**Stockholm Convention on Persistent Organic Pollutants**

**POPs Review Committee**

**PENTACHLOROPHENOL AND ITS SALTS AND ESTERS**

**DRAFT RISK PROFILE**

Draft prepared by the ad hoc working group on pentachlorophenol and its salts and esters under the POPs Review Committee of the Stockholm Convention

**Second draft**

16 April 2013

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# Introduction

1. The European Community and its member States that are Parties to the Convention submitted a proposal to list pentachlorophenol and its salts and esters in Annex A, B and/or C of the Convention, which was considered by the POPs Review Comittee at its seventh meeting held October 10-14, 2011 in Geneva (UNEP/POPS/POPRC.7-4). In this proposal, the reasons for concern were that Pentachlorophenol (PCP) and its related compounds (sodium pentachlorophenate, pentachlorophenyl laurate and pentachloroanisole, a transformation product of PCP) are persistent in the environment and are frequently found in environmental compartments in remote areas. Information indicates that these substances are highly toxic to wildlife and humans, have the potential for long range transport and the potential to bioaccumulate. In addition, contaminants including hexachlorobenzene, dioxins and furans are produced in the manufacturing process, although these chemicals should already be controlled as they are listed in the Convention.
2. At its seventh meeting, the Committee agreed to defer the decision concerning the proposal, pending the receipt of additional information on the transformation of pentachlorophenol to pentachloroanisole and the proposal by Japan to fill information gaps concerning the conversion of PCP to pentachloroanisole (PCA). It was suggested that quantitative information was insufficient to conclude whether PCA is a major transformation product of PCP under environmentally relevant conditions and additional information should be collected on the extent of the conversion of PCP to PCA. Intersessionally, the Japanese government also conducted a review of the literature on PCP transformation in the environment, especially in soil, which is considered to be the most relevant PCP-contaminated compartment in the environment.
3. At its eighth meeting, held from 15 to 19 October 2012 in Geneva, both the literature review and preliminary results from the laboratory studies conducted by Japan were presented to the Committee. The Committee had before it a note by the Secretariat on a proposal to list pentachlorophenol and its salts and esters in Annexes A, B and/or C to the Convention (UNEP/POPS/POPRC.8/5) and additional information on the substances collected since the Committee’s seventh meeting (UNEP/POPS/POPRC.8/INF/7). The Committee adopted decision POPRC-8/4, on PCP and its salts and esters, which stated:
4. *Having examined* the proposal by the European Union and its member States, parties to the Stockholm Convention on Persistent Organic Pollutants to list pentachlorophenol and its salts and esters in Annexes A, B and/or C of the Convention and having applied the screening criteria specified in Annex D to the Convention,
5. *Decided*, in accordance with paragraph 4 (a) of Article 8 of the Convention, that it is satisfied that the screening criteria have been fulfilled for pentachlorophenol and its salts and esters, as set out in the evaluation contained in the annex to the present decision. The conclusion in the annex to decision POPRC-8/4 was: While the pentachlorophenol molecule does not meet all the screening criteria specified in Annex D, the Committee concluded, taking into account its transformation product pentachloroanisole, that pentachlorophenol and its salts and esters meet the screening criteria specified in Annex D;
6. *Also decided*, in accordance with paragraph 6 of Article 8 of the Convention and paragraph 29 of decision SC-1/7 of the Conference of the Parties to the Convention, to establish an *ad hoc* working group to review the proposal further and to prepare a draft risk profile in accordance with Annex E to the Convention;
7. *Invited*, in accordance with paragraph 4 (a) of Article 8 of the Convention, parties and observers to submit to the Secretariat the information specified in Annex E.
8. A number of parties and observers have responded to this invitation. Information on the transformation of PCP to PCA as well as additional information on persistence, bioaccumulation, monitoring and effects of PCA was submitted for review at Annex E. Information on contaminants (e.g., dioxins, furans and hexachlorobenzene) was also submitted for consideration.

##### 

##### 1.1 Conclusion of the Review Committee regarding Annex D information

1. The Committee evaluated Annex D information at its eighth meeting held in Geneva from 15 to 19 October 2012 and decided that, while the PCP molecule itself does not meet all the screening criteria specified in Annex D, PCP and its salts and esters meet the screening criteria specified in Annex D, taking into account its transformation product PCA.

##### 1.2 Data sources

1. The primary source of information for the preparation of this risk profile was the proposal submitted by the European Community and its member States that are Parties to the Convention, contained in documents UNEP/POPS/POPRC.7/4, UNEP/POPS/POPRC.7/INF/5, UNEP/POPS/PORC.7/INF/5/Add.1; additional information submitted for Annex D contained in documents UNEP/POPS/POPRC.8/5 and UNEP/POPS/PORC.8/INF/7\*; and additional information submitted for Annex E evaluation. In particular:

* 2012. Government of Canada. PCA monograph;
* September 2008 – USEPA Reregistration Eligibility Decision (RED) and supporting documentation (e.g., USEPA memos dated 16 February 2008 and 14 April 2008) for PCP. The RED documents are available at: www.regulations.gov – EPA Docket OPP-2004-0402-0078;
* September 2010 – USEPA Integrated Risk Information System (IRIS) Summary for PCP;
* February 16, 2008 – USEPA memo – Environmental Fate and Transport Assessment of PCP for Reregistration Eligibility Decision (RED).

1. In addition, the following parties and observers have responded to the request for information specified in Annex E of the Convention: Canada, Croatia, Estonia, Mexico, Nigeria, Romania, Slovakia, Sri Lanka, Sweden, United States of America, joint submission of IPEN and Alaska Community Action on Toxics (ACAT) and Wood Preservation Canada.
2. A more elaborated summary of the submissions is provided in a separate information document titled Supporting document for the risk profile under Appendix I: Summary of data submitted by Parties and observers for information specified in Annex E of the Convention.

##### 1.3 Status of the chemical under international conventions

1. PCP is subject to a number of regulations and action plans:

* Rotterdam Prior Informed Consent Convention;
* OSPAR List of Chemicals for Priority Action (1998);
* Nominated as candidate for inclusion in Annex I of LRTAP Protocol on POPs.

##### 1.4 Chemical identity

1. PCP is an aromatic hydrocarbon of the chlorophenol family and was first introduced for use as wood preservative in the 1930’s. Since its introduction, PCP has had a variety of other applications (biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent and used in the production of pentachlorophenyl laurate). The salt sodium pentachlorophenate (Na-PCP) was used for similar purposes as PCP and readily dissociates to PCP. The ester pentachlorophenyl laurate (PCPL) was used in textiles. PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst. Contaminants including hexachlorobenzene, dioxins and furans are produced in the manufacturing process. Pentachlorobenzene is also suspected to be present. These compounds are inherently toxic, as well as environmentally persistent and their presence may increase the ecological risk associated with the use of pentachlorophenol. This information is captured in , and .
2. PCA is not a registered substance and is not released directly into the environment. It is produced through the transformation of PCP.

