicate approximate collection location. Circles indicate relative population sizes of the indicated cities.
The highest concentration of SCCPs in Lake Ontario sediments measured by Marvin et al. (2003) was 410 ng/g dry wt. from the Niagara basin, in an industrialized area. SCCPs were detected in all 26 samples from Lake Ontario, and the average SCCP concentration was 49 ng/g dry wt., which is much higher than sediment concentrations reported for lakes influenced primarily by atmospheric sources (see Table 16 below). Marvin et al. (2003) found that sediment samples from industrialized areas had higher proportions (17–44%) of the longer C13 SCCPs compared with urban non-industrial areas or lakes affected mainly by atmospheric deposition.

The historical concentration profile of SCCPs in Lake Ontario is shown in Figure 6. SCCP residues are found dating back to 1913, with the maximum residues (800 ng/g dry wt.) occurring in the 1970s. SCCP residues observed in 1996 in the surface sediment layer are approximately 390 ng/g dry wt. SCCPs in pre-industrial sediment slices were attributed to sample contamination due to an artefact of sampling.

SCCPs were determined in a dated sediment core collected in Lake St. Francis (Lac Saint-François) downstream of Cornwall in 1996 (there is a CPs manufacturing plant at Cornwall, Ontario – CPs are currently not manufactured at this site). The historical profiles of SCCPs in the core are shown in Figure 7. The results show the presence of relatively low levels of SCCPs compared with Lake Ontario (Muir et al. 2002). The highest SCCP concentrations were found in slice 6, which has a median date of 1985 ± 4 years (Turner 1996). The predominant chain length groups in sediments were C11 and C12. These were present at almost equal proportions of total SCCP.
SCCPs have also been detected in sediments from Hazen Lake (northern Ellesmere Island) and Yaya Lake (Mackenzie River delta, Northwest Territories) in the Canadian Arctic (Tomy et al. 1999) and recently in sediments from Lake DV09, a very remote lake on Devon Island, Nunavut, at low ng/g levels (Stern and Evans 2003; Figure 8). Results for Hazen Lake (Tomy et al. 1999) are close to detection limits; however, the analysis also showed that SCCPs are readily detectable above a method detection limit based on sediment samples pre-dating 1900 that were used as blanks.

The sediment core from Lake DV09, Devon Island, Nunavut (Figure 8), is laminated (or varved) and therefore has much less mixing than the cores analyzed by Tomy et al. (1999); hence, the SCCP profile is sharper than reported in Tomy et al. (1999). A full geochemical description of this core is given by Lockhart et al. (2000). SCCP concentrations in DV09 surface sediments were higher than in Hazen and Yaya lakes. However, the SCCP stratigraphy in DV09 shows the same trends as for Hazen and Yaya lakes.

The detection of SCCPs in sediments from Lake DV09, which is a remote Arctic lake and thus unlikely to be affected by local sources of contamination, is very good evidence for long-range transport and deposition in the Arctic. The downcore profile clearly shows that SCCP concentrations are well above concentrations in the lowest depth slices, dated to prior to manufacture of SCCPs. These “premanufacture” sediments are good indicators of the relative amount of sampling and laboratory
contamination, especially in the case of laminated sediments, where little or no physical mixing occurred. The detection of SCCPs at this remote site and also in other remote Arctic lakes, especially Yaya Lake in the Mackenzie River delta, where levels of SCCPs were also well above background, illustrates the wide dispersal of SCCPs.

Fluxes (µg/m² per year) of SCCPs to various Canadian lake sediments are plotted in Figure 9. These fluxes are for surface slices of each sediment core and represent recent inputs (last 5–10 years). The highest fluxes are observed in lake sediments near urban areas (western Lake Ontario and the south basin of Lake Winnipeg). The lowest fluxes are observed in more remote lakes, including Lake Superior, which are influenced mainly by atmospheric inputs. These results suggest that the most elevated SCCP residues observed in lake sediments are mainly derived from urban areas. The sources contributing to SCCP residues observed in Fox Lake, Yukon, are of uncertain origin. Elevated fluxes of SCCPs to these lakes (Table 16) are higher than for PCBs, which range from about 0.1 to 0.5 µg/m² per year at these latitudes (Muir et al. 1996).

Figure 9: Fluxes (µg/m² per year) of SCCPs to lake sediments in Canada (from Muir et al. 1999; Tomy et al. 1999)

Table 16: Locations, concentrations and fluxes of SCCPs in three Arctic lake sediment cores (Tomy et al. 1999; Stern and Evans 2003)

<table>
<thead>
<tr>
<th>Lake</th>
<th>Latitude/longitude</th>
<th>Surface concentration (ng/g dry wt.)</th>
<th>Sedimentation rate (g/m² per year)</th>
<th>Focusing factor</th>
<th>Flux (µg/m² per year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yaya</td>
<td>69°10’N, 134°39’W</td>
<td>1.6</td>
<td>476</td>
<td>1.6</td>
<td>0.454</td>
</tr>
<tr>
<td>DV09</td>
<td>75°34’N, 89°19’W</td>
<td>17.6</td>
<td>304</td>
<td>2.4</td>
<td>2.21</td>
</tr>
<tr>
<td>Hazen</td>
<td>81°45’N, 71°30’W</td>
<td>4.5</td>
<td>278</td>
<td>4.5</td>
<td>0.893</td>
</tr>
<tr>
<td>S. Winnipeg¹</td>
<td>50°23’N, 96°22’W</td>
<td>176</td>
<td>1000</td>
<td>1.2</td>
<td>147</td>
</tr>
<tr>
<td>N. Winnipeg²</td>
<td>52°28’N, 98°20’W</td>
<td>8</td>
<td>645</td>
<td>1.3</td>
<td>4.00</td>
</tr>
<tr>
<td>Nipigon</td>
<td>49°25’N, 85°30’W</td>
<td>18</td>
<td>411</td>
<td>2.8</td>
<td>2.66</td>
</tr>
</tbody>
</table>
Ballenschmiter (1994) found SCCPs in sediments in Germany at concentrations ranging from <5 to 83 µg/kg dry wt. The 83 µg/kg dry wt. sample was from the Rhine River. The method of analysis was GC-MS using NCI and is reasonably specific for SCCPs (U.K. Environment Agency 2003a).

A recent study of SCCPs and MCCPs in the U.K. environment included 20 aquatic and eight agricultural sites (Nicholls et al. 2001). Nicholls et al. (2001) selected surface sediments from three locations, ranging from 1 to 100 m, from 200 to 300 m and from 1–2 km downstream of municipal sewage treatment effluents. Analysis was by GC-LRMS in negative ion mode with ion trap set at low voltage to reduce fragmentation of the SCCPs/MCCPs. Both SCCPs and MCCPs were judged to be widely distributed in the U.K. environment (Nicholls et al. 2001).

Pribylova et al. (2006) reported concentrations of SCCPs in 36 sediment samples from 11 Czech rivers and 5 drainage vents near industrial areas. Concentrations ranging from non-detect to 347.41 ng/g dry weight were measured. The Ministry of the Environment (2006) in Japan, has monitored SCCPs in 6 bottom sediment samples from across the country and did not find any concentrations above the detection limits (which varied from 0.34 to 3.0 ng g\(^{-1}\) among carbon lengths).

### Biota

#### Fish

SCCPs were analyzed in lake trout and forage fish samples collected in Lake Ontario in 2001 (Muir et al. 2002) and in lake trout samples collected in 1996 (Muir et al. 2001). C\(_{12}\) SCCPs predominated in lake trout, whereas C\(_{11}\) was the major SCCP in sculpin and smelt (Table 17). SCCP concentrations (lipid wt.) were about 2-fold lower in the samples from 2001 compared with concentrations in lake trout of similar age collected in 1996 (Muir et al. 2001). Further studies with additional sampling times are needed to determine if this represents a consistent temporal trend.

### Table 17: Concentrations of SCCPs in lake trout, carp and food web samples from Lake Ontario, including comparison with results from samples collected in 1996 (from Muir et al. 2001, 2002)

<table>
<thead>
<tr>
<th>Species</th>
<th>Year</th>
<th>N</th>
<th>% lipid</th>
<th>Concentrations (ng/g wet wt.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SCCPs</td>
</tr>
<tr>
<td>Lake trout</td>
<td>2001</td>
<td>6</td>
<td>16</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td></td>
<td>22.9</td>
</tr>
<tr>
<td>Rainbow smelt</td>
<td>2001</td>
<td>2</td>
<td>5</td>
<td>21.5</td>
</tr>
<tr>
<td>Slimy sculpin</td>
<td>2001</td>
<td>2</td>
<td>5</td>
<td>27.0</td>
</tr>
<tr>
<td>Alewife</td>
<td>2001</td>
<td>2</td>
<td>3</td>
<td>7.01</td>
</tr>
<tr>
<td>Diporeia</td>
<td>2001</td>
<td>1</td>
<td>2.9</td>
<td>10.6</td>
</tr>
<tr>
<td>Lake trout (Niagara-on-the-Lake)</td>
<td>1996</td>
<td>5</td>
<td>21</td>
<td>58.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5</td>
<td></td>
<td>50.8</td>
</tr>
<tr>
<td>Lake trout (Port Credit, Mississaugaha)</td>
<td>1996</td>
<td>5</td>
<td>26</td>
<td>67.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td></td>
<td>52.5</td>
</tr>
<tr>
<td>Carp (Hamilton Harbour)</td>
<td>1996</td>
<td>3</td>
<td>–</td>
<td>2630</td>
</tr>
</tbody>
</table>

\* sd = standard deviation.
Carp (*Cyprinus carpio*) collected from Hamilton Harbour and lake trout (*Salvelinus namaycush*) collected from two locations in western Lake Ontario (Port Credit [northwest] and Niagara-on-the-Lake [southwest]) in 1996 were analyzed for SCCPs. SCCPs were detected in all samples of carp and lake trout from Lake Ontario (Table 17). The higher concentrations observed in carp are probably due to higher exposure of fish to SCCPs in Hamilton Harbour. Table 15 (above) shows a higher concentration of SCCPs in sediment at Hamilton Harbour than was found at Port Credit Harbour, which would imply that water concentrations of SCCPs in Hamilton Harbour may also have been higher. Alternatively, the higher concentrations of SCCPs in carp from Hamilton Harbour could simply be as a result of bioconcentration. Reth et al. (2005) measured SCCP concentrations ranging between 19 and 286 ng/g wet weight in fish liver (North Sea dab, cod, and flounder) from the North and Baltic Seas.

SFT (2002) measured concentrations of SCCPs in blue mussel and cod livers from Norway. SCCPs were present in all samples with concentrations ranging from 14-130 µg/kg wet weight in mussel and 23-750 µg/kg in cod liver. The Ministry of the Environment (2006) in Japan, has monitored SCCPs in 6 aquatic wildlife samples from across the country and did not find any concentrations above the detection limits (which varied from 0.2 to 1.5 among carbon lengths). Lahaniatis et al. (2000) reported mean values for individual chain length (C10-C13) SCCPs ranging between 7 - 206 µg/kg of fish oil and 6 – 135 µg/kg in fish (sprat, redfish, herring, halibut, sardine, and trout) from a variety of sites in England, Norway, Chile, Greece, Germany, Iceland, France, USA, and the North Sea.

**Marine mammals**

Tomy et al. (2000) reported levels of SCCPs in the blubber of ringed seal from Eureka, southwest Ellesmere Island, beluga whales from northwest Greenland, the Mackenzie Delta and the St. Lawrence River estuary, and walrus from northwest Greenland (Table 18). The data in Table 18 show that concentrations of SCCPs in the St. Lawrence beluga were approximately 4 times higher than concentrations in beluga from Greenland and the Mackenzie Delta. The elevated levels of SCCPs in belugas from the St. Lawrence River are consistent with the findings of elevated levels of other organochlorines by Muir et al. (1996), who suggested that this was a food chain effect attributed to local source contamination. The data also show that in many cases SCCP concentrations in Arctic biota are lower than those of other persistent organochlorines, however a few samples showed higher SCCPs.

**Table 18: Concentrations of C_{10-13} SCCPs and other persistent organic pollutants in blubber of marine mammals from the Arctic and the St. Lawrence River estuary (from Tomy et al. 1998b, 2000)**

<table>
<thead>
<tr>
<th>Species</th>
<th>Location</th>
<th>Gender</th>
<th>Year</th>
<th>N</th>
<th>Lipid (%)</th>
<th>Concentrations' (ng/g wet wt.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ΣDDT</td>
</tr>
<tr>
<td>Beluga</td>
<td>St. Lawrence estuary</td>
<td>M + F</td>
<td>1988</td>
<td>5</td>
<td>83.2 ± 7.3</td>
<td>79 800 ± 50 846</td>
</tr>
<tr>
<td>Beluga</td>
<td>Mackenzie Delta</td>
<td>M</td>
<td>1995</td>
<td>17</td>
<td>90.9 ± 1.1</td>
<td>3390 ± 1090</td>
</tr>
<tr>
<td>Beluga</td>
<td>Sanikiluaq, Hudson Bay</td>
<td>M</td>
<td>1994</td>
<td>10</td>
<td>94.8 ± 0.7</td>
<td>14 740 ± 6850</td>
</tr>
<tr>
<td>Beluga</td>
<td>Pangnirtung, Cumberland Sound</td>
<td>M</td>
<td>1994</td>
<td>31</td>
<td>90.5 ± 3.2</td>
<td>4530 ± 1840</td>
</tr>
<tr>
<td>Beluga</td>
<td>Kimmirut</td>
<td>M + F</td>
<td>1994</td>
<td>6</td>
<td>93.2 ± 1.6</td>
<td>5330 ± 3330</td>
</tr>
<tr>
<td>Species</td>
<td>Location</td>
<td>Gender</td>
<td>Year</td>
<td>N</td>
<td>Lipid (%)</td>
<td>Concentrations(^1) (ng/g wet wt.)</td>
</tr>
<tr>
<td>-----------</td>
<td>------------------------</td>
<td>--------</td>
<td>------</td>
<td>---</td>
<td>-----------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ΣDDT</td>
</tr>
<tr>
<td>Beluga</td>
<td>NW Greenland (Sassat/Nuussuaq)</td>
<td>M + F</td>
<td>1989</td>
<td>4</td>
<td>88.3 ± 3.9</td>
<td>2220 ± 584</td>
</tr>
<tr>
<td>Ringed seal</td>
<td>Pangnirtung, Cumberland Sound</td>
<td>M + F</td>
<td>1993</td>
<td>6</td>
<td>94.6 ± 1.2</td>
<td>855 ± 1122</td>
</tr>
<tr>
<td>Ringed seal</td>
<td>SW Ellesmere Island, Eureka</td>
<td>M + F</td>
<td>1994</td>
<td>6</td>
<td>90.3 ± 1.8</td>
<td>660 ± 240</td>
</tr>
<tr>
<td>Walrus</td>
<td>NW Greenland</td>
<td>M</td>
<td>1978</td>
<td>2</td>
<td>83 ± 0.2</td>
<td>33 ± 9.2</td>
</tr>
</tbody>
</table>

\(^1\) Arithmetic means ± standard deviations.

Tomy et al. (2000) also observed that the concentration profiles for the Arctic marine mammals show a predominance of the shorter carbon chain length congeners (i.e., the C\(^{10}\) and C\(^{11}\) formula groups). This is significant, because Drouillard et al. (1998a) showed that these congeners are the more volatile components of SCCP mixtures, which show a trend of decreasing VPs with increasing carbon chain length and degree of chlorination. These results suggest that these compounds enter this region by long-range atmospheric transport. Tomy et al. (2000) concluded that “Although only a few samples have been analyzed in this study, it is clear that SCCPs are present in Arctic food webs and are being transported to these remote regions either in the atmosphere or ocean currents.” In contrast, the formula group abundance profile for the belugas from the St. Lawrence River estuary shows a shift towards the less volatile components — i.e., higher carbon chain length inherent to commercial formulations. The higher proportions of the less volatile components in the concentration profile suggest that local sources of SCCPs, possibly from the Great Lakes or the industrialized regions of the lower St. Lawrence River, are the most important sources of input of SCCPs to this area.