**Table 1‑1: Names and registry numbers**

|  |  |  |
| --- | --- | --- |
| Common name  Chemical name | pentachlorophenol  2,3,4,5,6-pentachlorophenol | |
| CAS registry numbers | pentachlorophenol  sodium pentachlorophenate  pentachlorophenyl laurate | 87-86-5  131-52-2 and 27735-64-4 (as monohydrate)  3772-84-9 |
| Trade name and other names for pentachlorophenol | Pentachlorophenol is abbreviated as PCP. Product names include Acutox, Block Penta, Chem-Penta, chem-Tol, Chlon, Chlorophen, Cryptogil Oil, Cryptogil OL, Dirotox, Dow Pentachlorophenol DP-2 Antimicrobial, Dowcide 7, Dowcide 7/EC-7/G, Dowicide 6, Dowicide 7, Dowicide 7 Antimicrobial, Dowicide EC-7, Dowicide G, Dura TreetII, Durotox, EP 30, Forpen-50 Wood Preservative, Fungifen, GlazdPenta, Grundier Arbezol, 1-hydroxypentachlorobenzene, Lautor A, Lauxtol, Lauxtol A, Lauxtrol A, Liroprem, OnTrack We Herbicide, Ortho Triox Liquid Vegetation Killer, Osmose Wood Preserving Compound, Penchlorol, Penta, Penta C 30, Penta Concentrate, Penta Plus 40, Penta Pres 1-10, Penta Ready, Penta WR, Penta WR1-5 Penwar, Pentachlorophenate, 2, 3, 4, 5-pentachlorophenol, Pentachlorophenol DP-2, Pentachloropheno, Pentachlorphenol, Pentacon, Penta-kil, Pentasol, Pentchloral, Penwar, Peratox, Permacide, Permagard, Permasan, Permatox, Permatox DP-2, Permatox Penta, Permite, Persasan, Prevenol, Priltox, Santobrite, Santophen, Santophen 20, Sautox, Sinithuo, Sinituho, Term-I-Trol, Thompson's Wood Fix, Watershed Wood Preservative, Weed and Brush Killer, Weedone, Witophen P, Woodtreat, Woodtreat A. | |
| Trade names and other names for sodium pentachlorophenol | Penta-ate, Pentachlorophenate sodium, Pentachlorophenol sodium salt, Pentachlorophenoxy sodium, Pentaphenate, Phenol pentachloro- sodium derivative monohydrate, Sodium PCP, Sodium pentachlorophenolate, Sodium pentachlorophenoxide, Penta-ate, Pentachlorophenate sodium, Pentachlorophenol sodium salt, Pentachlorophenoxy sodium, Pentaphenate, Phenol pentachloro- sodium derivative monohydrate, Sodium PCP, Sodium pentachlorophenolate, Sodium pentachlorophenoxide. | |
| Microcontaminants | Dioxins, furans, hexachlorobenzene and suspected to contain pentachlorobenzene | |

**Table 1‑2: Structures**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Pentachlorophenol | Sodium Pentachlorophenate | Pentachlorophenyl laurate | Pentachloroanisole |
| Molecular formula | C6HCl5O and C6Cl5OH | C6Cl5ONa and C6Cl5ONa x H2O (as monohydrate) | C18H23Cl5O2 | C7H3Cl5O |
| Molecular Mass | 266.34 g/mol | 288.32 g/mol | 448.64 g/mol | 280.362 g/mol |
| Structural formulas of the isomers and the main transformation product |  |  |  |  |

**Table 1‑3: Physical and chemical properties of pentachlorophenol and pentachloroanisole**

|  | **Pentachlorophenol** | **Pentachloroanisole** | |
| --- | --- | --- | --- |
| **Properties** | **Value1** | **Value** | **Reference** |
| **Water solubility 25°C** | 0.13% (% weight)  5 mg/L at 0ºC1,2  14 mg/L at 20 ºC1,2  35 mg/L at 50 ºC1,2  14 mg/L at 25 ºC1,2  Low solubility to soluble in water | <1 mg/L  0.24 mg/L  0.19 mg/L  Sparingly soluble in water | <http://cameochemicals.noaa.gov/chemical/20850>  EVA method  logKOW method |
| **Vapour pressure**  **(25ºC)** | 2 mPa (20 ºC)  0.0070-0.213 Pa (25 ºC)  1.1 x 10-4 mm Hg (25 ºC)2  Intermediate volatility | 0.0458 Pa (25 ºC)  0.0933 mm Hg  Intermediate to high volatility | Modified Grain Method  Dobbs and Grant (1980)  Kennedy and Talbert, 1977 classification scheme |
| **Henry’s law constant atm/m3/mol** | 2.45x10-6 atm.m3/mol 2  0.0248 to 0.284 Pa m3/mol 2  Potential to volatilise from water or moist soil | 1.94x 10-3 atm-m3/mole (25 ºC) (Group method)  (1/H = 12.7, KAW = 0.003)  7.12 x 10-5 atm-m3/mole (25 ºC) (Bond method)  Potential to volatilise from water or moist soil | HENRYWIN v3.2 in U.S. EPA 2011  Mackay and Wolkoff, 1973 classification sheme |
| **Dissociation constant (pKa)** | pH 4.60-5.30  pH 4.72  At neutral pH of most natural waters, PCP is more than 99% ionised. | Not expected to dissociate under environmentally relevant pHs. | **-** |
| **Log Octanol/water partition coefficient (LogKow)** | Between 1.3 and 5.86 and strongly pH. Recommended values are 5.12 and 5.18  Potential to bioaccumulate in biota | 5.30 (modelled)  5.45 (laboratory)  Potential to bioaccumulate in biota | KOWWIN v1.68 in U.S. EPA 2011  Opperhuizen and Voors (1987) |
| **KOC** | 293 to 900 L/Kg(at 0.0125 mg/L)  1000 L/Kg (calculated)  3000 to 4000 L/Kg (measured)  293-4000 L/Kg2  706-3420 L/Kg (measured)2  Slight mobility to moderate mobility in soil | 2474 L/kg  13800 L/kg  Immobile | MCI method, KOCWIN 2.0  KOW method, KOCWIN 2.0 in U.S. EPA (2011)  McCall et al., 1981 classification scheme |

**1values reported in UNEP/POPS/PORC.7/INF/5 unless otherwise indicated**

**2 values reported in Annex E submission from the United States of America, Environmental Fate Assessment of Pentachlorlophenol for the Reregistration Eligibility Decision (RED).**

**PC Code 063001, Case 2505, Antimicrobials Division, 11/19/2004**

# 2. Summary information relevant to the risk profile

##### 2.1 Sources

###### 2.1.1 Uses, Production and Trade

1. Historically, according to the data profile of IRPTC (1983), 90 000 tonnes of PCP per year were produced globally. The Economist Intelligence Unit (1981) estimated world production to be of the order of 50 000-60 000 tonnes per year, based on the North American and European Community output (UNEP/POPRC.7/INF/5). By the 1990s, the widespread use was discontinued in most counties.
2. In Europe, historical uses included use in the remedial treatment of timber and as a surface biocide for masonry. It was used in the preservation of textiles (wool cotton, flax and jute fabrics and yarns used in covers, tarpaulins, awnings, tents, webbing and netting and also sisal and manila ropes). It was also used as a preservative in oil-based paint, glues, adhesives, as an intermediate in the synthesis of pharmaceuticals, as an intermediate product in colouring substances, in mushroom farms, in slime control in pulp and paper as well as an agricultural chemical in weed control.
3. In Australia, historical uses include uses as an antisapstain fungicide and timber preservative.
4. In Canada, historical uses include sapstain and specialty applications (paints, stains, wood joinery products, industrial water treatment products, oil field biocides and material preservatives) (CCME, 1990).
5. In Sweden, PCP was used in large quantities mainly as a wood preservative and in pulp production. An important, but minor use of PCP was in the protection of textiles.
6. In the U.S., PCP was used in rice and sugar production, in water treatment, as a pre-harvest defoliant in cotton and as a general pre-emergence herbicide. It has also been utilised in numerous products including adhesives, construction materials, leather and paper.
7. Currently, PCP has either no uses or is banned in all E.U. member states, Australia, India, Indonesia, New Zealand, Russia and Switzerland. PCP is only allowed for wood preservation with additional restrictions and/or regulations in Belize, Canada, China, Mexico and the United States. In Canada and the U.S. the wood preservation uses are for heavy duty wood preservation uses only. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico; ready-for-use products in Nigeria; and snail elimination to control the spread of schistosmoniasis in China.
8. The only manufacturing site for North America is in Mexico, which the Wood Preservation Industry indicated produced 7 257 tonnes/year in 2009 for the United States, Canada and Mexico.
9. The Mexican Government reported similar production information for 2009 (6 610 tonnes) and also supplied import/export information. Mexico reported that 3670-7343 tonnes were exported yearly between 2007 and 2011 to the United States, Colombia and Peru. Mexico reported importing PCP from the United States, China and Germany between 1997 and 2011. The highest importation estimate was for 2007 where 100 kg of PCP (2 695 000 dollars) was imported into Mexico.
10. Although all uses are restricted to wood preservation in China, the production and use of PCP for snail elimination and schistosomiasis control has increased due to re-emergence of this disease. The annual national output reached approximately 3000 tonnes in 2003 (CESE, 2004; Tan et al., 2008 as cited in Zheng et al., 2012).
11. The United States reported that in 2002, approximately 4 990- 5 444 tonnes were used for the treatment of utility poles, lumber and timbers (construction). Of the amount used, 4 083 tonnes were imported and 1361-1815 tonnes were produced domestically.
12. Canada reported 372-537 tonnes of PCP were produced yearly between 2008 and 2012 for the treatment of utility poles and crossarms.
13. World import and export information from the U.N. Food and Agriculture Organizations’s FAOSTAT database (downloaded on 02/16/2012) obtained from Mongabay, 2013a; Mongabay, 2013b; Mongabay 2013c and Mongabay 2013d are provided in Table 2-1, Table 2-2, Table 2-3 and Table 2-4.

**Table 2‑1: Quantities of Pentachlorophenol Salts Exported by Top 35 Countries.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Country** | **Kilograms Exported** |  | **Country** | **Kilograms Exported** |
| United States of America | 47,808,284 |  | Guatemala | 2,372 |
| China | 9,845,446 | Hungary | 2,300 |
| Israel | 7,693,926 | Columbia | 1,725 |
| Japan | 6,941,217 | South Africa | 1,040 |
| Belgium | 3,308,976 | Canada | 765 |
| Germany | 2,024,919 | Turkey | 700 |
| United Kingdom | 593,744 | New Zealand | 670 |
| Brazil | 122,751 | Finland | 560 |
| Italy | 116,545 | Russian Federation | 418 |
| Mexico | 89,920 | Australia | 225 |
| Egypt | 57,000 | Romania | 200 |
| France | 20,150 | Czech Republic | 109 |
| Malaysia | 20,000 | Norway | 45 |
| Switzerland | 16,151 | Portugal | 33 |
| Panama | 14,357 | Lithuania | 30 |
| Jamaica | 11,121 | Sweden | 17 |
| Thailand | 10,003 | Slovakia | 2 |
| Costa Rica | 7,947 |  |  |

**Table 2-2. Quantities of Pentachlorophenol Salts Imported by Top 62 countries.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Country** | **Kilograms Imported** |  | **Country** | **Kilograms Imported** |
| China | 53,501,688 |  | Panama | 6,612 |
| Japan | 17,678,728 | Jordan | 6,201 |
| United States of America | 7,432,052 | Hungary | 4,728 |
| Germany | 3,405,774 | Slovenia | 4,161 |
| Belgium | 3,291,082 | Algeria | 3,310 |
| Brazil | 885,747 | Denmark | 2,217 |
| United Kingdom | 833,182 | Portugal | 1,568 |
| Israel | 804,358 | Ethiopia | 1,502 |
| France | 793,518 | Norway | 1,500 |
| Canada | 675,235 | Nepal | 900 |
| Italy | 534,502 | Greece | 635 |
| Thailand | 414,360 | Finland | 408 |
| Malaysia | 223,905 | Republic of Moldova | 269 |
| Australia | 174,594 | Bulgaria | 176 |
| Turkey | 118,932 | Paraguay | 149 |
| Mexico | 108,371 | Sri Lanka | 127 |
| Czech Republic | 86,732 | Belarus | 125 |
| Romania | 82,401 | Ireland | 108 |
| Pakistan | 71,468 | Croatia | 102 |
| New Zealand | 56,623 |  | Cyprus | 37 |
| South Africa | 47,941 |  | Lithuania | 11 |
| Costa Rica | 44,191 |  | Montenegro | 10 |
| Guatemala | 44,079 |  | Bolivia (Plurinational State of) | 9 |
| Switzerland | 42,595 |  | Serbia | 5 |
| Nigeria | 36,289 |  | Zimbabwe | 4 |
| Egypt | 20,332 |  | Azerbaijan | 3 |
| Columbia | 17,097 |  | Iceland | 2 |
| United Republic of Tanzania | 16,000 |  | Armenia | 2 |
| Peru | 13,439 |  | Luxemburg | 1 |
| Russian Federation | 12,549 |  | Jamaica | 1 |
| Sweden | 10,651 |  | Estonia | 1 |

**Table 2-3 Quantities of Pentachlorophenol Exported by Top 7 Countries.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Country** | **Kilograms Exported** |  | **Country** | **Kilograms Exported** |
| Mexico | 6,195,680 |  | Canada | 6,490 |
| United States of America | 481,127 | Japan | 1,050 |
| Italy | 18,961 | South Africa | 6 |
| United Kingdom | 10,225 |  |  |

**Table 2-4 Quantities of Pentachlorophenol Imported by Top 27 Countries.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Country** | **Kilograms Exported** |  | **Country** | **Kilograms Exported** |
| United States of America | 1,376,862 |  | Colombia | 840 |
| Canada | 507,154 | Oman | 811 |
| Belgium | 75,031 | Australia | 750 |
| United Kingdom | 73,597 | Nigeria | 344 |
| Malaysia | 41,380 | Cyprus | 235 |
| Romania | 23,640 | Republic of Moldova | 100 |
| Sri Lanka | 21,350 | Czech Republic | 32 |
| Germany | 12,000 | Slovakia | 16 |
| Thailand | 10,955 | Kyrgyzstan | 10 |
| Zimbabwe | 10,000 | South Africa | 4 |
| Zambia | 6,519 | Luxembourg | 2 |
| Jordan | 2,000 | Denmark | 2 |
| France | 1,700 | Greece | 1 |
| Italy | 958 |  |  |

1. More detailed information on current uses as informed by countries is provided in *Supporting document for the risk profile, Appendix I:* *Summary of data submitted by Parties and observers for information specified in Annex E of the Convention, UNEP/POPS/POPRC.7/INF5 and UNEP/POPS/POPRC.7/INF/6*