There are few other published data on SCCPs in marine mammals for comparison, and differences in analytical methodology make comparisons problematic. Jansson et al. (1993) reported an SCCP concentration of 130 ng/g wet wt. in ringed seal blubber from Svalbard, which is quite similar to levels found by Tomy et al. (2000). It should be noted, however, that the substances measured in Jansson’s study were CPs of unspecified chain length with 6–16 chlorine atoms per molecule and so could have also included MCCPs and LCCPs.

### Terrestrial wildlife

To date, very limited information is available on SCCP concentrations in tissues of terrestrial wildlife. In Sweden, Jansson et al. (1993) reported CP concentrations (unspecified chain length) in rabbit (Revingeshed, Skåne), moose (Grismsö, Västmanland), reindeer (Ottsjö, Jaämtland) and osprey (from various regions in Sweden) to be 2.9, 4.4, 0.14 and 0.53 µg/g lipid wt., respectively.

CEFAS (1999) reported the concentrations of SCCPs in sewage sludge, soil, and earth worms associated with uses of chlorinated paraffins in the United Kingdom in the summer of 1998. Concentrations in earthworms ranged between <0.1 to 0.7 mg/kg dry weight.

Campbell and McConnell (1980) determined levels of C\(^{10–20}\) CPs in birds (Table 19) and seabird eggs in the United Kingdom (Table 20), as well as in water and sediments. The C\(^{10–20}\) levels are likely to be dominated by contributions from the
SCCPs and MCCPs. The method of analysis used was TLC with argentation. The results of Campbell and McConnell (1980) are generally regarded as valid, because similar concentrations in various media in the United Kingdom have been reported using GC-MS methods (e.g., CEFAS 1999; Nicholls et al. 2001). Nevertheless, they must be regarded as semi quantitative.

Table 19: Concentrations of CPs in birds from the United Kingdom (from Campbell and McConnell 1980)

<table>
<thead>
<tr>
<th>Species</th>
<th>Organ</th>
<th>Concentration (µg/kg wet wt.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>C_{10–20}</td>
</tr>
<tr>
<td>Grey heron (Ardea cinerea)</td>
<td>Liver</td>
<td>100–1200</td>
</tr>
<tr>
<td>Guillemot (common murre) (Uria aalge)</td>
<td>Liver</td>
<td>100–1100</td>
</tr>
<tr>
<td>Herring gull (Larus argentatus)</td>
<td>Liver</td>
<td>200–900</td>
</tr>
</tbody>
</table>

1 Table taken from U.K. Environment Agency (2001).
2 ND = Not detected (detection limit = 100 µg/kg wet wt.).

Table 20: Concentrations of CPs in seabird eggs (from Campbell and McConnell 1980)

<table>
<thead>
<tr>
<th>Concentration (µg/kg)</th>
<th>Number of eggs containing CPs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C_{10–20}</td>
</tr>
<tr>
<td>Not detected (&lt;50)</td>
<td>7</td>
</tr>
<tr>
<td>50</td>
<td>3</td>
</tr>
<tr>
<td>100</td>
<td>3</td>
</tr>
<tr>
<td>200</td>
<td>5</td>
</tr>
<tr>
<td>300</td>
<td>1</td>
</tr>
<tr>
<td>400</td>
<td>2</td>
</tr>
<tr>
<td>600</td>
<td>1</td>
</tr>
<tr>
<td>&gt;600 (=2000 µg/kg)</td>
<td>1</td>
</tr>
</tbody>
</table>

1 Species included were great cormorant (Phalacrocorax carbo); northern gannet (Morus bassanus); great skua (Catharacta skua); guillemot (common murre) (Uria aalge); black-legged kittiwake (Rissa tridactyla); Atlantic puffin (Fratercula arctica); Manx shearwater (Puffinus puffinus); razorbill (Alca torda) and shag (Phalacrocorax aristotelis).

Reth et al. (2006) quantified SCCPs in liver from Arctic Char and seabirds (little auk and kittiwake) collected at Bear Island (European Arctic) as well as in cod from Norway. Concentrations between 5 and 88 ng/g wet weight were measured.

Human breast milk and food

Tomy (1997) found that SCCPs (around 60–70% chlorine by weight) were present at a concentration of 11–17 µg/kg lipid (mean concentration 13 µg/kg lipid) in human breast milk from Inuit women living on the Hudson Strait in northern Quebec, Canada.

A recent study has found SCCPs to be present in human breast milk samples from the United Kingdom (Thomas and Jones 2002). In all, 22 breast milk samples were analyzed (eight from Lancaster and 14 from London, randomly chosen from a limited number of samples collected for a different study). SCCPs were found at concentrations of 4.6–110 µg/kg lipid in five out of eight samples from Lancaster and at concentrations of 4.5–43 µg/kg lipid in seven out of 14 samples from London. No SCCPs were found in the remaining samples (the detection limit of the method used varied with sample size but was in the range 1.6–15 µg/kg lipid). Although not calculated in the original paper, it is possible to estimate that the mean level found in breast milk was around 20 ± 30 µg/kg lipid (based on the positive findings alone) or 12 ± 23 µg/kg lipid (assuming that not detected = half the detection limit).
Thomas and Jones (2002) also determined the levels of SCCPs in a sample of cow’s milk from Lancaster and single butter samples from various regions of Europe (Denmark, Wales, Normandy, Bavaria, Ireland and southern and northern Italy). SCCPs were not detected in the cow’s milk sample (detection limit <1.2 µg/kg lipid) but were found in the butter samples from Denmark at 1.2 µg/kg and Ireland at 2.7 µg/kg. The detection limit for the butter samples ranged between 0.72 and 1.1 µg/kg.

A follow up study by Thomas et al. (2003) used more sensitive analytical procedures to analyze SCCPs in breast milk samples (20 from London, 5 from Lancaster). They found concentrations ranging between 49 and 820 µg/kg lipid.

In a market basket survey (KAN-DO Office and Pesticides Team, 1995) of 234 ready-to-eat foods, which represented approximately 5000 food types in American diets, “Chlorowax 500C”) was detected once, in enriched white bread, at a concentration of 0.13 µg/g. Food items were screened by gas or liquid chromatography using ion-selective detectors. Findings were confirmed by unspecified analysis.

2.4 Hazard Assessment for Endpoints of Concern

ENVIRONMENTAL EFFECTS

Microorganisms
Hildebrecht (1972) concluded that a C_{10–13} CP (59% chlorine) and Exchlor 5C (composition unknown) did not affect oxygen utilization by sewage sludge bacteria (species not reported) at concentrations between 1 and 200 mg/L. Birtley et al. (1980) reported that there was no indication that a C_{10–13} CP (49% chlorine) was toxic to four strains of Salmonella typhimurium at concentrations as high as 2500 µg/plate. Madeley et al. (1983c) found that a short-chain polychlorinated alkane (58% chlorine) caused significant inhibition (>10%) of gas production by anaerobic microorganisms at concentrations of 3.2, 5.6 and 10%. Effects were observed only for the first 3–4 days of the experiments; by day 10, gas production had returned to normal levels.

The following study is described in EC (2000) and U.K. Environment Agency (2003a): Koh and Thiemann (2001) investigated the toxicity of two SCCPs to bioluminescent bacteria Vibrio fischeri. The endpoint was the concentration of the SCCP solution that would cause <20% inhibition of the light emission of the bacteria at 585 nm. The long-term (24-hour) test found a No-Observed-Effect Concentration (NOEC)/EC_{20} value of 0.1 mg/L for the 56% chlorine by weight C_{10–13} CP and a NOEC/EC_{20} of 0.05 mg/L for the 62% chlorine by weight C_{10–13} CP.

Pelagic aquatic organisms
There have been only a limited number of studies on the aquatic toxicity of SCCPs that have been published since the reviews of Tomy et al. (1998a) and the risk assessment of SCCPs by the EU (EC 2000). A summary of these studies is given in Table 21, and they are described further below.

Table 21: Recent studies of the aquatic toxicity of SCCPs

<table>
<thead>
<tr>
<th>Species</th>
<th>CP</th>
<th>Exposure</th>
<th>Concentration</th>
<th>Notes</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Japanese medaka embryó³ (Oryzias latipes)</td>
<td>C_{10}H_{15.5}Cl_{6.5}</td>
<td>20-day static test</td>
<td>5.9–9600 µg/L</td>
<td>LOEC = 460 µg/L NOEC = 62 µg/L</td>
<td>Fisk et al. (1999)</td>
</tr>
<tr>
<td>Species</td>
<td>CP</td>
<td>Exposure</td>
<td>Concentration</td>
<td>Notes</td>
<td>Reference</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------</td>
<td>---------------------------------</td>
<td>----------------</td>
<td>-------------------------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Japanese medaka</td>
<td>C_{10}H_{15.3}Cl_{6.7}</td>
<td>20-day static test</td>
<td>4.7–7700 µg/L</td>
<td>LOEC = 370 µg/L NOEC = 50 µg/L</td>
<td>Fisk et al. (1999)</td>
</tr>
<tr>
<td>Japanese medaka</td>
<td>C_{11}H_{18.4}Cl_{5.6}</td>
<td>20-day static test</td>
<td>5.4–8900 µg/L</td>
<td>LOEC = 420 µg/L NOEC = 57 µg/L</td>
<td>Fisk et al. (1999)</td>
</tr>
<tr>
<td>Japanese medaka</td>
<td>C_{12}H_{19.5}Cl_{6.5}</td>
<td>20-day static test</td>
<td>0.7–270 µg/L</td>
<td>LOEC = 55 µg/L NOEC = 9.6 µg/L</td>
<td>Fisk et al. (1999)</td>
</tr>
<tr>
<td>Rainbow trout (Oncorhynchus mykiss)</td>
<td>C_{10}H_{15.3}Cl_{6.7}</td>
<td>40 days dietary</td>
<td>0.021–15 µg/g food</td>
<td>No negative effects on mortality, growth or liver somatic index observed</td>
<td>Fisk et al. (1996, 2000)</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>C_{10}H_{15.5}Cl_{6.5}</td>
<td>21–85 days dietary</td>
<td>0.87–62 µg/g in food</td>
<td>85-day NOEC = 0.10 µg/g in whole fish 21-day LOEC = 0.84 µg/g in whole fish</td>
<td>Cooley et al. (2001)</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>C_{10}H_{15.3}Cl_{6.7}</td>
<td>21–85 days dietary</td>
<td>0.84–74 µg/g in food</td>
<td>85-day NOEC = 0.099 µg/g in whole fish 21-day LOEC = 0.92 µg/g in whole fish</td>
<td>Cooley et al. (2001)</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>C_{11}H_{18.4}Cl_{5.6}</td>
<td>21–85 days dietary</td>
<td>0.18–14 µg/g in food</td>
<td>85-day NOEC = 0.10 µg/g in whole fish 21-day LOEC = 5.5 µg/g in whole fish</td>
<td>Cooley et al. (2001)</td>
</tr>
<tr>
<td>Rainbow trout</td>
<td>C_{12}H_{19.5}Cl_{6.5}</td>
<td>21–85 days dietary</td>
<td>1.9–58 µg/g in food</td>
<td>85-day NOEC = 0.14 µg/g in whole fish 21-day LOEC = 0.79 µg/g in whole fish</td>
<td>Cooley et al. (2001)</td>
</tr>
</tbody>
</table>

1 Lowest-Observed-Effect Concentration (LOEC) and NOEC in medaka are based on any effect in egg or larvae within the first 20 days after laying.
2 LOEC and NOEC in rainbow trout are based on feeding behaviour, response to disturbance and histopathological lesions in the liver of group exposed to intermediate concentrations.

Fisk et al. (1999) studied the toxicity of four C_{10}, C_{11}, and C_{12} SCCP compounds (single chain lengths with mixtures of isomers) to Japanese medaka (Oryzias latipes) embryos. Lowest-Observed-Effect Concentrations (LOECs) ranged from 55 µg/L for C_{10}H_{20}Cl_{7} to 460 µg/L for C_{10}H_{16}Cl_{7}. Effects in eggs and larvae over the first 20 days after laying included oil globule migration away from the head and thinning of blood vessels between the head and the oil globule. Toxicity was independent of carbon chain length and chlorine content. The mechanism of toxicity to the embryos was suggested to be narcosis. It should be noted that this study did not meet some of the criteria specified in the OECD 201 test guideline for a fish early life stage test, including the following points: there were only 10 eggs per test concentration as opposed to 60 stipulated by OECD, the test took place in sealed vials and the study does not describe dissolved oxygen levels, and
the study was carried out for approximately 3 days post-hatch, as opposed to the OECD guideline of 30 days post-hatch.

Hill and Maddock (1983) found that the hatchability and survival of larvae of the sheepshead minnow (*Cyprinodon variegates*) was unaffected by 28-day exposure to concentrations of SCCPs (58% chlorinated) ranging between 2.4 and 54.8 \( \mu g/L \). They also observed increases in growth over the acetone controls. In a similar 32-day study with sheepshead minnow larvae and SCCP concentrations ranging between 36.2 and 620.5 \( \mu g/L \) the larvae from the highest exposure group were significantly smaller than the acetone group, however larvae from lower exposures were larger than the control. No effects on survival or hatchability were observed.

Fisk et al. (1996, 2000) studied the accumulation of several \(^{14}\)C-labelled SCCPs (56–69% chlorine by weight) by juvenile rainbow trout (initial weights 2–7 g) during a 40-day exposure period (Table 21). The daily feeding rate was 1.5% of the mean body weight, and two exposure concentrations for each substance were used. At these feeding rates, none of the compounds was found to have any negative effect on the growth or liver somatic index of juvenile rainbow trout. Concentrations in the trout were much less than the LOECs reported for SCCPs in the Japanese medaka study described above (Fisk et al. 1999).

Cooley et al. (2001) examined the behaviour and liver and thyroid histology of juvenile rainbow trout of the same four \( C_{10}, C_{11}, \) and \( C_{12} \) SCCP compounds as in Fisk et al. (1999) via dietary exposure. The exposed trout showed responses indicative of a narcotic mode of action, such as delayed or absent startle response and reduced feeding. Severe liver histopathologies were observed in trout exposed to \( C_{10}H_{15}Cl_7 \) and \( C_{11}H_{18}Cl_6 \) (whole fish concentrations of 0.92 and 5.5 \( \mu g/g \) wet wt., respectively), consisting of extensive fibrous lesions and hepatocyte necrosis not seen in controls or lower exposed fish. No thyroid lesions were observed. LOECs for the \( C_{10–12} \) SCCPs ranged from 0.79 to 5.5 \( \mu g/g \) in whole fish tissue following dietary exposure to concentrations ranging from 0.84 to 74 \( \mu g/g \) in food. While a reduced feeding rate was observed in some fish at medium exposure, the fish weight and Liver Somatic Index (LSI) remained the same. These concentrations are within the range of SCCP concentrations seen in carp from Hamilton Harbour (Muir et al. 2001) and in yellow perch from the Detroit River (Tomy et al. 1997) but considerably higher than concentrations found in Lake Ontario fish. Nevertheless, this suggests that histological effects resulting from exposure to SCCPs may be occurring in fish in areas of relatively high exposure, such as Areas of Concern in the Great Lakes.

The lowest toxic effect level identified for a pelagic freshwater aquatic species is 8.9 \( \mu g/L \), which is the 21-day chronic LOEC for *Daphnia magna* (Thompson and Madeley 1983a). The effect was for mortality of the offspring. The NOEC is 5 \( \mu g/L \).

Effects on *Daphnia* and mysid shrimp have been reported at similar concentrations in two other studies. In a 14-day static renewal study using daphnids, 50% mortality was observed after 5 days at 10 \( \mu g/L \) (Thompson and Madeley 1983a). The most sensitive measurement endpoint identified for a marine species is 7.3 \( \mu g/L \), which is the 28-day chronic NOEC for the mysid shrimp (*Mysidopsis bahia*) (Thompson and Madeley 1983b).