###### 2.1.2 Releases to the environment

1. There are several sources of PCP in the environment, including the release of PCP when used in accordance with currently registered uses as a wood preservative. During the life cycle of treated wood, PCP is potentially released into the environment during the manufacturing process, wood treatment processes (sapstain, heavy-duty wood), use and disposal of treated wood. Other releases are during the impregnation of heavy-duty textiles and fibres contaminated sites and natural sources or burning processes (OSPAR, 2004).
2. Based on the current bans and restrictions in place, the releases listed above are no longer relevant to all countries and all situations. Specifically, the following information should be noted:

* Registered use on textiles was only reported for Mexico. However, the production information submitted by Mexico indicated that all PCP produced is used for wood preservation;
* Countries such as the United States and Canada have implemented strict regulatory programs to minimize worker and environmental releases for heavy duty wood preservation for treatment facilities and disposal. In both countries, various authorities are involved in the regulation of air emissions, storm water, waste water and control of soil wastes (collection, transportation, handling, storage, treatment, use, diversion, recycling, re-use, recovery, reduction or disposal). Burning of treated wood is not permitted in either country.

1. The following country specific release information was submitted to Annex E:
2. For the 2011 reporting year, thirty-four PCP Form Rs were submitted to the US EPA for the Toxics Release Inventory (TRI). The total on- and off-site disposal and other releases were 43.5 tonnes. Of this total,40.8 tonnes were “on-site releases” and 2.8 tonnes were “off-site” releases. The majority of the on-site releases were classified as on-site hazardous waste landfill (40.5 tonnes) (i.e., a RCTA Subtitle C landfill). Other on-site releases were fugitive air emissions (47 kg), point source air emissions (71 kg) and surface water discharge (232 kg). The majority of off-site releases were classified as “unknown” (1.1 tonnes), “RCRA Subtitle C landfill” (834 kg) and “other landfills” (735 kg).
3. Mexico reported the following releases: Incineration of 17 776 kg. Emissions to air were 38 kg in each year between 2006 and 2009. Emissions to soil were estimated to be 0.0029 kg (2005).

###### 2.1.3 Other sources of PCP

1. The widespread use of PCP for the treatment of wood and textiles prior to regulations and bans has resulted in contaminated aquatic and terrestrial environments which continue to be potential sources of PCP and PCA in the aquatic environment.
2. PCP is also a transformation product and metabolite of other organochlorines such as HCB (hexachlorobenzene), HCH (lindane) and PCNB (quintozene). The presence of PCP and subsequently PCA in the environment is also as a result of these other sources.



**Figure 1: Examples of Sources of PCP in the environment**

##### Environmental fate of PCP and PCA

1. Aqueous photolysis is the fastest known pathway of PCP degradation and can lead to total mineralization of PCP in water within hours of its release. In air and clean water this is the relevant degradation mechanism as PCP is stable to hydrolysis at environmentally relevant pHs. In waters where turbidity and depth prevent exposure to light, in sediment and in soil, biodegradation is the relevant process.
2. Under normal environmental conditions, the microflora will adapt and biodegrade PCP in water with half-lives less than 4 weeks, in the sediment less than 20 weeks and in soil less than 10 weeks. Many other studies discuss the degradation of PCP in terms of mineralization, with some of them showing a slow rate of mineralization. PCP is moderately mobile in lower pH soils and mobile in higher pH soils.
3. Under aerobic conditions, large numbers of PCP-degrading bacteria have been identified and there are several pathways for degradation of PCP, depending on the experimental or environmental conditions.
4. PCA can be generated from PCP as a result of methylation in the presence of some bacterial and fungal species (e.g., white-rot fungi) under aerobic conditions. Since PCA results from the addition of a methyl group to PCP, it should not be considered degradation product as PCA can be demethylated back to PCP.
5. PCA cannot be produced by abiotic processes such as hydrolysis and photolysis. It is not expected to hydrolyse based on its chemical structure (Lyman et al 1982 in U.S. EPA 1992). PCA is sparingly soluble in water and has a high Koc value indicating that it is likely to be immobile to slightly mobile in soils and partition to sediment in aquatic systems. The Henry’s law constant indicates that PCA is expected to be volatile from moist soil and aquatic systems. Volatility from water was also observed under laboratory conditions.
6. In the organisms that preferentially convert PCP to PCA, conversion appears to be a detoxification step that allows metabolism of otherwise toxic levels of PCP. Unlike PCP, PCA is not an inhibitor of oxidative phosphorylation and is therefore less toxic to wood-rotting fungi and other microbes (Chung and Aust 1995; Suzuki 1983b). The rate of PCA formation from PCP can be high. Up to 86% of PCP was transformed to PCA in some studies using isolated strains of white-rot fungi (Walter et al, 2004; Badkoubi et al., 1996; Pfender et al., 1997; Rigot and Matsumura, 2002). Trace amounts of PCA (up to 5.1%) were formed from PCP when other species or mixed-microflora systems were tested (Walter et al. 2004; Walter et al. 2005; Ford et al., 2007, Machado et al., 2005; Haimi et al. 1993; D’Angelo and Reddy 2000 and Kuwatsuka and Igarashi 1975; Rubilar et al., 2007; Rigot and Matsumura 2002). Percent formation and mineralisation half-lives are available from these studies, but are of limited environmental relevance since they are only indicative of the degradation for a specific strain of organism under the conditions of the study.
7. Several studies conducted with mixed microflora showed the formation of PCA, followed by a decrease in concentration with formation of successor transformation products in aerobic soils (Haimi et al. 1993; D’Angelo and Reddy 2000 and Kuwatsuka and Igarashi 1975; Rubilar et al., 2007; Rigot and Matsumura 2002). Observed half-lives for PCA are between 20-35 days.However, there is uncertainty with these estimates as studies were conducted with PCP as a starting material, resulting in simultaneous formation and degradation of PCA thereby confounding half-life estimates and making half-lives appear longer than they actually are. Calculated half-lives of transformation products need to be corrected for the rate of formation.. In addition, these estimates are based on low or trace amounts of PCA. These are not full mineralisation half-lives.
8. In the Haimi et al. (1993), D’Angelo and Reddy (2000), Mardones et al. (2009) and Kuwatsuka and Igarashi (1975) papers, it is unclear whether vessels were securely sealed over the course of the study. However, PCA was not volatile in any test conducted with soil where the methods indicated that systems were sealed with volatile traps (Walter et al. 2005; Chung and Aust 1995; Pfender et al., 1997).
9. Biotransformation via dechlorination is the principle degradation pathway in anearobic soil, sludge and aquatic systems once in the sediment. Most studies reviewed showed no formation of PCA under anaerobic conditions (UNEP/POPS/PORC.8/INF/7\*).
10. In aquatic systems, PCA is expected to partition to sediment and volatilise to air over time based on its physical-chemical properties and the volatility observed in the laboratory. Pierce and Victor 1978 examined the fate of PCP and its transformation products after an oil spill showed that under field conditions, PCP is biomethylated to PCA and that both PCP and PCA partitioned to the sediments. There was evidence that PCP transformed to lower chlorinated phenols and anisoles (tetrachlorophenols, trichlorophenols, tetrachloroanisole) as was observed in the aerobic and anaerobic laboratory studies.