Thompson and Shillabeer (1983) carried out a further study on mussels *Mytilus edulis* using a 58% chlorinated SCCP using only two exposure concentrations. Groups of 30 mussels were exposed to concentrations of 2.3 \( \mu g/L \) or 9.3 \( \mu g/L \) in a flow through sea water system for 12-hours. No mortalities were observed but growth was reduced in the 9.3 \( \mu g/L \) exposure.
Thompson and Madley (1983c) reported a NOEC of 12.1 µg/L in a 10-day study with marine algae *Skeletonema costatum*. The toxic effects were transient with no effects seen at any concentration after 7 days.

Buryškova et al. (2006) observed developmental malformations and reduced embryo growth in *Xenopus laevis* frog at 5 mg/L and higher concentrations of a commercial mixture of SCCPs (C12 56% chlorine). The results were not related to chlorinated pattern and significant induction of the biomarker phase II detoxification enzyme glutathione S-transferase was observed at 0.5 mg/L.

**Benthic organisms**

An equilibrium partitioning approach (Di Toro et al. 1991) using the most sensitive chronic measurement endpoint identified for a pelagic freshwater invertebrate aquatic species (8.9 µg/L) was used to estimate the toxicity to benthic organisms, since a valid measurement endpoint was not available for a sediment-dwelling invertebrate. A study was conducted using the midge *Chironomus tentans* (EG&G Bionomics 1983), but exposure was via water only. The following equation was used to calculate the LOEC for benthic invertebrates:

\[
LOEC_{\text{benthic}} = f_{oc} \cdot K_{OC} \cdot LOEC_{\text{pelagic}}
\]

where:

- \( f_{oc} \) is 0.02, based on the mean organic carbon content for surficial sediment samples from Lake Ontario, expressed on a dry weight basis (Kemp et al. 1977);
- \( K_{OC} \) is the organic carbon–water partition coefficient, based on a measured value of 199 500 L/kg for a C\(_{10}\) and C\(_{13}\) CP with ~55% by weight chlorine content (Thompson et al. 1998); and
- \( LOEC_{\text{pelagic}} \) is 8.9 µg/L, based on a 21-day chronic study for *Daphnia magna* (Thompson and Madeley 1983a).

Therefore:

\[
LOEC_{\text{benthic}} = 0.02 \times 199\ 500\ \text{L/kg} \times 8.9\ \mu g/L
\]

\[
= 35\ 511\ \mu g/kg\ \text{dry wt.}
\]

\[
= 35.5\ \text{mg/kg dry wt.}
\]

**Soil-dwelling organisms**

A very recently published study (Bezchlebova, et al, 2007) investigated the effects of SCCPs (64% chlorine content) on five species of soil organisms (colembola, earthworms, nematodes) and on soil microorganisms (for carbon transformation). *Folsomia candida* (colembola) was identified as the most sensitive organism, with LC\(_{50}\), EC\(_{50}\) and EC\(_{10}\) values of 5733 mg/kg, 1230 mg/kg, and 660 mg/kg respectively. A PNEC of 5.28 mg/kg was estimated for the soil environment based on their data. The authors compared their results with those of Sverdrup, et al (2006), who investigated the effects of SCCPs (60% chlorine content) on one soil organism (earthworm), soil nitrifying bacteria, and red clover. These authors found the nitrifying bacteria to be the most sensitive, with an EC\(_{10}\) value of 570 mg/kg. This led to an estimated PNEC of 57 mg/kg.

As a comparison, an equilibrium partitioning approach (Di Toro et al. 1991) using the most sensitive measurement endpoint identified for a pelagic freshwater invertebrate species (8.9 µg/L) was used to estimate the toxicity to soil-dwelling organisms:

\[
LOEC_{\text{soil}} = f_{oc} \cdot K_{OC} \cdot LOEC_{\text{pelagic}}
\]
where:

- \( f_{oc} \) is 0.02, a standard value given in Mackay (1991); and
- \( K_{OC} \) and LOEC\textsubscript{pelagic} values are given in Section 6.1.3.

Therefore:

\[
\text{LOEC\textsubscript{soil}} = 0.02 \times 199\ 500 \text{ L/kg} \times 8.9 \text{ µg/L}
\]

\[
= 35\ 511 \text{ µg/kg dry wt.}
\]

\[
= 35.5 \text{ mg/kg dry wt.}
\]

The LOEC\textsubscript{soil} for SCCPs is therefore calculated to be 35.5 mg/kg dry wt.

**Birds**

The EU (EC 2000) reviewed the following avian reproduction study (no reference given), carried out with a C\textsubscript{10-12} CP (58% chlorine), and deemed it to be of good quality. The study was carried out to GLP and was based on the Mallard Reproduction Test (August 1982) of the EPA Environmental Effects Test Guidelines (U.S. EPA 1982).

The study was a 22-week feeding study, including a 9-week pre-egg-laying period without photostimulation, a 3-week pre-egg-laying period with photostimulation and a 10-week egg-laying period with photostimulation. Birds were induced (by photoperiod manipulation) to lay eggs. Eggs were collected over a 10-week period, and the young were observed for 14 days (note that the young were not fed with the test substance). Mortality of adults, egg production, cracked eggs, eggshell thickness, viability, hatchability and effects on young birds were all compared with controls.

The mean measured concentrations were 29, 168 and 954 mg/kg in diet. Twenty pairs of adults were used at each concentration and as control. The lowest level seen to cause slight effects in this study was 954 mg/kg food, which caused a slight, but statistically significant, decrease (by 0.020 mm) in mean eggshell thickness. EC (2000) considered the biological significance of the decrease in eggshell thickness to be questionable, since the mean eggshell thickness in the 954 mg/kg group (0.355 mm) was still in the range of normal values given in the OECD guidelines (0.35–0.39 mm), and no increase in cracked eggs was seen at this dose (EC 2000). No significant difference in the number of eggs laid, number of cracked eggs or mean egg weight was seen in any treatment group when compared with controls.

**Mammals**

The following is a brief summary of the relevant information reviewed in the EU Risk Assessment Report (EC, 2000) and the draft Health Canada Assessment Report (2003). Health Canada (2003) reviewed mammalian toxicity studies for SCCPs that have been published since the Government of Canada (1993a) risk assessment. However, most of the studies were available only in abstract or summary form, with no quantitative data or statistical analyses presented. These studies are related to effects of SCCPs on the liver, kidney and thyroid in rats. Only one complete report was identified (Wyatt et al. 1993).

Wyatt et al. (1993) exposed male rats (Alpk:APfSD strain) by gavage for 14 days to two SCCPs (Chlorowax 500C, C\textsubscript{10-13}, 58% chlorine; or Cereclor 56L, C\textsubscript{10-13}, 56% chlorine). Doses were 0, 10, 50, 100, 250, 500 and 1000 mg/kg-bw per day. For the 58% chlorine product, absolute and relative liver weights
were significantly increased in a dose-related manner, beginning at 100 mg/kg-bw per day. For the 56% chlorine SCCP, the pattern for absolute liver weight was irregular; however, relative liver weight was increased in a dose-related manner, which was significant at 50 mg/kg-bw per day and higher. Peroxisomal fatty acid β-oxidation activity, as indicated by palmitoyl coenzyme A oxidation, was significantly increased at and above 250 mg/kg-bw per day.

In a 13-week oral (gavage) rat study by IRDC (1984), increases in liver and kidney weight and hypertrophy of the liver and thyroid occurred at doses of 100 mg/kg-bw per day. The No-Observed-Effect Level (NOEL) was the next lowest dose of 10 mg/kg-bw per day. Health Canada (2003) used this NOEL as the Critical Toxicity Value (CTV) for the risk assessment of SCCPs.

The results from available acute studies and skin irritation studies indicate that effects are not dependant upon the chain length and degree of chlorination. SCCPs are of low acute toxicity in animals and are not mutagenic. No evidence is available for human carcinogenicity due to SCCPs. Rodent studies showed dose related increases in adenomas and carcinomas in the liver, thyroid, and kidney. There continues to be contention over the mechanisms of these tumors and whether they are relevant for human health.

There are no data on fertility or developmental effects for humans. No changes in reproductive organs were observed in a 13 week study with rats and mice dosed with 5000 and 2000 mg/kg/day of an SCCP. In addition, developmental effects were observed in rats at 2000 mg/kg/day but not at lower doses.

Overall, SCCPs are of low toxicity with the principal toxicological issue being for general non-specific toxicity following repeated exposure. NOAELs for general toxicity of 100 and 1000 mg/kg/day were identified in rats and mice respectively. SCCPs were found to fulfill the criteria of decision 1998/2 of the Executive body (UNECE, 1998) in regard to adverse effects on human health and the environment.

Upper-bound estimates of intake of SCCP for the general Canadian population and the assumptions upon which they are based were calculated by Health Canada (2003). For each age group in the Canadian population, virtually all of the estimated intake is from food. The upper-bound estimated intake of breast-fed infants was 1.7 µg/kg-bw per day, and that of formula-fed infants was 0.01 µg/kg-bw per day. For the remaining age groups, intakes ranged from 5.1 µg/kg bw per day for adults over 60 years of age to 26.0 µg/kg-bw per day for infants who were not formula fed (i.e., those being introduced to solid foods ). According to Health Canada (2003) the upper-bounding estimates of daily intake of SCCP approach or exceed the TDI for these compounds, which, on the basis of available information, is likely also protective for carcinogenicity.

The International Agency for Research on Cancer considers there is sufficient evidence for the carcinogenicity (possibly carcinogenic – groups 2B) of a commercial chlorinated paraffin product of average carbon-chain length C12 and average degree of chlorination 60% in experimental animals (IARC, 1990).

**Summary of the environmental toxicology of SCCPs**

The most sensitive toxicity endpoints for SCCPs is summarized in Table 22. The most sensitive aquatic species appears to be *Daphnia*, with chronic NOECs of 5 µg/L. It therefore appears that SCCPs can harm sensitive aquatic organisms at relatively
low concentrations (i.e. below threshold criteria of 1 mg/L used to categorize substances on Canada’s Domestic Substances List).

Table 22: Overview of the most sensitive long-term environmental LOEC/NOEC data for SCCPs

<table>
<thead>
<tr>
<th>Species/endpoint</th>
<th>Effect</th>
<th>NOEC</th>
<th>LOEC</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Daphnia magna</em></td>
<td>Mortality of offspring, 21 days</td>
<td>5 µg/L</td>
<td>8.9 µg/L</td>
<td>Thompson and Madeley (1983a)</td>
</tr>
<tr>
<td>Benthic organisms</td>
<td>Equilibrium partitioning using <em>Daphnia</em> LOEC</td>
<td>NA</td>
<td>88.8 mg/kg dry weight</td>
<td>Thompson and Madeley (1983a)</td>
</tr>
<tr>
<td>Japanese medaka embryos, early life stage effects</td>
<td>20-day study, oil globule migration away from head, thinning of blood vessels</td>
<td>9.6 µg/L</td>
<td>55 µg/L</td>
<td>Fisk et al. (1999)</td>
</tr>
<tr>
<td>Soil organisms</td>
<td>Equilibrium partitioning using <em>Daphnia</em> LOEC</td>
<td>NA</td>
<td>35.5 mg/kg dry weight (equilibrium partitioning)</td>
<td>Thompson and Madeley (1983a)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>570-5733 mg/kg (experimental)</td>
<td>Bezchlebova et al., (2007); Sverdup et al. (2006)</td>
</tr>
<tr>
<td>Mallard reproduction</td>
<td>Reduced eggshell thickness</td>
<td>168 mg/kg food</td>
<td>954 mg/kg food</td>
<td>EC (2000)</td>
</tr>
<tr>
<td>Rat histology</td>
<td>Hypertrophy of the liver and thyroid, increases in liver and kidney weight</td>
<td>10 mg/kg-bw per day</td>
<td>100 mg/kg-bw per day</td>
<td>IRDC (1984)</td>
</tr>
</tbody>
</table>

ASSESSMENT OF POTENTIAL TO CAUSE ECOLOGICAL HARM

It is acknowledged that when risks for persistent and bioaccumulative substances (such as SCCPs) are determined using standard methods, the risks may be underestimated. Table 23 provides exposure and toxicity values associated with margins of exposures for each identified class of receptors (e.g., pelagic organisms, benthic organisms) but does not incorporate application factors for uncertainties, nor for the conservative approaches that could be considered for persistent and bioaccumulative substances. An EEV (estimated exposure value) and CTV (critical toxicity value) was selected based on available empirical data. The maximum reported value was used as the EEV. CTVs typically represent the lowest chronic ecotoxicity value from an available and acceptable data set. Current exposures are below effects values however they may be approaching in some cases.

Table 23: List of Estimated Exposure Values (EEV) and Critical Toxicity Values (CTV), for SCCPs

<table>
<thead>
<tr>
<th>Organism</th>
<th>EEV</th>
<th>Sample</th>
<th>CTV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelagic</td>
<td>44.8 ng/L&lt;sup&gt;1&lt;/sup&gt;</td>
<td>STP, Hamilton, ON</td>
<td>8900 ng/L&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Benthic</td>
<td>0.41 mg/kg&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Lake Ontario sediment</td>
<td>35.5 mg/kg&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
### Soil Dwelling

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.64 mg/kg⁵</td>
<td></td>
</tr>
<tr>
<td>UK sewage after 10 years soil app.</td>
<td>35.5 mg/kg⁴</td>
</tr>
<tr>
<td>570 – 5733 mg/kg⁶</td>
<td></td>
</tr>
</tbody>
</table>

### Secondary Consumer

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.63 mg/kg⁷</td>
<td></td>
</tr>
<tr>
<td>Carp from Hamilton Harbour, Lake Ontario</td>
<td>1000 mg/kg⁸</td>
</tr>
</tbody>
</table>

1. A dilution factor of 10 applied to the value for final effluent of sewage treatment plant in Hamilton Ontario (448 ng/L) (Environment Canada, 2005)
2. 21-day chronic LOEC value for *Daphnia magna* (Thompson and Madeley, 1983a)
3. Measured in surface sediments from Lake Ontario (Marvin et al. 2003)
4. LOEC for *Daphnia magna* using equilibrium partitioning approach (Environment Canada, 2004)
5. Calculation of soil amended with sewage sludge for 10 years application (Environment Canada, 2004)
6. Experimental data for soil organisms and microorganisms reported by Bezchlebova et al., (2007) and Svedrup et al. (2006)
7. Carp from Hamilton Harbour in Lake Ontario (Environment Canada, 2004)
8. 13 week rat oral gavage study LOAEL (IRDC, 1984) adapted to otter (Environment Canada, 2004)

### 3. SYNTHESIS OF INFORMATION

SSCPs are considered to be persistent, bioaccumulative, inherently toxic to some species, and to undergo long range transport to remote areas.

Total reported annual usage of CPs was high in several countries but several have had notable reductions in recent years. For example, use in Canada was approximately 3000 tonnes in 2000 and 2001, in Switzerland 70 tonnes were used in 1994 and likely reduced by 80% now, and Australia reduced by 80% between 2001 and 2003. Releases can occur during production, storage, transportation, and use of SCSPPs. Facility wash down and spent metalworking / metal cutting fluids are sources to aquatic ecosystems. Although data are limited, the major sources of release of SCSPPs are likely the formulation and manufacturing of products containing SCSPPs, such as polyvinyl chloride (PVC) plastics, and use in metalworking fluids.