##### 2.3 Bioaccumulation

1. In the open literature, a range for the log Kow for PCP varies between 1.3 and 5.86. However, the recommended values are 5.12 and 5.18. The log KOW is strongly pH dependent. The log Kow may not be a good indicator of bioconcentration as PCP is subject to metabolic transformation (UNEP/POPS/POPRC.7/INF/5).
2. In a bioconcentration study conducted in bluegill sunfish, BCF values were 190-790 (U.S. EPA 2008 submitted Annex E information as: UNEP-POPS-POPRC8CO-SUBM-PCP-USA\_8-20130110.En[1].pdf). The BCF in crustaceans, bivalves, aquatic and terrestrial worms and in fish varies between 0.9 – 4900. Only one study reported a BCF > 5000. Considering the majority of residues are below 5 000 and PCP undergoes rapid biotransformation, PCP does not meet the bioaccumulation criteria (UNEP/POPS/POPRC.7/INF/5).
3. Letcher et al. (2009) reports BMFs of 1.5 for PCP in polar bear lipids. Since PCP is a metabolic product of HCB, a known contaminant of Arctic biota, it is expected that some of the PCP measured in the bear is from the metabolism of HCB (not from eating contaminated prey). There is some uncertainty with this BMF estimate for this reason and because the analytical method contained a methylation step that would have made it impossible to differentiate between PCP and PCA.
4. The log KOW value of 5.45 and the BCF values of 12000-20000 for PCA reported by Oliver and Niimi (1985) for fish exceed the laboratory criteria cut-off value of 5000 and indicate a higher potential for bioaccumulation than PCP. However, there are uncertainties with the BCF estimates given that test concentrations for PCA were highly variable over the testing period and multiple chemicals were tested simultaneously, which could have reduced the ability of the organisms to metabolise PCA. Also, additional laboratory information indicates that PCA is metabolised and depurated in various species including fish (Opperhuizen and Voors, 1987; Glickman et al., 1977), earthworms (Haimi et al, a1992; Haimi et al., 1993) and mammals (Vodicnik et al, 1980). In the Opperhuizen and Voors (1987) bioconcentration study on guppies, test concentrations could not be maintained and recovery rates were very low for PCA, as such, a BCF value could not be calculated. However, the authors concluded that chloroanisoles were eliminated rapidly from fish (half-lives for the tetra- and pentachloroanisoles were between 1-4 days). In the Glickman et al (1977) bioconcentration study on rainbow trout, half-lives in the fish tissues were 6.3, 9.8, 23 and 6.3 days in blood, liver, fat and muscle, respectively. PCA was demethylated to PCP.
5. Information on bioaccumulation can also be extracted from several field studies. As an example, Pierce and Victor (1978) observed the accumulation, depuration and biotransformation of PCP and PCA in an aquatic system after a PCP spill. The concentrations in fish decreased as the concentration in the water decreased, but required six to ten months to reach background levels.
6. The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 2009), which surveys chemical concentrations from a nationwide network, detected PCA in both bottom feeding and predator fish, however, the detection frequency was lower in the predators.
7. There is not enough information provided in monitoring studies to calculate true field bioaccumulation and biomagnification factors (BAF and BMF), since residues in biota are low and water concentrations are not often reported in the same studies and the number of studies is small. However, there are four references (Vorkamp et al. 2004; Bentzen et al. 2008; Swachkhammer et al. 1988; Muir, 2013) showing low levels of PCA residues in biota in remote locations. Information collected by Vorkamp et al. (2004), noted that concentrations in top predatory marine mammals (harp seal, narwhal and beluga) do not exceed the concentrations in marine fish. There was no indication of biomagnification in the biota sampled. The authors reported that compared with the results for chlorobenzenes and other chlorinated pesticides, the concentrations of PCA were considered to be very low in Greenlandic biota.
8. In Muir (2013), residues of PCA in biota from remote Canadian Arctic areas are summarised in Table 5-13 of the Supporting document. From 2000-2010, the range of concentrations in polar bears, ringed seals, arctic char, landlocked char, lake trout and burbot are reported to be <0.1-42 ng/g lipid, <LOD-0.82 ng/g lipid, <LOD-0.10 ng/g lipid, <LOD-1.83 ng/g lipid, <LOD-3.85 ng/g lipid, respectively. The information shows a higher range of concentrations detected in polar bears (<n.d.- 42 ng/g lw) than the other marine mammals. The animals were sampled from different parts of the Arctic and during a 10 year time span so it is difficult to compare concentrations with these variables confounding the results.
9. In the terrestrial environment, BAFs for earthworms exposed to PCA and tetrachloroanisoles were reported to range from 5-40 (Haimi et al. 1992 and Haimi et al. 1993). Concentrations in earthworms and soil decreased to low levels in 5 weeks.
10. Vodicnik et al. (1980) determined that following the injection of PCA into female mice, elimination of 14CPCA-equivalents was rapid with half-lives ranging from 5-10 hours in all tissues except for the liver.
11. Ikeda et al. (1994) determined that in the rat, metabolites were eliminated in both urine and feces, with blood elimination half-lives ranging from 6-15 hours. Metabolites included TCHQ, free PCP and conjugated PCP. The systemic bioavailability of PCA following oral dosing was low in both rats and mice, and was unaffected by sex. The systemic bioavailability was considered low due to significant first-pass metabolism of absorbed PCA to PCP by the liver. (Yuan et al. 1993).
12. PCA is not expected to bioaccumulate in humans due to its rapid metabolism (demethylation) to PCP, which is subsequently rapidly metabolised.
13. Thus, despite PCA meeting the indicative BCF criterion for bioaccumulation in the aquatic environment, it is rapidly depurated. In addition, in studies where BCFs exceeded the criterion, there was difficulty keeping test concentrations constant under laboratory conditions. There are measurable residues in field biota, but biomagnification has not been observed in the cited studies for the aquatic or terrestrial environment. In several studies, residue levels in predators were lower than in its prey.