SCCPs are not expected to degrade significantly by hydrolysis in water and dated sediment cores indicate that they persist in sediment longer than 1 year. SCCPs have atmospheric half lives ranging from 0.81 to 10.5 days indicating that they are also relatively persistent in air. SCCPs also have vapour pressures in the range of known persistent organic pollutants that undergo long range atmospheric transport. The Henry’s law constant implies substantial partitioning from water to air under certain conditions, thus facilitating atmospheric partitioning and transport. SCCPs have been detected in a diverse array of environmental samples (air, sediment, water, waster water, fish and marine mammals) and in remote areas such as the Arctic which is additional evidence of long range transport. In addition Arctic Contamination Potential (ACP) modeling and OECD LRTP screening tools suggests that SCCPs have moderate ACP when emitted to air and have properties similar to known POPs that are known to undergo long range transport.

Bioaccumulation factors (BAFs) of 16 440–25 650 wet weight (wet wt.) in trout from Lake Ontario indicate that SCCPs can bioaccumulate to a high degree in aquatic biota. This is supported by modeling data for log Kow and bioaccumulation factors which indicate that SCCPs bioaccumulate. In addition, biomagnification factors for some SCCPs have been found to be greater than 1. High concentrations of SCCPs in upper tropic level organisms is additional evidence of bioaccumulation. The bioaccumulation of SCCPs is generally supported by the high concentrations of SCCPs measured in marine mammals and aquatic freshwater biota (e.g. beluga whales, ringed seals and fish). High concentrations of SCCPs have also been measured in the breast milk of Inuit women in Northern Quebec.
There is evidence that SSCPs are toxic to sensitive aquatic organisms at relatively low concentrations – below thresholds for inherent toxicity (iT) used to categorize substances on Canada’s Domestic Substances List, as well as the OECD, and the USEPA. The most sensitive organism, *Daphnia magna*, has chronic NOECs of 5 µg/L.

Concentrations currently measured in the environment are generally below levels that have been associated with effects in laboratory studies. In some cases, concentrations are approaching those that may be of concern, for example in secondary consumers, and elevated levels have been measured in human breast milk, including in remote communities. Particularly in view of SSCP's persistence, bioaccumulation and their inherent toxicity to a range of organisms, it is considered that SSSCPs are likely to cause significant adverse effects as a result of long range transport.

4. CONCLUDING STATEMENT

In summary, the increasing regulation of SSSCPs have resulted in a decrease in SSSCPs currently in use. However evidence suggests that significant amounts are still in use and being released in several countries. The available empirical and modeled data strongly indicate that SSSCPs are persistent, bioaccumulative, and toxic to aquatic organisms at low concentrations. SSSCPs have characteristics similar to known POPs that undergo long range environmental transport. SSSCPs are considered as POPs pursuant to decisions taken under the UNECE POPs Protocol to the Convention on Long Range Transboundary Air Pollution (LRTAP). Concentrations in biota and sediment from remote Arctic locations also suggest long range transport of SSSCPs is occurring via air or ocean currents.

Based on the available evidence, it is thus likely that SSSCPs can, as result of long range environmental transport, cause significant adverse effects on human health and/or the environment, such that global action is warranted.
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C. OTHER POTENTIAL POPs (LISTS BY NGOs)

C1. LIST PROPOSED BY WWF

Stockholm Convention: “New POPs”
Screening Additional POPs Candidates
April 2005

Contact

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A-5. Hexachlorobutadiene (HCBD)
A-6. Dicofol (Kelthane)
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C-1. Octachlorostyrene (OCS)
C-2. Polycyclic aromatic hydrocarbons (PAHs)

3. CONCLUSION
1. Introduction

The Stockholm Convention targets 12 persistent organic pollutants (POPs) that threaten wildlife and people around the world. Placing these substances under global control is an important step in protecting the public and wildlife from harm. Envisioned by the international community to be a dynamic treaty, the Stockholm Convention provides a rigorous scientific process through which new chemicals with POPs characteristics can be added to the treaty. Several additional POP candidates await international attention; their expedited review, initially by the POP Review Committee (POPRC), will enable the Conference of Parties (COP) to address other dangerous chemicals sooner, rather than later.

Currently, approximately 80,000 chemicals (although the exact figure is unknown) are produced, marketed, used, and disposed of worldwide. Each year, hundreds of new chemicals are added to this ever-growing list. So far, very little is known about toxicity of these chemicals. Regulating these chemicals one-by-one will be a daunting and time-consuming effort. Over the longer term, relying solely on chemical-by-chemical regulation risks irreversible adverse effects to both human and wildlife health.

WWF applauds efforts of governments—at the national level, and through multilateral regimes—to address chemical groups such as polychlorinated biphenyls (PCBs) and dioxins (Stockholm Convention), or polycyclic aromatic hydrocarbons (PAHs) (United Nations Economic Commission for Europe /UNECE Protocol to the Convention on Long-Range Transboundary Air Pollution /LRTAP on POPs). The Stockholm COP and its POPRC should take the opportunity to seriously consider more chemical groups rather than individual chemicals for their inclusion as new POPs candidates. Substances and groups of chemicals that have similar chemical structures, properties, and/or potentials to harm, and those degrade to the Convention-regulated substances, all merit close scrutiny.

Screening Criteria for POPs Under the Stockholm Convention

The “Information Requirements and Screening Criteria” from the Stockholm Convention can be found in the Appendix. The criteria are summarized below:

**Persistence:** For the persistence requirement, the party nominating a chemical for listing in the Stockholm Convention should provide evidence that the half-life of the chemical in water is greater than two months, its half-life in soil is greater than six months, its half-life in sediment is greater than six months; or evidence that the chemical is otherwise sufficiently persistent to justify its consideration within the scope of the Convention.

**Bioaccumulation:** The bioaccumulation criteria requires evidence that the bioconcentration factor (BCF) or bioaccumulation factor in aquatic species for the chemical is greater than 5,000 or, in the absence of such data, that the log Kow is greater than 5; evidence that a chemical presents other reasons for concern, such as high bioaccumulation in other species, high toxicity, or ecotoxicity; or monitoring data in biota indicating that the bioaccumulation potential of the chemical is sufficient to justify its consideration within the scope of the Convention.

**Potential for long-range environmental transport:** To meet the long-range transport criteria, a nominating party must demonstrate measured levels of the chemical in locations distant from the sources of its release that are of potential concern; monitoring data showing that long-range environmental transport of the chemical, with the potential for transfer to a receiving environment, may have occurred via air, water, or migratory species; or environmental fate properties and/or model results that demonstrate that the chemical has a potential for long-range environmental transport through air, water, or migratory species, with the potential for transfer to a receiving environment in locations distant from the sources of its release. For a chemical that migrates significantly through the air, its half-life in air should be greater than two days.

**Toxicity (Adverse Effects):** The toxicity criteria requires evidence of adverse effects to human health or to the environment or toxicity or ecotoxicity data that indicate the potential for damage to human health or to the environment.

Identifying New POPs

Identification of chemicals likely to meet the POPs criteria is hindered by the fact that data on persistence, toxicity, bioaccumulation, and long-range transport are available for very few chemicals. WWF urges the chemical industry to develop and disseminate this important information.

The following 20 chemicals or groups of chemicals are potential POP candidates meriting further investigation for possible early inclusion in the Stockholm Convention. Among the 20 new POPs candidates, 18 are suggested for addition to Annex A (A-1 to A-18) and 2 are suggested for addition to
Annex C (C-1 and C-2), as shown in Box 1. The 18 chemicals suggested for addition to Annex A are clustered by their function and application. They are grouped as: 1) pesticides, insecticides, and other biocides; 2) brominated flame retardants; 3) perfluorochemicals; and 4) other chlorinated chemicals and chemical groups. Two chemicals suggested for addition to Annex C are unintentionally produced chemicals or chemical groups.

Four of the chemicals or chemical groups (A-1, 2, 9 and C-2) have been identified as POPs in the regionally-based LRTAP POPs Protocol. Nine chemicals or chemical groups (A-3-6, 10, 13, and 15-17) have been proposed for LRTAP listing after being evaluated by some European countries against screening criteria similar to that of the Stockholm Convention. Therefore, data are already available to suggest that these chemicals meet the UNEP POPs criteria. A-7 has been listed by the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR Convention) in its OSPAR List of Chemicals for Priority Action because available data show that it is persistent, toxic, and bio-accumulative (OSPAR 2004). The EU has proposed eight chemicals (A-1, 2, 5, 9, 10, 11, 15, 17) to be added to the Annexes of the Stockholm Convention (EU 2004, EC 2004). Another four chemicals (A-8, 14, 18, and C-1) are listed because of concerns regarding their toxicity and detection in environments remote from the source of release. These 20 chemicals or chemical groups represent a selection of highly problematic chemicals that possess POP-like properties.

### Box 1: New POP Candidates

<table>
<thead>
<tr>
<th>Category</th>
<th>Cluster</th>
<th>New POP Candidates</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-1</td>
<td>chlordecone</td>
<td>hexachlorocyclohexane (HCH) (includes lindane)</td>
</tr>
<tr>
<td>A-2</td>
<td>hexachlorocyclohexane (HCH) (includes lindane)</td>
<td></td>
</tr>
<tr>
<td>A-3</td>
<td>pentachlorophenol (PCP)</td>
<td></td>
</tr>
<tr>
<td>A-4</td>
<td>endosulfan</td>
<td></td>
</tr>
<tr>
<td>A-5</td>
<td>hexachlorobutadiene (HCBD)</td>
<td></td>
</tr>
<tr>
<td>A-6</td>
<td>dicofol</td>
<td></td>
</tr>
<tr>
<td>A-7</td>
<td>methoxychlor</td>
<td></td>
</tr>
<tr>
<td>A-8</td>
<td>hexabromocyclododecane (HBCD)</td>
<td></td>
</tr>
<tr>
<td>A-9</td>
<td>hexabromobiphenyl (Hexa-BB)</td>
<td></td>
</tr>
<tr>
<td>A-10</td>
<td>pentabrominated diphenyl ether (penta-BDE)</td>
<td></td>
</tr>
<tr>
<td>A-11</td>
<td>octabrominated diphenyl ether (octa-BDE)</td>
<td></td>
</tr>
<tr>
<td>A-12</td>
<td>decabrominated diphenyl ether (deca-BDE)</td>
<td></td>
</tr>
<tr>
<td>A-13</td>
<td>perfluoro-chemicals</td>
<td>perfluorooctyl sulfonate (PFOS)</td>
</tr>
<tr>
<td>A-14</td>
<td>perfluorooctanoic acid and its salts (PFOA)</td>
<td></td>
</tr>
<tr>
<td>A-15</td>
<td>pentachlorobenzene (penta-CB)</td>
<td></td>
</tr>
<tr>
<td>A-16</td>
<td>short-chained chlorinated paraffins (SCCPs)</td>
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</tr>
<tr>
<td>A-17</td>
<td>polychlorinated naphthalenes (PCNs)</td>
<td></td>
</tr>
<tr>
<td>A-18</td>
<td>tetrachlorobenzene (tetra-CB)</td>
<td></td>
</tr>
<tr>
<td>C-1</td>
<td>ochtachlorostyrene (OCS)</td>
<td></td>
</tr>
<tr>
<td>C-2</td>
<td>polycyclic aromatic hydrocarbons (PAHs)</td>
<td></td>
</tr>
</tbody>
</table>

Four of the chemicals or chemical groups (A-1, 2, 9 and C-2) have been identified as POPs in the regionally-based LRTAP POPs Protocol. Nine chemicals or chemical groups (A-3-6, 10, 13, and 15-17) have been proposed for LRTAP listing after being evaluated by some European countries against screening criteria similar to that of the Stockholm Convention. Therefore, data are already available to suggest that these chemicals meet the UNEP POPs criteria. A-7 has been listed by the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR Convention) in its OSPAR List of Chemicals for Priority Action because available data show that it is persistent, toxic, and bio-accumulative (OSPAR 2004). The EU has proposed eight chemicals (A-1, 2, 5, 9, 10, 11, 15, 17) to be added to the Annexes of the Stockholm Convention (EU 2004, EC 2004). Another four chemicals (A-8, 14, 18, and C-1) are listed because of concerns regarding their toxicity and detection in environments remote from the source of release. These 20 chemicals or chemical groups represent a selection of highly problematic chemicals that possess POP-like properties.

### 2. New POPs Candidates

**A-1–7. Pesticides, Insecticides, etc.**

These seven chemicals are commonly used as pesticides, insecticides, fungicides, fumigant, and biocides. Their ability to kill unwanted organisms also has the potential to harm wildlife and people.
A-1. Chlordecone
The insecticide chlordecone (Kepone) has already been banned in many countries. Its placement on the LRTAP POPs Protocol establishes its conformance with the UNEP POPs criteria, as these two international treaties have common criteria. Chlordecone is highly toxic to aquatic algae and invertebrate species and produces a range of health effects in mammals. These include reproductive impairment, liver damage, and neurological symptoms. It is also known to cause cancer in animals.

A-2. Hexachlorocyclohexane (HCH)
Also included on the LRTAP POPs Protocol, HCH is the predominant organochlorine chemical found in Arctic air and in the Arctic Ocean (AMAP 1997). It has also accumulated in humans and wildlife living in the Arctic. One form of HCH is lindane, which is neurotoxic and can adversely affect reproduction, the liver, and the immune system. Both “technical grade” HCH and lindane are used in wood treatments and as insecticides, and they are also used in the home and in forestry. The International Agency for Research on Cancer (IARC) has classified HCH isomers as possibly carcinogenic to humans.

A-3. Pentachlorophenol (PCP)
Used as a biocide and wood preservative, PCP was the subject of an International Declaration signed in 1998 by several countries party to the LRTAP POPs Protocol, including all EU countries, Iceland, Norway, and Switzerland. The Declaration states that these countries “maintain their belief in the need for pentachlorophenol use to be tightly controlled to minimize emissions to the environment and urge other Parties to the Convention on Long-Range Transboundary Air Pollution to adopt controls similar in effects to those in place in the European Community if they have not already done so” (ECE/EB.AIR/57 1998).

Toxicity: PCP is highly toxic and there is some evidence that it causes cancer in humans. The IARC has classified it as a possible carcinogen in humans, while the US Environmental Protection Agency (US EPA) considers it to be a probable human carcinogen. It can also affect the immune and endocrine systems (ATSDR 1999). Disruption of thyroid hormone transport is a particular concern because PCP can bind to one of the thyroid hormone transport proteins, transthyretin, with twice the affinity of thyroxin (Sandau et al. 2000a). Free thyroxine levels in Nunavik placental cord plasma were negatively correlated with the sum of PCP and PCB hydroxylated compounds, suggesting that these chemicals are altering thyroid hormone status in newborns which could lead to neurodevelopmental effects (Sandau et al. 2002). The degradation products of PCP also raise concerns (AEA 1994). Pentachloroanisole, for example, has been found as a contaminant of Arctic air (Macdonald et al. 2000) and biota (Muir et al. 2001).

Persistence: PCP may not meet the UNEP POPs criterion for persistence, but it may be more recalcitrant to degradation under certain conditions, such as low sunlight, or when adsorbed on acidic soils (ATSDR 1999).

Bioaccumulation: PCP does not typically have high values for bioaccumulation but the criterion is met in some aquatic organisms (ATSDR 1999). PCP has also been reported to accumulate in human adipose tissue, particularly in testicular tissue (Wagner et al. 1991), although other sources state that the highest concentrations are found in the liver, kidney, and brain and that the potential for bioaccumulation is low (UNECE 2002a). Hydroxylated PCP has been detected in umbilical cord plasma in women from Quebec, including the Inuit from Nunavik, and was found at levels of 638-7680 pg/g wet weight (Sandau et al. 2002).

Potential for long-range environmental transport: Despite ongoing debate about PCP’s ability to undergo long-range transport, PCP has been measured in the Arctic. Its half-life in air is estimated to be less than 2 days, but significant long-range transport might nonetheless occur when PCP is adsorbed onto particulates, as degradation in this form is likely to take occur more slowly. PCP has been found in the blood of polar bears and Inuit people (Sandau et al. 2000a).