##### 2.4 Potential for long-range environmental transport

1. PCP is a relatively volatile compound, while its sodium salt is non-volatile. Based on Henry’s law constant, it has the potential to volatise from water or moist soils. In the atmosphere, volatilized PCP may undergo photolysis or may react with photochemically produced hydroxyl radicals. Although the laboratory derived half-lives based on reactions with OH-radicals indicate a low potential for long range transport [(photolysis half-life of 12-44 hours in air (Sloof et al., 1991)], PCP has been detected in particulate matter in air. Atmospheric PCP associated with particulate matter or moisture will be subject to wet and/or dry deposition.
2. The Henry’s law constant of PCA indicates that PCA will likely volatize rapidly from water. The volatilization half-life from a model river (1 m deep, flowing 1 m/sec, wind velocity of 5 m/sec) is estimated to be 2.2 hours (EPIWIN, U.S. EPA, 2011). The volatilization half-life from a model lake (1 m deep, flowing 0.05 m/sec, wind velocity of 0.5 m/sec) is estimated as 6.9 days. Also, volatilisation of PCA has been observed in several laboratory studies using liquid medium (Badkoubi et al. 1996, Walter et al. 2004, Lamar et al. 1990). A QSAR estimate of the phototransformation half-life of PCA in air is estimated to be 9.8 days (U.S. EPA 2011).
3. Monitoring data show the presence of both PCP and PCA in air. Modelling calculations predict PCP transport over considerable distances, and it was occasionally also reported in more remote locations. For example, PCP was detected in samples of air taken in mountains in the La Paz region (Bolivia) at 5200 m above the sea level with concentrations from 0.25 to 0.93 n/m3 (ATSDR 1998, cited in Czaplicka 2004). PCA is generally found at higher concentrations and more frequently than PCP. Swedish air monitoring information reported PCA, a transformation product of PCP, detected at higher levels than PCP. PCA has been monitored at Alert since 1993 (details given in Section 2.5.1 below). The research station at Alert is part of Canada’s National Implementation Plan for Arctic Monitoring and Assessment Programme (Hung 2013, Fellin et al. 1996, Hung et al. 2010, Su et al. 2011, Barrie et al. 1998). There is also evidence of long range transport of PCA by sorption to fine soil particles (details in Section 2.5.1).
4. Although it is possible that PCA can be produced from local sources (i.e., transformation of HCB or PCP), the observations of PCA in Arctic air and snow as well as the QSAR estimate for the half-life in air (9.8 days), indicate that PCA is persistent in air and can be transported to remote locations. Although there is uncertainty regarding the transport of PCP itself to remote locations, PCP may be transported sorbed to fine particles or may form after transportation of PCA or other organochlorine substances.

##### 2.5 Exposure

###### 2.5.1 Environmental monitoring data

1. PCP is a metabolic transformation product of HCB, PCNB and lindane in a variety of different organisms including fungi, caterpillars, fish, rats, birds, monkeys and humans (Supporting document). The presence of PCP and PCA in remote regions is likely due to several sources, including the transformation of other organochlorine substances, which has to be taken into account when interpreting monitoring data.

**PCP in Abiotic Compartments**

1. Borysiewicz (2008) compiled levels of PCP from various European sources. Concentrations of PCP in European river waters have declined since early 1990, when marketing and use restrictions were first implemented (Euro Chlor 1999, in Borysiewicz 2008). Concentrations in rivers from the Netherlands, Germany and Belgium ranged from 0.01 to 0.17 μg/L from 1990 to 1997. The Seine River in France had an average concentration of 0.03 μg/L in 1995. Concentrations in the UK in 1990 to 1992 showed slightly higher concentrations, however, the median levels of PCP were below 1 μg/L; concentrations were higher in industrial areas (one site had a concentration of 40 μg/L). However, between 1994 and 1996, concentrations were considerably lower (0.15, 0.20, 0.02 μg/L in 1994, 1995, 1996, respectively), reflecting the use restrictions.
2. Monitoring data showed that PCP concentrations generally decreased between 1988 and 1993 in the River Elbe. This was attributed to the cessation of PCP production in Germany in 1986 and use ban in 1989. However, an increase was observed in the Rhine River and its tributaries in 1990-1991 vs 1980-1989 (Borysiewicz 2008).
3. In the marine environment, concentrations of PCP ranged from non-detect to 0.79 μg/L for the period 1983 to 1997 (average/median concentrations were below 1 μg/L) in the North Sea, coastal waters and estuaries of Germany, Netherlands and the UK (Euro Chlor in Borysiewicz 2008). In estuary waters, concentrations generally show a decreasing trend between 1983 and 1997 at all monitoring sites. Between 1983 and 1997 a “typical concentration” for coastal and marine water was estimated to be 0.07 μg/L.
4. Between 1994 and 1998 a median PCP concentration of 0.0706 μg/L (n=2,296 from 85 sample sites) was found in EU Member States in the context of the EC Water Framework Directive.
5. Surface water used for drinking water in the U.S.A. contained a range of 0.04 to 1 μg PCP/L (mean 0.4052 ± 0.4355 μg/L) (U.S. EPA 2001a, in Borysiewicz 2008). Concentrations of PCP in ground water ranged from 0.04 to 1.64 μg/L (Mean 0.459 ± 0.444 μg/L).
6. Hoferkamp et al. (2010) does not report detections of PCP in water in the Canadian Arctic but PCA was detected (see below).
7. Inland surface waters and other surface waters in Estonia had annual average concentrations of PCP of 0.4 μg/L (the LOQ).
8. Zheng et al. (2011) in their summary of PCP studies found that the most recent studies (1991 to 1996) they reviewed for freshwater from various countries (Belgium, Germany, UK, Netherlands and France) had average concentrations ranging from 0.01 to 0.169 μg/L. Average concentrations in marine water between 1993 and 1997 ranged from 0.001 to 0.012 μg/L in samples from the Netherlands and UK. Concentrations in precipitation ranged from <0.002 to 0.01 μg/L. Concentrations of PCP in the Niagara River and St. Lawrence River in Canada ranged from <0.2 to 21 ng/L.
9. Health Canada (2010) reports that water monitoring data on heavy duty wood preservatives (HDWPs) in Canada was limited. There were some detections of PCP in Manitoba, but no information was provided to link the detections to the use of heavy duty wood preservatives.
10. Data for PCP in water from the international convention for the Rhine provide annual data. Concentrations of PCP in the Rhine River range from 0.006 to 0.1 ng/L between 2000 and 2011.
11. Cessna et al. (1997) monitored PCP/PCA in air near Yellowknife and in Saskatchewan, Canada. Corresponding concentrations were 0.43 to 3.68 ng/m3 (mean: 1.53 ng/m3) in 1994 and 0.06 to 0.58 ng/m3 (mean 0.30 ng/m3), respectively. Because the analytical methodology in this study used diazomethane as a derivatizing agent, the authors could not differentiate between PCP and PCA.
12. In their summary of PCP studies, Zheng et al. (2011) found that more recent concentrations of PCP in outdoor air from urban areas in Canada and the United States ranged from not detected to 1233 pg/m3 (1995-2001). Concentrations generally ranged though from not detected to 51.5 pg/m3.
13. PCP was detected in air in New Zealand 7 years after it was banned in that country (Ministry for the Environment, New Zealand 1998). It was concluded that it was most likely due to historic use as a timber preservative.
14. Other studies of organochlorine compounds in air samples from remote locations (e.g., Su et al. 2008), do not report detection of PCP.
15. In a Swedish study (IVL Report B1474, June 2002, report not provided), samples were collected for analysis of PCP in air, soil, sediment and sludge and biota. The results show that the environmental levels of PCP in Sweden are generally lower than recommended quality limit values (details not provided). Slightly higher concentrations were detected near some potential point sources, but these were still below proposed critical levels. In air, PCA was detected in higher levels than PCP. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge but were comparable in biota. Sweden stated that “it is likely that possible long-range transport of PCP occurs in the form of PCA.”
16. Sediment showed a median PCP concentration of 15.5 μg/L (sic) (n= 66, from 20 sample sites) (Fraunhofer Institut 1999, in Borysiewicz 2008) within the EU Member States in the context of the EC Water Framework Directive between 1994 and 1998.
17. Sediment in the River Narva, Estonia and two points in Lake Peipus (Estonia) remained below the limit of quantification (0.1 μg/kg dw) and PCP was not detected in coastal sediment (2010 data).
18. In their summary of PCP studies, Zheng et al. (2011) found that more recent (1991-1996) average concentrations of PCP in sediment had a range of 0.9 to approaximately 40 μg/kg dw. Studies were from the UK, Netherlands, Germany and France. Temporal patterns are difficult to make as there are insufficient data.
19. PCP was not detected in marine sediment from Norway between 2004 and 2008.
20. Measurements in the Elbe River taken in the framework of the International Commission for the protection of the River Elbe show an overall decrease in contamination levels from 1997 to 2010 as well as differences in contamination levels between various sites (IKSE 2010).
21. Soils close to sawmills that used PCP heavily are still highly contaminated with PCP many years after use was discontinued (Salminen et al 1995). Researchers found that there was no significant decrease of PCP in highly contaminated soil from a high use site up to five years after the last use; especially in cold northern climates (Kitunen et al. 1987).