A-4. Endosulfan
The insecticide endosulfan is structurally similar to chlordane and dieldrin. It is used on a wide variety of food crops including tea, coffee, fruits, vegetables, rice, cereals, maize, sorghum, and other grains. It is also widely used on cotton, and there is more limited application as a wood preservative and to control termites and tsetse fly. Use of endosulfan has declined in Europe, although elevated levels have been noted in certain foods such as peppers sold in the EU (EC 2001). In 1984, the World Health
Organization estimated worldwide production at 10,000 metric tons. In the United States, it is no longer produced, but use continues, mainly on tobacco and fruit crops (ATSDR 2000). Technical-grade endosulfan contains at least 94% of two pure isomers, alpha and beta endosulfan, which are present in a ratio of 7:3.

**Toxicity:** Endosulfan has neurotoxic effects, as well as effects on the kidneys, testes, and liver. Endosulfan may lower the ability to fight infection. Experiments suggest that immune responses can be altered at dose levels that do not induce other overt signs of toxicity (ATSDR 2000). There are also concerns about human health in areas where spraying is conducted (Quijano 2002).

Endosulfan can act as an endocrine disruptor (BKH report 2000). Organisms treated with endosulfan exhibit toxic effects, such as developmental and reproductive changes that have been associated with endocrine disrupting chemicals (US EPA 2002a). In fish, effects on the onset of breeding behavior in juvenile male cichlid have been noted at 0.6 μg/l (Matthiessen and Logan 1984). Levels of 5μg/l have been shown to interfere with reproduction in the red-spotted newt, Notophthalmus viridescens, by disrupting the development of female glands that synthesize a pheromone used in female-male communication (Park et al. 2001). Other effects linked to its endocrine disrupting properties include: impaired development in amphibians; reduced cortisol secretion in fish; impaired development of the genital tract in birds; and altered hormone levels, testicular atrophy, and reduced sperm production in mammals (Herrmann 2002).

On the basis of standard tests, endosulfan is reported to be highly toxic for aquatic organisms, with, for example, a 96 hour LC50 value for the marine crustacean Penaeus duorarum of 0.04μg/l (Herrmann 2002). In the United States, a national database shows that during the 1980s, endosulfan was responsible for more fish kills in estuaries and coastal rivers than all other currently used pesticides (US EPA 2002a).

**Persistence:** Although endosulfan does not appear to meet the numerical UNEP POPs criterion for persistence in water, the criterion may be met in slightly acidic waters under anaerobic conditions. Similarly, the alpha and beta isomers both persist longer under more acidic soil conditions, with beta-endosulfan having a longer half-life as it is slowly converted to alpha-endosulfan. In many studies of soil and sediments it may not meet the criterion, although in sandy soils, the beta isomer can have a half-life greater than 6 months. Also, when considering degradation times in soils, if the persistent and toxic metabolite endosulfan sulfate is included, then the half-life of “total endosulfan” in soil exceeds 6 months in some cases (Herrmann 2002). Endosulfan could also arguably meet the non-numerical criterion, namely that it is sufficiently persistent to justify consideration within the scope of the Convention.

**Bioaccumulation:** Reported log Kow values suggest a high potential for bioconcentration, which is confirmed by laboratory data. For example, a BCF of 2,650 was reported for zebra fish exposed to 0.3μg/l for 21 days in a flow through system (ATSDR 2000), and one study has reported a BCF of 11,000 (Herrmann 2002). Also, endosulfan has been found in fish, crab, and mollusk samples, as well as in milk and vegetables. It is metabolized in mammals, so there is only limited biomagnification up the food chain, but endosulfan sulfate has been found in marine mammals (Muir 2001). Given reported BCFs of over 5000, and the presence of endosulfan and/or its metabolite in various biota, it could be judged to meet the bioaccumulation criterion (Swedish EPA 2002).

**Potential for long-range environmental transport:** The half-lives in air of alpha-endosulfan, beta-endosulfan, and endosulfan sulphate are respectively estimated at >2.7 days, >15 days, and >2.7 days (Herrmann 2002). Long-range transport is confirmed by monitoring data in the Arctic and elsewhere. For example, endosulfan has been identified in the Arctic atmosphere and in the Bering and Chukchi seas (Macdonald et al. 2000). It has also been found in Arctic biota, including ringed seals, beluga whales, and fish. Derek Muir of the Canadian National Water Research Institute in Ontario has also detected the metabolite, endosulfan sulfate, in fish and beluga whales from the Arctic (pers. comm.).

**A-5. Hexachlorobutadiene (HCBD)**
Russia has been reported to be one of the major users of HCBD, where it is applied as a fumigant on grape crops. HCBD has also been used to combat soil pests and is, or has been, used as a fumigant in France, Italy, Greece, Spain, and Argentina. In the United States, the largest use was for the recovery of “snift,” a chlorine-containing gas used in chlorine plants, but this use was discontinued. However, as of
the early 1990s it was still imported for use as a chemical intermediate in the manufacture of rubber compounds and, to a lesser extent, as a solvent, laboratory reagent, heat-transfer liquid, hydraulic liquid, fluid for gyroscopes, and chemical intermediate in the manufacture of chlorofluorocarbons and lubricants (ATSDR 1994). In the EU in 1988, annual production was estimated at around 2000 to 4000 metric tons (NRA1995), but it is no longer produced in Western Europe or the United States (industry submission to EU PBT Group).

HCBD can also be inadvertently released during the production of several other chlorinated chemicals, such as vinyl chloride, allyl chloride, epichlorohydrin, trichloroethylene, tetrachloroethylene, and carbon tetrachloride. In addition, HCBD can be released from incinerators, motor vehicle emissions, the manufacture of plastics (EC&HC 2000), and from waste dumps (UK COT 2000).

Toxicity: Based on results of animal laboratory studies, the kidney, and to a lesser extent the liver, appear to be the target organs of HCBD-induced toxicity. The US EPA has determined that HCBD is a possible human carcinogen. No studies were found on the effects of HCBD on the function of the human immune system and studies in animals seem to be limited to those showing lesions in lymphoid tissue, which occur at lethal doses (ATSDR 1994). With regard to environmental effects, HCBD is very toxic to aquatic organisms, with a “no observed effect concentration” (NOEC) for fish reported at 6.5µg/l (Swedish EPA 2002).

Persistence: HCBD biodegrades slowly in natural waters, with an estimated half-life of between 4 and 52 weeks, depending on the amount of organic matter (EC&HC 2000).

Bioaccumulation: HCBD accumulates in the tissues of freshwater organisms and BCFs of over 5000 have been reported in fish. It tends to be preferentially accumulated in the liver of the fish and a BCF of 10,000 has been reported for liver in dab (Limanda limanda). Once in the liver, polar metabolites can be formed and these may reach the kidneys to become nephrotoxic in fish. However, as HCBD is quite easily metabolized, it does not biomagnify through food chains (EC&HC 2000).

In a study reported in 1975, HCBD was found in the human liver at levels of 5.7-13.7µg/kg wet wt, and in adipose tissue at levels of 0.8-8µg/kg wet wt (ATSDR 1994).

Potential for long-range environmental transport: Research has suggested that HCBD can undergo long-range transport, with half-lives estimated at between 60 and 840 days. Monitoring data confirms this, and evidence for long-range transport of HCBD includes its detection in samples taken from various sediment depths in Canada’s Great Slave Lake (EC&HC 2000). HCBD is also routinely found at low levels in Arctic biota, e.g., ringed seal blubber and Arctic char muscle, with levels averaging 0.07 ng/g in blubber of male and female ringed seal from the Hudson Strait area of northern Quebec. Levels are higher in juvenile seals aged less than one year, and have been found at approximately 0.11 ng/g, presumably due to transfer in mothers’ milk (D. Muir, pers. comm.).

A-6. Dicofol (Kelthane)

Dicofol is an organochlorine pesticide which is manufactured from DDT and DDE, although modern manufacturing processes can produce technical grade dicofol which contains less than 0.1% of DDT and related substances. It is used as an acaricide and mite-control agent on a wide variety of fruit, vegetables, and ornamental and field crops. In the EU, it is or was made in Spain and Italy (according to the database of the ECB) (Rasenberg 2003).

Toxicity: Dicofol has a high acute toxicity in the aquatic environment, with a LC50 of 15µg/l for eastern oyster and of 120µg/l for rainbow trout. In birds, it is a reprotoxic chemical which can affect eggshell quality, and in falcons, feminized male embryos have been found from females given 5mg/kg. It is also a suspected endocrine disrupting chemical. In mice, an increase in liver tumors has been reported, although in rats carcinogenicity was not found. IARC has classified it in category 3 (not classifiable as a human carcinogen) (Rasenberg 2003).

Persistence: The p,p-isomer which makes up about 80% of the formulation is more persistent than the o,o-isomer, which makes up approximately the other 20%. The p,p-isomer has a half-life in water greater than 2 months only in acid conditions, and in soil the half-life is considered to be shorter than the 6 months criterion. In sediments the primary degradation is rapid, but one of the degradation products, dichlorobenzylhydrol (DCBH), is reported to have a half-life of 197-429 days. Given that many lakes are
already acidified, for example in Sweden and Norway, this chemical should be considered to meet the persistence criterion (Rasenberg 2003).

**Bioaccumulation:** Dicofol has a log Kow of around 5, and BCFs in fish are reported to range from 8,050 for the fathead minnow (Pimephales promelas) to 13,500 (Rasenberg 2003).

**Potential for long-range environmental transport:** Based on a low vapor pressure (less than 5.3 x 10^-5Pa) and a calculated atmospheric half-life of greater than 2 days, dicofol is considered to meet the criterion for long-range transport. However, data on levels in remote environments are sparse (Shaver 2003).

A-7. Methoxychlor

Methoxychlor has structural similarities with DDT, and has been used as an insecticide, a biocide, and a veterinary product. It was listed in the OSPAR List of Chemicals for Priority Action in 2000 (OSPAR Commission 2004).

**Toxicity:** Methoxychlor is extremely toxic to aquatic organisms, with an acute LC50 for rainbow trout of 52μg/l. The degradation products of methoxychlor are suspected endocrine disruptors (ATSDR 1994).

**Persistence:** Methoxychlor appears more persistent in aerobic conditions, but there is some uncertainty whether the persistence criterion is met. It has been reported that 6 months after the application of methoxychlor to the soil, 42% remained (ATSDR draft 2001). Therefore, it may be sufficiently persistent to merit inclusion. The half-life represents the calculated time for the loss of the first 50% of the substance, but the time required for the loss of the half that remains may be substantially longer.

**Bioaccumulation:** Methoxychlor has a log Kow of 4.7-5.08 and a reported BCF of 8,300 for fathead minnow (ATSDR draft 2001). In other fish species, however, quite different values have been reported, perhaps due to differences in metabolism and excretion. It has also been detected in the blubber and liver of harp seals (ATSDR draft 2001). Therefore, it can be considered to meet the criterion for bioaccumulation.

**Potential for long-range environmental transport:** Although its half-life in air is reported to be far less than the requisite 2 days, it has been detected in rain and snow in remote areas in Canada. Therefore, it appears that this substance may undergo long-range transport (ATSDR draft 2001). It has also been reported to be found in the Arctic (AMAP 1997).

A-8–12. Brominated Flame Retardants

The following four POP candidates belong to a family of commercially produced flame retardants known as brominated flame retardants (BFRs). Research has shown that certain BFRs exist in the environment far from the locations where they are produced and/or used and that the concentrations of some of the BFRs in both wildlife and humans in certain locations are increasing (Alaee and Wenning 2002).

A-8. Hexabromocyclododecane (HBCD)

HBCD is an additive flame retardant used mainly in polystyrene and in formulations for textiles. It may be found in a variety of articles including: upholstered furniture; automobile textiles and seat materials; interior textiles; insulation blocks in building and in transport vehicles; cable and textile coating; and electrical and electronic equipment (e.g., distribution boxes for electrical lines, video cassette recorder housings).

Production has increased dramatically since it was first marketed in the 1960s, and occurs in the EU, the United States, Japan, and probably elsewhere.

Trout and eels have been found contaminated with HBCD at levels of up to 6,758 and 9,432μg/kg, respectively (UK COT 2003). In addition to discharges from sites producing and processing HBCD,
significant amounts of HBCD are likely to be released from articles in use, or when HBCD articles are put in landfills, where releases may prevail for a very long time, perhaps hundreds of years. Uncontrolled spreading of HBCD in the environment may also arise from products produced by using recycled expandable polystyrene. When HBCD is incinerated improperly, polybrominated dibenzofurans and dioxins may be released.

**Toxicity:** Daphnids exposed to 5.6 µg/l for 21 days had statistically significant reduced mean lengths. The lowest observed effect concentration (LOEC) was determined to be 5.6 µg/l. However, no statistical effects on survival, reproduction, or growth were observed in Daphnia magna exposed for 21 days to 3.1 µg/l.

In mammals at relatively high doses (100mg per kg/day for 90 days in rodents), effects on liver weight and on the thyroid hormone system (T3 and TSH) have been noted. A recent study on behavioral effects has suggested that HBCD may cause developmental neurotoxic effects, including significant changes in spontaneous behavior, learning and memory defects, and a reduced number of nicotine receptors. (Eriksson et al. 2002). An indicative lowest observed adverse effect level (LOAEL) of 0.9 mg/kg/day can be deduced from this latter study. Data are lacking with regard to the potential carcinogenicity of HBCD.

**Persistence:** The levels found in biota and sediment indicate a high degree of persistence and laboratory tests show that HBCD is not readily biodegradable. There are also concerns about the persistence of the breakdown product, cyclooctadecatriene (EU RAR 2004).

**Bioaccumulation:** HBCD is considered to have a log Kow value of 5.625 and in fathead minnow a BCF of 18,100 has been reported. HBCD has been found in peregrine falcon eggs, and it has also been found in herring and guillemot in the Baltic, where the levels found show that biomagnification is occurring. Guillemot eggs from the Baltic, sampled between 1969 and 2001, showed an increase in HBCD levels over this time, although peak levels of HBCD were noted in the middle of the 1970s (Sellstrom et al. 2003).

HBCD has been detected in the breast milk of Swedish women at a maximum of 2.4 ng/g fat. HBCD has also been found in the breast milk of women from Mexico and it has been recorded in the serum of mothers from The Netherlands. HBCD was found in food samples, including the fat of pork, lamb, chicken, and beef, as well as fish, cows’ milk, and eggs (EU RAR 2004).

**Potential for long-range environmental transport:** HBCD has been found in air samples in the northernmost area of Scandinavia, supporting the assumption that long-range air transport can occur (for all data see EU draft RAR 2002).

A-9. Hexabromobiphenyl (Hexa-BB)

The flame retardant hexa-BB is also included on the LRTAP POPs Protocol and therefore meets the criteria of a UNEP POP. Hexa-BB is banned in Europe and production of Firemaster flame-retardant products (containing primarily hexa- and hepta-bromobiphenyl) ceased in the United States following a notorious incident in Michigan in 1973 when it was accidentally mixed into animal feed, causing widespread contamination of milk, meat, and eggs. It has also been found in wildlife, including seals from the Baltic and Svalbard. The effects of exposure to hexa-BB are similar to those from PCBs.

Although there is no known production of hexa-BB, inclusion in the Stockholm Convention would prevent any new production. Other polybrominated biphenyls (PBBs) include the commercial products octa- and deca-BB. These products are all mixtures to some extent. Production of octa-BB is considered to be very low. Deca-BB, because of its large size, is considered to have a low bioaccumulation potential, but this needs to be verified. Deca-BB was produced in Britain and is, or has recently been produced, by Atochem in France (Passivirta 2000). Concern about deca-BB may focus on its potential debromination to hexa-BB (KEMI 1999).
A-10. Pentabromodiphenyl Ether (Penta-BDE)

Commercial penta-BDE is used as a flame retardant in a mixture that contains roughly 24-38% tetrabrominated diphenyl ether and 50-62% penta-brominated diphenyl ether. Used mainly in polyurethane foam filled furniture and car interiors, penta-BDE was also used in cot mattresses in the EU and in the production of packaging. Some printed circuit boards produced outside the EU, including perhaps some produced in Asia, may also contain penta-BDE. In the United States it is used in re-bond, a type of carpet underlay (EU RAR 2000, RPA 2000).

Toxicity: Experiments have shown that penta-BDE can alter thyroid hormone levels in mammals and can affect learning ability in mice. It may also adversely affect the immune system (Fowles et al. 1994, Darnarud and Sinjari 1996, Eriksson et al. 1998, Darnarud and Thuvander 1998). Penta-BDE is now found as a widespread contaminant in wildlife. The possibility of subtle impacts on brain development, as well as the potential interactive effects with other endocrine disrupting chemicals, should be sufficient to merit global control over the use of this substance. The EU has already agreed to take action, largely due to concern about the rising levels in breast milk. In Sweden, in the 25 years from 1972-1997, the concentration of polybrominated diphenyl ethers in mothers’ milk doubled every 5 years, with levels peaking in the late 1990s (Meironyte et al. 1998). However, much higher levels have since been found in UK breast milk (Betts 2003).

Persistence: Penta-BDE is very persistent and is not inherently biodegradable in the OECD Test (EU RAR 2000).

Bioaccumulation: Penta-BDE meets the UNEP POPs criterion for bioaccumulation. A log Kow of 6.57 and measured BCFs of well over 5000 have been reported (EU RAR 2000). In the Canadian Arctic between 1981 and 2000 there has been an exponential increase in the levels of this substance in biota (Ikonomou et al. 2002).
Potential for long-range environmental transport: Penta-BDE is a widespread contaminant in wildlife (EU RAR 2000) and has been found in many aquatic species including beluga whales in the Arctic (Stern and Ikonomou et al. 2000). It has also been measured in Arctic air (Muir et al. 1999), indicating that it can undergo long-range transport.

A-11. Octabrominated diphenyl ether (Octa-BDE)
The commercial octa-BDE includes some hepta- and hexa-BDE, and it is the hexa which is more bioavailable. Octa-BDE is used as an additive in polymers for use in plastic housings for office equipment and business machines, but since August 2004 it has been banned in the EU (EU 2003).

Toxicity: In the EU, octa-BDE was classified as toxic for reproduction and a study by Viberg et al. (2001) suggests that hexa-BED, one component of commercial octa-BDE, is a developmental neurotoxicant in mice (CSTEE 2002). Therefore, octa-BDE may also represent a possible risk of harm to the human fetus (Danish EPA 2000). Repeated doses of octa-BDE induced liver changes, indicative of an inducer effect.

Bioaccumulation: Octa has a reported log Kow of 6.9 (OSPAR 2002a). Furthermore, octa-BDE was detected (70-8,000 pg/g) in human adipose tissue selected from the US National Human Adipose Tissue Survey (FY 87 NHATS) Repository (Stanley et al. 1991). In addition, hexa-BDE a component of octa-BDE, has been measured in breast milk in several studies (EU RAR 2003). Bioaccumulation therefore seems to occur.

Potential for long-range environmental transport: Octa-BDE has a very low vapor pressure (6.59x10-6 Pa) (OSPAR 2002a). Combined with its persistence, octa-BDE could have the potential to undergo long-range transport.

A-12. Decabrominated diphenyl ether (Deca-BDE)
Deca-BDE is used as an additive flame retardant primarily in electrical and electronic equipment, as well as in textiles, where it is applied as a polymer backcoat to the fabric. Globally, deca-BDE has become the most used polybrominated diphenyl ether product, with production mostly concentrated in the Americas and Asia (BSEF website).

Emissions arise from production sites and from losses of the substance from flame-retarded products in use and after disposal. Such diffuse sources clearly occur and will be difficult to control without the phase-out of its use. For example, deca has been detected in sediments associated with effluents from waste dumps. It is also known to be present in indoor air and has been detected in household dust. Moreover, a number of studies show that deca-BDE is present in sewage sludge from waste water treatment plants (EU draft RAR R013_0405_env 2004), and it has been found in high concentrations at mg/kg level (dry weight) in North American sewage sludge (Environment Canada 2004). Deca-BDE has also been found in marine sediments and in a study around the Scheldt basin deca has been reported by de Boer et al. (2002) at levels of up to 700 μg/kg dry weight (EU draft RAR R013_0405_env 2004).

The combination of evidence of debromination with high accumulation of deca-BDE in the environment has led researchers to note that even slight and very long term degradation to lower brominated diphenyl ethers could have serious ecological consequences over periods spanning several decades. The overall persistence of deca and potential transformation to lower PBDEs that are persistent, toxic and bioaccumulative, and observed commercial and environmental trends, indicate environmental concerns (Environment Canada 2004).

However, the concern does not relate just to the potential debromination in the environment, but also to deca itself, because a study in mice has reported it to be a developmental neurotoxicant (Viberg et al. 2003). Indeed, in view of the concerns raised regarding the potential developmental neurotoxicity of deca-BDE, in 2005 the European Commission was proposing further testing for such effects.

Toxicity: Deca-BDE is reported to have low general toxicity comparing to penta- and octa- BDE. However, the concern is mostly focused on its potential developmental neurotoxicity and the toxicity of
its lower brominated breakdown products. In addition, the International Agency for Research and Cancer (1990) has concluded it to be a Group 3 carcinogen, with limited evidence for the carcinogenicity in experimental animal and not classifiable as to its carcinogenicity to humans (EU draft RAR R013_0402_hh 2002). Thus, a 2 year deca-BDE exposure study in rats showed increased incidence of neoplastic nodules in the liver and in a 2 year deca-BDE feed study in mice, thyroid gland follicular cell hyperplasia was increased (Danish EPA 2000).

**Persistence:** Deca-BDE in natural sediments has been shown to be stable and resistant to biodegradation under anaerobic conditions for up to 2 years (de Wit 2000).

**Bioaccumulation:** Deca has a log Kow value of 6, but was formerly erroneously considered to be too large a molecule to cross biological membranes. However, deca has now been detected in human blood and breast milk and in many wildlife species.

For example, studies in Japan (Hori et al. 2002) and in Texas (Schecter et al. 2003) and in other USA states (Lunder and Sharp 2003) show that deca is a frequent contaminant of human breast milk, being found in approximately 30% of the Texan samples and in 80% of the samples in the Lunder and Sharpe study. Deca has also been detected in blood serum of workers, including hospital cleaners, clerks working full-time at computer screens, personnel at an electronics dismantling plant, and rubber workers. The highest blood serum deca-BDE concentrations were in rubber wire producers (maximum 278 µg/kg fat). Deca has also been detected in blood serum samples from the general public, including Members of the European Parliament (MEPs), in 2 biomonitoring studies done by WWF at a median concentration of 83 µg/kg fat and 53 µg/kg fat, in those with positive results. A maximum concentration of 2400 µg/kg fat was found in the blood of one male participant, who testified that he had no known occupational chemical exposure, other than the use of a computer in a small office (EU draft RAR, 2004 and 2005 addendum, R013_0502hh and pers. comm. to Lyons 09-03-05). Similarly, wildlife species, including fish, polar bears, and the eggs of predator birds have been found to be contaminated with deca-BDE and the extent of this contamination in birds of prey has become more widespread in recent years (EU draft RAR R013_0502hh 2004 and 2005 addendum).

Therefore, despite the absence of a bioconcentration factor in fish of greater than 5000, there is monitoring data in biota indicating that the bio-accumulation potential of the chemical is sufficient to justify its consideration within the scope of the Stockholm Convention.

**Potential for long-range environmental transport:** A study by Muir and coworkers (2003) has detected deca-BDE in Arctic sediments. Deca-BDE was likely transported on particles to remote places such as Canadian Arctic due to its low vapor pressure (4.63x10^-6 Pa) and high log Kow (OSPAR 2002b). The data appear to show an increasing trend in the concentration found in the samples taken, but with generally lower concentrations and a later date of first occurrence in the more northerly samples, which are considered to be consistent with transport to remote regions mainly on particulates. Detection of this substance in moss in relatively remote regions of Norway has also been attributed to long-range particulate transport and the substance has also been found in some birds in polar regions (EU draft RAR R013_0405_env 2004).

A-13, 14. Perfluorochemicals (PFCs)

Perfluorochemicals (PFCs) are non-polar, highly fluorinated compounds that are chemically and biologically inert. They are used in surface treatments to provide soil and stain resistant coatings, in paper treatments to provide oil, grease, and water resistance, and as performance chemicals in fire fighting foams, cosmetics, and cleaners. Some of PFCs including perfluorooctyl sulfonate (PFOS) and perfluorooctanoic acid and its salts (PFOA) have proven to be persistent global pollutants.

A-13. Perfluorooctanyl sulfonate (PFOS)

(Note: Perfluorooctyl sulfonyl fluoride (POSF) and other chemicals that can degrade to Perfluorooctanyl sulfonate (PFOS) are included in this section.)

PFOS is a member of a large family of chemicals based on perfluorooctyl sulfonyl fluoride (POSF). POSF-based chemicals are used, or have been recently used, in many industrial and consumer
applications including stain resistant coatings for fabrics, leather, furniture, and carpets; fire fighting foams; cleaning products; and commercial and consumer floor polishes. Exposure may also result from perfluoro chemicals used in food wraps, particularly those used for moist or greasy fast food products. Perfluorooctyl sulfonic acid and its salts are also used as surfactants in a variety of performance applications. Some salts of PFOS chemicals are also used as pesticides. Countries that produce PFOS include, or have included, the United States, Belgium, Japan, Italy, and possibly the Russian Federation, China, and Germany. The Minnesota Mining and Manufacturing Corporation (3M), once a dominant PFOS producer, has committed itself to the “phasing out of the perfluorooctanyl chemistry used to produce certain repellents and surfactant products,” (3M 2000). However, other chemical companies have been linked with chemicals of the “perfluoro” group, all of which need to be scrutinized with regard to their POPs characteristics.

**Toxicity:** PFOS is highly toxic to honey bees. Studies in rats and monkeys also raise serious concerns. Adverse signs of toxicity include increases in liver enzymes and other effects on the liver, gastrointestinal effects, blood abnormalities, decreased serum cholesterol, and death. In a multigenerational study in rats, perinatal mortality was increased at PFOS doses of 0.8mg/kg/day and higher. Also, in subsequent tests on rats, at all the dose levels tested (0.4mg/kg/day and above) PFOS exposure caused reduced levels of the thyroid hormones T3 and T4. However, it is not yet clear whether this finding was connected to the underlying mechanism responsible for the perinatal mortality (US EPA 2001).

With regard to human health there is an association between exposure to PFOS and bladder cancer. Furthermore, there appears to be an increased risk of episodes of neoplasms of the male reproductive system, the overall category of cancers and benign growths, and neoplasms of the gastro intestinal tract (OECD 2002)

**Persistence:** PFOS and its salts are highly persistent in the environment and do not appear to degrade. Other substances based on POSF chemistry have the potential to degrade back to PFOS, although research is underway to try to improve the understanding of the breakdown of large molecules based on POSF chemistry. It is clear that substances that break down to PFOS could be judged to meet the UNEP POPs criterion for persistence.

**Bioaccumulation:** PFOS has an estimated half-life of 4 years in humans. Biological monitoring highlights that the bioaccumulation potential of this chemical is sufficient to justify its consideration within the scope of this Convention. Fish-eating birds appear to bioaccumulate PFOS to a considerable extent. PFOS has also been found in human blood samples, with concentrations in the parts per million range for exposed workers. In the general population, the levels in adults have been found to be in the parts per billion range (mean 30-44 ppb). Preliminary data suggest that children may have higher levels than adults; mean levels of 54 ppb were found in a small sample. Unlike many other bioaccumulating chemicals, PFOS does not accumulate in body fat but is found instead in the blood.

**Potential for long-range environmental transport:** Long-range transport is taking place. PFOS has been found at the parts per billion level in a wide variety of wildlife species around the globe, including seals from the Arctic and albatross from the Midway Atoll in the Pacific (Geisy and Kannan 2001).

A-14. Perfluorooctanoic acid and its salts (PFOA)
Perfluorooctanoic acid and its salts (PFOA) are another group of perfluorochemicals (PFCs). PFOA are fully fluorinated organic compounds that can be produced synthetically or through the degradation or metabolism of other PFCs. Perfluorooctanoic acid is primarily used as a reactive intermediate, while its salts are used as processing aids in the production of fluoropolymers such as polytetrafluoroethylene, a compound more commonly known as Teflon. Products made by using PFOA have been used in almost all industrial sectors, such as the automotive, defense, telecommunications, packaging, and textile industries. PFOA are not major ingredients in these commercial products. Instead, these products typically contain fluorothermals that may degrade into PFOA. In recent years, about 600 metric tons of PFOA are manufactured per year in the United State, but other countries may also produce PFOA.

**Toxicity:** Toxicological studies have shown that PFOA could be carcinogenic, immunotoxic, and endocrine disruptive. For example, PFOA have been found to be carcinogenic and immunotoxic in rodents (US EPA 2002b). Also, PFOA exposure causes changes in body, liver, and kidney weight in animals (US EPA 2003) and causes Leydig cell (the cells that produce androgens) tumors in rats. PFOA
directly modify the steroidogenic function of Leydig cells in vitro (Liu et al. 1996). Serum PFOA may change T3 hormone levels and increase cholesterol levels in humans (US EPA 2003).

**Persistence:** PFOA are persistent because of their long carbon chains saturated with fluorine atoms which are very resistant to physical, chemical, or biological attack. The very properties of PFOA that make them useful also make them problematic. There is no evidence that they ever fully degrade.

**Bioaccumulation:** PFOA have been detected in wildlife such as polar bears and fish in remote areas in the Canadian Arctic (US EPA 2002b, Ritter 2004, Kannan et al. 2002), and in both occupationally exposed workers and the general US population. PFOA distribute predominantly to the liver, plasma, and kidney and to a lesser extent the lungs, testis, and ovary (US EPA 2002b, Kudo and Kawashima 2003). The arithmetic mean PFOA levels in the serum of occupationally exposed workers ranged from 0.84 to 6.8 ppm, with the highest level at 81.3 ppm. Mean serum PFOA levels in general population range from 3 to 17 parts per billion (ppb), with the highest level in children at 56.1 ppb (US EPA 2003).

The biological half-life of PFOA in humans is 4.37 years (US EPA 2002b) and repeated exposure may result in accumulation of PFOA in humans and wildlife. The BCF of one of the PFOA salts (ammonium perfluorooctanoate) has been reported to be 56 (Giesy and Kannan 2002). Nevertheless, the monitoring data in biota indicate that the bioaccumulation potential of the chemical is sufficient to justify its inclusion in the Stockholm Convention.

**Potential for long-range environmental transport:** PFOA, a type of carboxylic acids, are often converted from other PFCs such as polyfluorinated alcohols that are a group of compounds known as telomers. Scientists from North America have found that fluorotelomer alcohols include CF3(CF2)nCF2CH2CH2OH, where n=2, 4, or 6. These fluorotelomer alcohols can degrade to carboxylic acids such as PFOA (Ritter 2004). Studies of C10 (i.e., when n=6) fluorotelomer alcohol suggest that C10 fluorotelomer alcohol may stay in the air for as long as 20 days (Ellis et al. 2003). This is long enough for these potential PFOA predecessors to be transported to remote areas.

A 15–18. Other Chlorinated Chemicals and Chemical Groups

Individual and groups of chemicals that cannot be clustered are listed in this category.