**PCP in Biota**

1. PCP has been detected in birds eggs from Norway at concentrations ranging from <LOQ to 1350 pg/g ww (Berger et al 2004).
2. PCP levels have been reported in polar bear, fish, and other Arctic biota. Hoekstra et al. (2003) found that PCP was the most abundant halogenated phenolic compound found in the Arctic bowhead whale plasma. The authors suggested that the PCP could be present as a result of the biotransformation of PCA or HCB.
3. The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 2009) did not detect PCP during 4 years of the study (2000-2003).
4. Letcher et al. (2009) found PCP/PCA at mean concentrations of 1.0±0.4 ng/g lw in all ringed seals sampled from East Greenland in 2002 and in similar concentrations in polar bears. The results of this study should be interpreted with caution given that PCP is a major metabolite of HCB in animals and the analytical method entailed methylation using diazomethane that would have made it impossible to differentiate between PCA and PCP.
5. Pine needles in Saskatchewan, Canada, contained PCP/PCA at concentrations ranging from 0.42-2.08 ng/g in 100% of their samples (Thompson and Treble, 1995) and in Europe ranged from 0.09 to 1.39 ng/g fw (Eriksson et al. 1989). The extraction methods in these studies included a diazomethane derivatization step, therefore, they were unable to differentiate between PCA/PCP.
6. PCP was not detected in blue mussel and cod liver in Norway between 2004 and 2008 (UNEP-POPS-POPRC7FU-SUBM-PCP-Norway-120702.En[1]).
7. Darnerud et al. (2006) estimated the mean Swedish per capita intake of selected POPs and compared the results with a previous intake estimation from 1999. PCP was only found in fish and meat above the LOQ.

**PCP in Human Biomonitoring**

1. PCP is detected in air, water, and soil throughout the world, as well as in the blood, urine, seminal fluid, breast milk and adipose tissue of humans (Zheng et al. 2011b).
2. PCP has been reported as the dominant chlorinated phenolic compound in blood from Nunavik (Inuit) and southern Quebec adults in Canada (Sandau 2002), in human milk from women in Bratislava (median concentration of 2.21 μg/kg whole milk) (Veningerova et al. 1996), in blood serum of pregnant and lactating women in Sweden (up to 3 ng/g serum wet wt.) (Larsdotter et al. 2005), and within a representative population of women in Norway sampled in 2004 (711 ng/L w.w.) (Rylander et al. 2012). Reported levels in blood were in the same range as samples from Canadian Inuit (801 ng/L, n=567), and in infant cord blood from Slovakia (Park et al. 2008).
3. Concentrations of PCP in umbilical cord blood in Quebec Canada ranged from 628 to 7,680 pg/g ww plasma. Concentrations were not affected by the location of the samples, with similar concentrations in Inuit from northern Quebec and in southern populations from Québec City.
4. PCP accounted for up to 85% of the total quantified phenolics found in human blood serum in Belgian samples and 35% in Romanian samples (Dirtu et al. 2010). Sandanger et al. (2004) found levels of PCP in blood plasma of the Indigenous Chukotka people of the Russian Arctic. The median PCP level was 642 pg/g plasma. Concentrations in blood are generally within the same range as samples from Canadian Inuit (801 ng/L, n=567). PCP has been detected in blood serum of 4 year old children in urban and rural Spain (means = 6.4 ng/mL and 0.61 ng/mL, respectively).
5. Wilson et al. (2007) found PCP in urine from children in 257 randomly selected households and daycare centers in Ohio (mean = 0.605 ng/mL) and North Carolina (mean = 1.27 ng/mL), U.S.A. It has been detected in urine in other studies as well (Cooper and Jones 2008, Hill et al. 1989).
6. Bradman et al. (2003) detected PCP in amniotic fluid of women in California (USA), indicating direct exposure to the fetus. Guvenius et al. (2003) found concentrations of PCP in maternal blood plasma, cord blood plasma, and breast milk samples with median levels of 2.83, 1.96, and 0.02 ng/g fresh weight, respectively (n=15) and indicated that the fetus is likely to be continuously exposed during development. PCP levels in maternal and cord blood plasma were 30 and 36 times higher than the sum of OH-PCBs on average.
7. Sjödin et al. (2000) determined PCP in blood plasma in Latvian and Swedish men and compared this with their consumption of fish as a possible factor influencing uptake of PCP. The PCP level in plasma was inversely related to fish consumption and statistics showed that it was not affected by age, but was strongly correlated with the country in which the subjects lived, with the PCP levels being much lower in Latvia than in Sweden. Consumption of fish was not a major source of exposure to PCP.
8. Fréry et al. (2013) report biological concentrations of contaminants in a representative sample of the French population. PCP was detected in 66.2% of urine samples (LOQ: 0.03 to 0.1 µg/L). The mean and median concentrations were 0.88 µg/g of creatinine (0.90 µg/L) and 0.90 µg/g of creatinine (0.85 µg/L), respectively. None of the values exceeded the German toxicological value HBM-II for PCP (30 µg/L (40 µg/g of creatinine)) or HBM-I for PCP (25 µg/L). One person, however, did exceed the HBM-I limit when the value was expressed on a creatinine basis (20 µg/g).
9. The German Environmental Survey for Children 2003/06 - GerES IV - Human Biomonitoring (Becker et al. 2008) reported on the levels of PCP in urine of children aged 3-14 years. They found that no measured variable was seen to affect the concentrations of PCP in urine of children (ages 3-14). Combining the data, concentrations of PCP in urine ranged from <0.60 to 9.71 μg/L with a detection frequency of 49% and a geometric mean of <0.6 µg/L.
10. Schulz et al. (2007) summarized the PCP data for German children (GerES studies) for the samples taken in 1990/1992 and those taken in the years 2003/2006. They found that PCP levels in children during 1990-1992 were statistically significantly higher in West German children compared to East German children, however, by 2003/2006 that difference had disappeared. Overall (combined data from the former East and West German countries), concentrations of PCP in children decreased from 1990/92 to 2003/2006 from geometrical means of 4.5 μg/L to <0.6 μg/L, respectively.
11. Schulz et al. (2007) also shows that concentrations of PCP in urine in adults aged 25-69 years old from the former West Germany have decreased during the sampling periods 1985/86, 1990/92, and 1998 from 4.4, 2.7 and 1.1 μg/L, (geometric means) respectively. Sampling also took place in the former East Germany during the 1990/92 and 1998 sampling periods, but there were no statistical differences between the two regions.