**A-15. Pentachlorobenzene (penta-CB)**

Pentachlorobenzene is used, or has been used, as a dielectric fluid, a fungicide, and a flame retardant. It can also be produced by degradation of other organochlorine compounds, such as lindane and HCB, and is suggested to be a contaminant of HCB. Similarly, it is used as a starting material in the manufacture of the fungicide quintozene (pentachloronitrobenzene) and is a technical impurity in this compound. Other possible sources of chlorinated benzene contamination include use as chemical intermediates, as solvents in the manufacture of dyes, as lubricant and pesticides, and as transformer oils. It results as a byproduct or contaminant during the production of other chlorinated substances and is found in certain industrial effluents. It can be released from waste incinerators where it is produced during the combustion of plastics and chlorinated waste (US EPA 1999). In Canada, based on 1991 data, up to 200 metric tons of penta-CB were present in dielectric fluids in storage, destined for destruction (Rezek 1999). The UN ECE dossier concludes that penta-CB meets the POPs criteria (van de Plassche et al. 2002, UN ECE 2002).

**Toxicity:** The liver, kidney, adrenals, and thyroid appear to be the main target organs. No data were found concerning the toxicity of penta-CB on sediment and soil dwelling organisms, terrestrial invertebrates, birds, or wild mammals. In a 16-day EC50 (median effective concentration) test on the water flea (Daphnia magna), the most sensitive indicator of toxic stress was a reduction in productivity after exposure to 25µg/l. The most sensitive indicator of toxic stress during an early life stage toxicity test on fish was larval growth and a 28-day NOEC for survival hatching and growth of 34µg/l was noted for Brachydanio rerio (EC&HC 1993a). A NOEC for crustaceans of 10µg/l has been reported (van de Plassche et al. 2002).

**Persistence:** In surface water, the estimated half-life of penta-CB is reported to range from 194-1250 days, while the half-life for the anaerobic biodegradation of penta-CB in deeper waters is estimated to range from 776-1380 days. The half-life of penta-CB in soil has been calculated at 270 days and estimated at between 194-1250 days. Therefore, penta-CB meets the UNEP POPs criterion for
persistence, as it appears to have a half-life in water of greater than 2 months, and a half-life in soil of greater than 6 months.

Bioaccumulation: The log Kow value is reported to be around 5. However, penta-CB undoubtedly meets the bioaccumulation criterion for a UNEP POP. For example, a whole body BAF of 20,000 has been reported for rainbow trout, and a BAF of 401,000 has been reported for earthworms (EC & HC 1993a).

Low levels have also been detected in human breast milk. In a study of Canadian women reported in 1987, an average concentration of 2 ppb (milk fat basis) was found in 17% of 18 samples taken from the indigenous population, and in 97% of the general population, penta-CB was found at an average of 3 ppb (US EPA 1999).

Potential for long-range environmental transport: The estimated half-life of penta-CB in air ranges from 45 days to over 1 year, which is sufficient to permit long-range transport (EC&HC 1993b). Long-range transport is confirmed by monitoring data provided by Muir, showing its presence in Arctic sea water and in the blubber of ringed seals from the Arctic (e.g., mean of 2.96ng/g wet wt in blubber from seals in Northern Labrador). In addition, penta-CB has been reported in air masses over the Pacific (EC&HC 1993a).

A-16. Short-chained chlorinated paraffins (SCCPs)
Short-chained chlorinated paraffins (SCCPs) are used in metal working fluids to cool and lubricate the tool/metal interface and to flush away the chips of cut metal. They are also used in paints and sealants, as flame-retardants in rubber and textiles, and in fat liquors in the leather working industry.

In 1998, SCCPs were the subject of an International Declaration in which EU countries, Iceland, Norway, and Switzerland shared “the objective of controlling and limiting the risks arising from the dispersive uses of short-chain chlorinated paraffins using appropriate national and/or international procedures” (ECE/EB.AIR/57 1998). Subsequently, in March 2003, the LRTAP Working Group reviewed the Draft II dossier of 3 March 2003 on SCCPs and their expert judgment based on the dossier was that SCCPs met the criteria for UNECE POPs (COM010_env_NI-LRTAP 2003). In 2004, the European Commission went on the record as wanting SCCPs to be included in the UNECE Protocol to restrict its uses (EC 2004).

Toxicity: The main concerns involve toxicity to aquatic invertebrates and the ability to cause cancer in rodents. In animal experiments, the principal signs of toxicity were effects on the liver and thyroid (EU RAR 2000). SCCPs also feature on Sweden’s list of suspected endocrine disruptors (BKH report 2000).

Persistence: SCCPs are persistent in water. The available screening studies indicate that the half-life for mineralization is likely to be greater than 60 days in marine water and greater than 180 days in marine sediment (EU RAR 2000). In line with EU requirements, in 2005 industry was proposing to undertake further testing to verify whether or not the criterion for persistence was met.

Bioaccumulation: SCCPs meet the UNEP POPs criterion for bioaccumulation, as BCFs of over 5,000 have been measured in fish and in the common mussel (EU RAR 2000). SCCPs have also been found in three samples of breast milk taken from women living along the Hudson Straight at levels of 10.6-16.5ng/g lipid (EU RAR 2000).

Potential for long-range environmental transport: SCCPs are found in Arctic biota, including seals and beluga whales (with a mean of 142 ng/g wet wt in 6 whales from Kimmirut in 1995), showing that long-range transport can occur (Muir et al. 1999 and 2001). This is confirmed by findings of SCCPs in fish at Ellasjoen, at a latitude of 74oN in the Norwegian Arctic, which is far from any point source (COM010_env_NI-LRTAP 2003). The half-life in air of SCCPs is estimated to be between 1.9 and 7.2 days, long enough for significant long-range transport to occur (EU RAR 2000).

A-17. Polychlorinated naphthalenes (PCNs)
PCNs were in use prior to PCBs as dielectric (insulating) fluids in electrical equipment and as heat-transfer fluids and plasticisers. In limited quantities, PCNs have also been used as lubricants, dye carriers in paints, and wood preservatives. They are also found as contaminants in PCB products.
Stockpiles may still exist, for example in 1997, British Telecom was reported to have significant stocks (ENDS Report 1999). Putting these chemicals under global control could help to ensure their proper disposal. Recently, total PCN concentrations in digested sludge from 14 UK sewage treatment plants were found to range from 50 to 90μg/kg, suggesting numerous and ongoing diffuse sources (Stevens et al. 2003). Indeed, mono- through to octa-chlorinated naphthalenes have been measured in start-up, steady operation, and shutdown of flue gases and fly ash during different stages of a municipal incinerator and other incineration processes (Takasuga et al. 2004). Moreover, although PCNs are no longer used in most countries, production in developing countries cannot be ruled out; their manufacture is relatively simple.

**Toxicity:** Long-term exposure to PCNs has been associated with cancer and chronic liver disease in workers (Hayward 1998). Several of the 75 possible PCN congeners exhibit dioxin and PCB-like properties and have similar mechanisms of action, such that additive-type effects are possible. In wildlife, PCNs may account for around half of the dioxin-like toxicity exhibited (Kannan et al. 2000). Potential effects in porpoise are a concern (Ishaq 2000). PCNs are considered to have endocrine disrupting properties; they appear to cause an imbalance in vitamin A in wildlife (Swedish EPA 2002).

**Persistence:** PCNs are considered to be persistent chemicals, but only limited data are available.

**Bioaccumulation:** Some PCNs have been found to bioaccumulate to a significant extent, particularly in birds. Experimental BCF values in fish are greater than 5000 for di, tri, tetra, and pentachloronaphthalenes (Crookes and Howe 1993). The levels of PCNs in Swedish breast milk have declined, and were reported at 0.48ng/g lipid in 1992 (Noren and Meironyte 2000).

**Potential for long-range environmental transport:** PCNs are widespread global environmental contaminants. Elevated PCN levels in the remote Arctic have been attributed to air masses arising from Europe (Harner et al. 2001). PCNs have also been found in the Arctic and Antarctic marine food webs, with levels ranging from 1.5 pg/g in krill to 2550 pg/g in South polar skua on a wet weight basis. Lower chlorinated PCNs were the predominant congeners in most of the wildlife tested, except for skua and polar bears (Corsolini et al. 2002).

**A-18. Tetrachlorobenzene (Tetra-CB)**

Environmental releases of tetrachlorobenzene may result from direct usage, from its accidental production as a byproduct, and perhaps also from the degradation of pentachlorobenzene and hexachlorobenzene. There are several different isomers of tetrachlorobenzene. 1,2,4,5- tetra-CB has been produced and used as an insecticide and as an intermediate in the production of herbicides and defoliants. It is no longer produced in the United States or Canada, but releases to the environment occurring through various waste streams, including pulp mill effluents and the effluents of municipal waste incinerators. 1,2,3,4-tetra-CB is used as a component of dielectric fluids and as a chemical intermediate in the synthesis of pentachloronitrobenzene (quintozene). It is no longer produced in Canada, but was reportedly still produced in the United States, and presumably elsewhere. Based on 1991 data, in Canada alone there were 1,300 metric tons of tetra-CB in use in dielectric fluids (US EPA 1999, Rezek 1999, EC & HC 1993b).

**Toxicity:** The major target organs in mammalian species seem to be the liver, kidney, and thyroid. With regard to ecotoxicity, there is a lack of data on the effects of tetra-CB on benthic and soil dwelling organisms, despite the fact that tetra-CBs can persist under anaerobic conditions. Data are also lacking on the acute and chronic toxicity of tetrachlorobenzenes to terrestrial invertebrates, aquatic plants, birds, and wild mammals (EC&HC 1993b).

**Persistence:** The half-life of 1,2,4,5-tetra-CB in surface water is estimated to range from 28 to 417 days, while its half-life for anaerobic biodegradation in deeper waters is suggested to range from 120 to 720 days. The half-lives in soil of the tetrachlorobenzenes are estimated to range from 28 to 417 days. Also, 1,2,4,5- and 1,2,3,4-tetra-CB were not biodegraded after incubation in sewage sludge under anaerobic conditions for 32 days. The tetrachloro-benzenes are therefore expected to persist in sediments, and despite strongly adsorbing to sediment, some can be removed by re-suspension. The half-life for the biodegradation of 1,2,4,5-tetra-CB in sediment is estimated to range from 56 to 1250 days. These values suggest that tetrachlorobenzene meets the persistence criteria for a UNEP POP (EC&HC 1993b).
Bioaccumulation: The following BCF values have been reported for the various isomers in fish (ATSDR 1998):

- 1,2,3,4-tetrachlorobenzene 3,800-12,000
- 1,2,3,5-tetrachlorobenzene 1,800-3,900
- 1,2,4,5-tetrachlorobenzene 4,000-13,000.

This suggests that 1,2,3,4- and 1,2,4,5-tetra-CB would meet the UNEP criterion for bioaccumulation, despite quoted log Kow values of between 4.51 and 4.65 for the three isomers. The terrestrial food chain may also be at risk. For example, a BAF of 84,000 has been reported for earthworms (Eisenia andrei). 1,2,4,5-tetra-CB has also been found to biomagnify in the lichen-caribou-wolf food chains in the Canadian Arctic at Bathurst Inlet (Kelly and Gobas 2001).

In approximately one-third of 412 samples of Canadian breast milk taken in 1986, 1,2,3,4-tetra and 1,2,3,5-tetra were found. Maximum levels were 99ng/g milk fat and 138 ng/g milk fat respectively (Mes et al. 1993).

Potential for long-range environmental transport: 1,2,4,5-tetra-CB has been found as a contaminant in the Arctic terrestrial food chain by Kelly and Gobas (2001), demonstrating that this substance is able to undergo long-range transport. Similarly, monitoring data from the Arctic provided by Muir (pers. comm.) show that 1,2,3,4-tetra-CB and a range of other chlorobenzenes may be found in aquatic biota and sea water. Levels in the blubber of ringed seals from Northern Labrador were 1.52 ng/g wet weight (mean of 20 samples).

C-1, 2. Unintentional Production

The following two chemicals are generated unintentionally. WWF recommends that these chemicals be listed in Annex C of the Stockholm Convention.

C-1. Octachlorostyrene (OCS)

Octachlorostyrene is not commercially manufactured. Rather, it is an unwanted byproduct of processes that combine carbon and chlorine at high temperatures. These processes include chlorine production, magnesium production, commercial production of chlorinated solvents, and aluminium processes. OCS appears to be a ubiquitous contaminant, having been found in human breast milk (in Canada), in the eggs of little owls from Belgium (Jaspers et al. 2005), and in both Arctic and Antarctic biota.

Toxicity: Effects on the liver, thyroid, kidney, and blood have been noted in experimental animals exposed to OCS (OME & MDNR 2000). Also, 4-hydroxy-heptachlorostyrene, a suspected metabolite, has been shown by Sandau and co-workers to bind to the thyroid hormone transport protein, raising the possibility of thyroid hormone disrupting effects and effects on retinol transport (Sandau et al. 2000b). However, testing of OCS in Japan has not confirmed grounds for concern with regard to its endocrine disrupting properties (Hori 2003). The structural similarity of OCS and hexachlorobenzene (which is already designated as a UNEP POP) suggests that OCS has a similar toxicological profile.

Persistence: The structure of OCS indicates that it is likely to be persistent. However, there is a lack of persistence data for this substance in various media.

Bioaccumulation: Octachlorostyrene is very bioaccumulative, with recorded BCF values of over 5,000. It therefore meets the bioaccumulation criterion for a UNEP POP. It has also been found in 59% of 412 samples of breast milk taken from Canadian women. The mean level was 5.2 ng/g milk fat, with a maximum value of 191ng/g milk fat (Mes et al. 1993).
Potential for long-range environmental transport: The presence of OCS in the tissue of polar bears and Antarctic seabirds demonstrates that it can undergo long-range transport. 4-hydroxy-heptachlorostyrene, the suspected metabolite, is also found in the plasma of polar bears (Sandau et al. 2000a).

C-2. Polycyclic aromatic hydrocarbons (PAHs)
These substances are listed on the LRTAP POPs Protocol and thus meet the UNEP POPs screening criteria. PAHs are a group of chemicals released during the incomplete burning of oil, coal, gas, and other organic materials such as trees during forest fires. Significant sources of PAHs include domestic combustion; vehicles; the production sites of metals, coke, and asphalt; and power stations. A survey of sludge samples from 14 UK sewage treatment plants found that total PAH (24 compounds) concentrations ranged from 67-370 mg/kg dry weight; all the samples would exceed the proposed EU limit for sludge used on land (Stevens et al. 2003).

Many PAHs have been designated by IARC as probable or possible human carcinogens and certain PAHs have also been linked to birth defects and reproductive problems in animals (ATSDR 1995). Some PAHs have endocrine disrupting properties and some have been shown to affect the immune system. In addition, recent studies have indicated that PAHs are more toxic in the presence of UV light, such that laboratory studies may underestimate their effects in the marine environment (MAFF 2000, Lyons 2002).

The LRTAP POPs Protocol requires, in Article 3(5), that each Party reduce its total annual PAH emissions from the levels recorded in any specified year between 1985 and 1995. Because PAHs are a group of over 100 different chemicals, four indicator compounds have been designated for the purpose of emission inventories: benzo(a)pyrene (BaP), benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno (1,2,3-cd) pyrrene.

3. Conclusion
The Stockholm POPs Convention is a historic, forward-looking agreement that was designed to respond to emerging scientific knowledge about chemicals beyond the initial 12. The adding mechanism is a key element of the treaty and Parties to the Convention should not hesitate to act when there is convincing evidence of a chemical’s toxicity, persistence, bioaccumulation, and long-range transport. WWF urges Parties to consider the 20 chemicals detailed in this paper for nomination to the POPs Review Committee.
C2. LIST FROM OZTOXICS

Source: www.oztoxics.org

NGO Candidate Chemicals

There are many chemicals with POP-like characteristics which need priority consideration. Some are already scheduled for elimination through countries’ national action or regional treaties like the UNECE Convention on Long-Range Transboundary Air Pollution (LRTAP) on POPs and the Convention for the Protection of the Marine Environment of the North-East Atlantic (OSPAR).

NGOs from across the globe have identified a range of persistent toxic substances that they wish to be considered for inclusion in the Stockholm Convention. Once added to the Stockholm Convention, their production and use can be eliminated in many more countries.

Unfortunately, NGOs are not allowed to nominate chemicals themselves but must build the body of evidence to convince their national representatives to submit a proposal to list a new chemical.

Candidate POPs in Consumer Products:

**Polybrominated diphenylethers (PBDEs)** - Three commercial formulations of PBDEs (penta, octa, decaBDEs) are used as flame retardants in plastics for TVs and computers, in carpets, car interiors and polyurethane foams for furniture and bedding. PBDEs are persistent, mobile in the environment, bioaccumulate, disrupt thyroid hormones and are linked with cancer and reproductive damage.

**Perfluorooctane Sulfonate (PFOS) Perfluorooctanoic Acid (PFOA)** - These perfluorochemicals are used as soil/stain resistance treatments for fabrics/paper, in coatings for metal surfaces including non-stick cookware and in electronics components and fire fighting foams. They are very persistent, bioaccumulate, have developmental and reproductive effects and are linked with cancer.

Candidate Industrial Chemicals:

**Short-Chained Chlorinated Paraffins (SCCPs)** - used as lubricants, surface coatings, rubber / leather finishing; persistent, mobile, bioaccumulates, highly toxic to aquatic invertebrates and algae, toxic to liver, kidney and thyroid, inhibits intercellular communication.

**Polychlorinated Napthalenes** - uses include cable insulation, wood preservative, engine oil additive, capacitor fluids, dye intermediate, flame retardant; similar chemical and physical properties as PCBs, e.g., induce dioxin-like responses in fish and mammals.
Hexabromobiphenyl (HxCB) - used as a fire retardant in thermoplastics for industrial and electrical products; persistent, endocrine disruptor and linked to cancer (included in LRTAP)

Hexachlorobutadiene - used as a solvent and heat transfer fluid; persistent, causes kidney and liver damage, developmental effects, carcinogen.

Pentachlorobenzene - used as a fire retardant and to make fungicides, high potential for PCB generation; can affect the central nervous system, liver and kidneys, linked to toxic effects on human reproduction.

**Candidate Pesticides:**

Lindane/ hexachlorocyclohexane - an insecticide for the treatment of seeds, lice and scabies; persistent, long-range transport in the atmosphere; endocrine disruptor and linked to cancer (included in LRTAP, OSPAR)

Endosulfan - an insecticide and acaricide (kills mites), moderately persistent, toxic to birds and very toxic to aquatic life, potential for endocrine disruption

Dicofol - an acaricide, structurally similar to DDT; persistent in food and water, highly toxic to aquatic life, causes egg-shell thinning in some bird species

Chlordecone - an insecticide, fungicide and degradation product of the POPs insecticide Mirex; persistent, affects nervous system, skin, kidney, liver, and reproductive system, causes tumours in liver, adrenal gland, and kidneys in laboratory test animals (included in LRTAP)

**Candidate Byproducts:**

Brominated dioxins and bromo-chloro-dioxins - formed through combustion of brominated products and chemicals; suspected carcinogens and endocrine disruptors

Polycyclic Aromatic Hydrocarbons (PAHs) - formed during incomplete combustion of organic material. Also used in dyes, plastics and pesticides; persistence, bioaccumulates in aquatic organisms; five PAHs probable or possible human carcinogens (included in LRTAP, OSPAR)

Octachlorostyrene - formed during electrolytic production of magnesium when graphite anodes used; persistent, bioaccumulates, toxic to aquatic life, suspected endocrine disruptor

Organometals - e.g., organotins, methyl mercury – industrial uses and biocides; very persistent, neurotoxins, developmental effects, linked with cancer (included in LRTAP, OSPAR)
D. INDIA AND NEW POPS

NEW POPs AND THEIR RELEVANCE TO INDIA

PRODUCERS

Of the nominated chemicals, the following were/ are produced in India. In addition to those intentionally produced, the unintentionally produced chemicals are also of relevance.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Past and Present Producers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha HCH</td>
<td>Not intentionally produced. Since India produced Lindane, these are relevant.</td>
</tr>
<tr>
<td>Beta HCH</td>
<td>Not intentionally produced. Since India produced Lindane, these are relevant.</td>
</tr>
<tr>
<td>Chlordane</td>
<td>India (All India Medical Corp, Bharat Pulverizing Mills, Excel Industries, Krishi Rasayan, Mewar Oil and General Mills)</td>
</tr>
<tr>
<td>Endosulfan</td>
<td>India (KCIL, Kanoria, India Pesticides Ltd). Only Romania and India are current producing countries.</td>
</tr>
<tr>
<td>HBB</td>
<td>-</td>
</tr>
<tr>
<td>Lindane</td>
<td>India (KCIL, Kanoria, India Pesticides Ltd). Only Romania and India are current producing countries.</td>
</tr>
<tr>
<td>OctaBDE</td>
<td>-</td>
</tr>
<tr>
<td>PentaBDE</td>
<td>-</td>
</tr>
<tr>
<td>PeCB</td>
<td>It is believed to come primarily from unintentional production from sources that include: PCBs, chlorinated solvents, pesticides, chemical manufacturing, aluminum casting, waste combustion including barrel burning, ore treatment for metal production of magnesium, copper, niobium, tantalum, titanium dioxide production, wood treatment plants, and hazardous waste incineration.</td>
</tr>
<tr>
<td>PFOS</td>
<td>India (Indofine Chemical Co.),</td>
</tr>
<tr>
<td>SCCPs</td>
<td>-</td>
</tr>
</tbody>
</table>

USES

Following uses have been listed for the nominated chemicals. It should be noted that when some of these are not produced in India, they would still hold relevance due to their usage in products elsewhere. Products containing these chemicals could be available in the country.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha HCH</td>
<td>None; waste product</td>
</tr>
<tr>
<td>Beta HCH</td>
<td>None; waste product</td>
</tr>
<tr>
<td>Chlordane</td>
<td>Pesticide formerly used on banana root borer, fly larvicide, apple scab, powdery mildew, Colorado potato beetle, rust mite, wireworm, and household ant and roach traps.</td>
</tr>
<tr>
<td>Endosulfan</td>
<td>Insecticide for control of aphids, thrips, beetles, foliar feeding larvae, mites, borers, cutworms, bollworms, whiteflies, and leafhoppers. Used on cotton, tobacco, cantaloupe, tomatoes, squash, eggplant, sweet potato, broccoli, pears, pumpkins, corn, cereals, oilseeds, potatoes, tea, coffee, cacao, soybean, and other vegetables. Historically used to control termites and tsetse fly. Used in some countries in the past as a wood preservative.</td>
</tr>
<tr>
<td>HBB</td>
<td>Hexabromobiphenyl has been used as a fire retardant in acrylonitrile-butadiene-styrene (ABS) thermoplastics for constructing business, machine housings and in industrial and electrical products and in polyurethane foam.</td>
</tr>
<tr>
<td>Chemical</td>
<td>Description</td>
</tr>
<tr>
<td>-----------</td>
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</tr>
<tr>
<td>Lindane</td>
<td>Lindane has been used as a broad-spectrum insecticide for seed and soil treatment, foliar applications, tree and wood treatment and against ectoparasites in both veterinary and human applications.</td>
</tr>
<tr>
<td>OctaBDE</td>
<td>Flame retardant primarily for ABS plastics used in office equipment and business machines. Other uses include nylon, low density polyethylene, polycarbonate, phenol-formaldehyde resins, and unsaturated polyesters.</td>
</tr>
<tr>
<td>PentaBDE</td>
<td>PentaBDE been used almost exclusively in the manufacture of flexible polyurethane (PUR) foam for furniture and upholstery in homes and vehicles, packaging, and non-foamed PUR in casings and electronic equipment (EE). They are also used to some extent in specialized applications in textiles and in industry.</td>
</tr>
<tr>
<td>PeCB</td>
<td>No current intentional use believed though PeCB has been found in the following uses: PCBs, dyestuff carriers, flame retardant, and pesticides (quintozene, endosulfan, chlorpyrifos-methyl, atrazine, and clopyrilid). PeCB has been used to make paranitrochlorobenzene (quintozene).</td>
</tr>
<tr>
<td>PFOS</td>
<td>PFOS uses include: fire fighting foams, carpets, leather/apparel, textiles/upholstery, paper and packaging, coatings and coating additives, industrial and household cleaning products, pesticides and other insecticides, photographic industry, photolithography and semiconductor manufacturing, hydraulic fluids, and metal plating.</td>
</tr>
<tr>
<td>SCCPs</td>
<td>SCCPs are used primarily in metalworking applications. Other uses include uses as flame retardants or plasticizers in PVC, paints, adhesives, sealants in buildings, PCB substitutes in gaskets, leather fat liquors, and flame retardants in rubber, car carpets, textiles, and other polymers. SCCPs used as flame retardants are added to rubber in a proportion of 1–10%.</td>
</tr>
</tbody>
</table>

Most important, since POPs knows no boundaries, India is vulnerable to the threats posed by these chemicals even when these are not produced or used in the country.

**SOME REFERENCES PERTAINING TO INDIA**

**ENDOSULFAN**

NRA Review of Endosulfan, (1998) by Australian National Registration Authority for Agricultural and Veterinary Chemicals ([www.nra.gov.au/chemrev/prsendo71.pdf](http://www.nra.gov.au/chemrev/prsendo71.pdf)) mentions that besides Germany manufacturing of endosulfan has been reported for Israel, India and South Korea. Recently, a production start has been reported for Peoples Republic of China.

**In India**, Endosulfan is recommended for use in a number of crops in India including cotton, rice, pulses, plantation crops, fruit crops and vegetables for control of various pests. It is highly recommended in IPM programs and also in Helicoverpa resistance management programs especially in cotton crop. Endosulfan is one of the very economic solutions for managing various pests in a number of crops. It is still one of the widely used molecules by farmers in India. It is recommended in the package of practices of almost all Agriculture Universities in India for control of various pests in different crops especially in Integrated Pest Management Programs due to its selectivity and lower toxicity to various beneficial insects.

**LINDANE**
Historical production of technical HCH and lindane occurred in many European countries, including the Czech Republic, Spain, France, Germany, United Kingdom, Italy, Romania, Bulgaria, Poland, and Turkey, and took place mainly from 1950 or earlier and stopped in 1970 to the 1990s. According to a research by IHPA, technical HCH and lindane have also been produced in other countries including Albania, Argentina, Austria, Azerbaijan, Brazil, China, Ghana, Hungary, India, Japan, Russia, Slovakia and the United States. Exact information is difficult to obtain, as many countries do not keep records of historical pesticides production, sales and usage or the industry considers this to be proprietary information (IHPA, 2006).

It appears that in the last years the production of lindane has rapidly decreased leaving only a small number of producing countries. Romania, India, and possibly Russia are the only countries in the world still currently producing Lindane (IHPA, 2006 and USEPA, 2006, CEC, 2005 Annex A). Other sources indicate that Russia (Li et al., 2004) and China (USEPA, 2006) have stopped producing lindane. India produces and uses lindane for the control of mites in sugarcane at 200 tonnes per year.

For each ton of lindane produced, around 6-10 tons of other isomers are also obtained.

Lindane has been detected in cow’s milk in countries that still use the chemical as a pesticide. In a study performed in Uganda, Africa, the concentrations of gamma-HCH in cow’s milk was 0.006–0.036 mg/kg milk fat, respectively. Mean levels of gamma-HCH analyzed in cow’s milk samples from two separate areas in India were 0.002 and 0.015 mg/kg. A monitoring study of 192 samples of cow’s milk from Mexico revealed 0.002–0.187 mg/kg of gamma-HCH (ATSDR, 2005).

Exposure of children to lindane is a particular concern. Gamma-HCH has been found in human maternal adipose tissue, maternal blood, umbilical cord blood and breast milk. Lindane has also been found to pass through the placental barrier. Mean breast milk concentration of lindane was 0.084 mg/l in a study in India. An average level of 6 ppb lindane in breast milk was obtained in a study in Alberta, Canada (ATSDR, 2005).

In India, blood levels of gamma-HCH were significantly higher in 135 breast cancer patients, 41-50 years of age, compared to a control group without the disease. However, in similar studies in other countries, a correlation between breast cancer incidence and elevated levels of gamma-HCH in blood was not observed (ATSDR, 2005).

PFOS

The US Environmental Protection Agency (US EPA) compiled a list of non-US companies, which are believed to supply PFOS-related substances to the global market. Of these (and excluding the plant of 3M in Belgium), six plants are located in Europe, six are located in Asia (of which four are in Japan) and one in Latin America (OECD, 2002). However, this list may not be exhaustive or current. According to the recent submission from Japan to the SC there is one manufacturer in Japan still producing PFOS and with a production amount of 1-10 tonnes (2005).

Alpha HCH

According to Li and Macdonald (2005) global usage of technical HCH was dominated by 10 countries headed by China, which consumed almost half of the total global quantity. The other countries were (in order of decreasing usage): Former Soviet Union, India, France, Egypt, Japan, United States, East Germany, Spain and Mexico. Usage of technical HCH was banned in most western countries and Japan in the 1970s but continued in China and Russia until 1983 and 1990. In 1990, India also banned technical HCH for agricultural use but kept it for public health uses (AMAP, 2004). Technical HCH usage steadily declined and now technical HCH is virtually no longer used worldwide. However, there are indications that the use of stockpiles, limited use for public health purposes and/or illegal use cannot be excluded (Zhulidov et al., 2000; Bakore et al., 2003; Qian et al., 2006).
Levels in biota vary, depending on the location (recent usage and/or high pollution) and species. Alpha-HCH is in most cases the dominant isomer in fish (Willett et al., 1999). E. g. concentrations of HCHs (mainly the alpha-isomer) in several fish species from India ranged between 6 to 68 ng/g ww. Fish samples collected from the Nile River near Cairo in 1993 showed a concentration of alpha-HCH of 0.5 ng/g ww (UNEP, 2003).

Alpha-HCH has been found in cow’s milk in countries where HCH had been used recently. Mean levels of alpha HCH in cow’s milk of two different regions in India were 0.012 mg/kg lipid and 0.0045 mg/kg lipid, respectively (ATDSR, 2005). 140 bovine milk samples from 14 districts of Haryana, India (sampled within 1998 - 1999) were analysed for organochlorine pesticide residues. Four percent of the samples exceeded the maximum residue limit (MRL) of 0.05 mg/kg as recommended by WHO for alpha-HCH (Sharma et al., 2006). A monitoring study (192 samples) of cow’s milk from Mexico revealed 0.001 - 0.201 mg/kg alpha-HCH (ATDSR, 2005).

Breast milk samples from India contained 0.16 mg/l (mean) (Nair and Pillai, 1992). Another Indian study reports 0.045 mg/l alpha-HCH in breast milk (Nair et al., 1996).

The association between alpha-HCH exposure and intrauterine growth retardation (IUGR, < 10th percentile of birth weight for gestational age) was examined in India. Statistically significant associations (p < 0.05) between maternal blood levels of alpha-HCH and interauterine growth retardation were found (Siddiqui et al., 2003).

**Beta HCH**

According to Li and Macdonald (2005) global usage of technical HCH was dominated by 10 countries headed by China, which consumed almost half of the total global quantity. The other countries were (in order of decreasing usage): Former Soviet Union, India, France, Egypt, Japan, United States, East Germany, Spain and Mexico. Usage of technical HCH was banned in most western countries and Japan in the 1970s but continued in China and Russia until 1983 and 1990. In 1990, India also banned technical HCH for agricultural use but kept it for public health uses (AMAP, 2004). Technical HCH usage steadily declined and is now virtually out of use worldwide. However, there are indications that the use of stockpiles, limited use for public health purposes and/or illegal use cannot be excluded (Zhulidov et al., 2000; Bakore et al., 2003; Qian et al., 2006).

High concentrations were reported for India due to agricultural use and Malaria control activities. Blood serum samples from India contained up to 0.02 mg beta-HCH/l, whereas adipose tissue contained up to 0.18 mg/kg (Nair and Pillai, 1992).