**PCA in Abiotic Compartments**

1. PCA has been detected in air, water, sediment and snow from many areas around the globe including impacted sites such as industrial rivers as well as remote areas such as the Canadian and Russian Arctic. PCA has most often been detected in air compared to other abiotic compartments.
2. PCA is one of the more abundant high molecular weight halocarbons in the remote marine troposphere. In earlier studies from the 1980’s and 1990’s, levels of PCA in the South Pacific Ocean (American Samoa) in the northern hemisphere were on average 9.0 pg/m3, while those in the southern hemisphere (New Zealand) were 2.1 pg/m3 (Atlas et al, 1986). Air samples collected on a cruise on the Atlantic Ocean between 50’N and 50’S ranged from 1.8 to 40 pg/m3 (Schreitmuller et al., 1995).
3. Time trend data are available for PCA in Arctic air. In the Canadian High Arctic, Hung et al. 2012 measured PCA concentrations ranging from 2.6-4.0 pg/m3. Current monitoring information shows mean concentrations of PCA in air are generally below 5 pg/m3 in Arctic air at Alert, NU, Canada (a high Arctic site). There appeared to be a strong seasonal gradient, with concentrations peaking in winter and spring. However, the winter/spring maxima appear to have decreased in recent years (2007-2009) and tend to be more episodic with less seasonal variability. Concentrations have shown a decline in the years 2003-2009 from a period of relatively consistent concentrations (1993-2002) (Hung et al. 2013, Fellin et al. 1996, Hung et al. 2010, Su et al. 2011, Barrie et al. 1998). The actual cause of atmospheric concentration decline and changes in seasonal variability of PCA at Alert is unclear.
4. PCA has also been reported at several Arctic monitoring stations in Canada, the USA, and Russia with different seasonal profiles (Su et al 2008). Three episodes of elevated PCA concentrations were observed in June–August 2002 at Point Barrow, Alaska. Corresponding back-trajectories indicated that the air masses largely originated from the Eurasian portion of the Arctic Ocean or the Russian Arctic. Overall, mean and median concentrations of PCA measured at the Arctic monitoring stations were 4.9 and 3.8 pg/m3, respectively, which were comparable to those of γ-HCH and endosulfan I.
5. In a Swedish study (IVL Report B1474, June 2002, summary in English), samples were collected for analysis of PCP and PCA in air. PCA was detected at higher levels than PCP.
6. PCA has been detected in sediment from impacted areas (Mississippi River, US.A; Yangtze River, China; Alexandrian Harbour, Egypt, and the Yellow Sea) as well as remote areas as the Canadian Arctic. Concentrations were below 7.4 ng/g in all areas (Supporting document Table 5-15).
7. Concentrations of PCA in a dated sediment core from Lake Hazen, NU, Canada (high Arctic) collected in 2006 ranged from <DL to 0.523 ng/g dw (Muir, 2013). The sediment core encompasses a time series from 1898 to 2005. Concentrations were much higher in the upper layers compared to the lower layers. Analysis of HCB in the sediment core also demonstrated that HCB and PCA concentrations track each other quite well in the upper sediment layers (2001-2005), however, that trend was not apparent in deeper sediment layers (prior to 1996). Decreased concentrations in the lower sediment layers could be a result of mineralization of both HCB and PCA over time.
8. Only two studies have shown PCA in soil (Finland and Sweden). In a Swedish study (IVL Report B1474, June 2002, summary in English), samples were collected for analysis of PCP and PCA in soil, water, sediment, and sludge. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge. Actual concentrations were not included in the information submitted by Sweden.
9. Concentrations of PCA in soil of the Taurus Mountains ranged from a low of 1.44 pg/g dw at 121 m to a high of 6.02 pg/g dw at 1881 m (Turgut et al. 2012). There was no correlation with altitude or any other variable that they measured (soil characteristics).
10. Only two studies have shown PCA in snow. A brown snow event in the Canadian Arctic (Welch et al. 1991) had very high concentrations. Air mass trajectories, clay mineral composition, soot particles and visible organic remains indicated the source the long-range transport of fine particles, most likely from Asia. PCA has also been found in snow from the Devon ice-cap in northern Canada (Muir 2007 in Hoferkamp et al. 2009).
11. PCA is rarely found in water. In fact, only one study (Jiang et al. 2000) has shown PCA in water and this was from an impacted area of the Yangzte River, China.

**PCA in Biota**

1. Information on residues in biota has been reported previously in UNEP/POPS/POPRC.7/INF/5 UNEP/POPS/POPRC.7/INF/5/Add.1 and shows that concentrations of PCA have been found in aquatic biota in remote areas. However, four studies (Vorkamp et al. 2004; Bentzen et al. 2008; Swackhammer et al. 1988; Muir 2013 (see shaded rows) in Supporting document Table 5-16) show low level residues in biota at or below detection limits in remote locations.
2. The National Study of Chemical Residues in Lake Fish Tissues (USEPA 2009) detected PCA in both bottom feeding (range = <MDL to 9 ng/g) and predator fish (range = <MDL to 4 ng/g), however, the detection frequency was lower in the predators (years 2000-2003).
3. The concentrations of PCA in biota from the remote Canadian Arctic between 2000-2010 range from <0.1-42 ng/g lipid, <LOD-0.82 ng/g lipid , <LOD-0.10 ng/g lipid, <LOD-1.83 ng/g lipid, <LOD-0.35 ng/g lipid and <LOD – 3.85 ng/g lipid, in polar bears, ringed seal, arctic char, landlocked char, lake trout and burbot, respectively (Muir 2013). The information shows a slightly higher range of concentrations detected in polar bears (<n.d.- 42 ng/g lw) than the other marine mammals. The animals were sampled from different parts of the Arctic and during a 10-year time span so it is difficult to compare concentrations with these variables confounding the results.
4. A study from Greenland shows bioaccumulation of PCA in a range of species varying from aquatic invertebrates to fish, birds and mammals (Vorkamp et al, 2004). However, the concentration of PCA found in these different trophic levels showed no evidence of biomagnification. Compared with the results for chlorobenzenes and other chlorinated pesticides, the concentrations of PCA were considered to be low in biota.
5. PCA is detected frequently in Arctic biota at low concentrations. In aquatic food webs, concentrations in top predatory animals do not exceed concentrations in lower trophic organisms.

##### 2.6 Hazard assessment

1. PCP acts by uncoupling oxidative phosphorylation, inhibiting ATP pathways important to respiration in both animal and plant cells. Moreland and Hilton (1976) described pentachlorophenol as a more general inhibitory uncoupler. They suggest that it has several sites of action, including phosphorylation, protein synthesis and lipid biosynthesis (Morrod 1976). All of the mechanisms of PCP’s toxicity have not been precisely defined, but may generally involve the disruption of cellular membranes (Jayaweera et al. 1982; Senger and Ruhl 1980; and Smejtek et al. 1983).
2. PCA is not industrially produced and, therefore, not well studied. There is only limited data available dealing with its toxicity. When assessing the toxicological potential of PCA, it should be considered that PCA can be demethylated back to PCP in living organisms. The principal route of PCA metabolism in mice, rats, rabbits and fish is demethylation to PCP (Glickman et al., 1977; Ikeda et al., 1994 and Vodnick et al., 1980). Therefore, toxicity information for PCP is considered relevant for PCA.

###### 2.6.1 Adverse effects on aquatic organisms

1. On an acute basis, both PCP and PCA are very highly toxic to aquatic organisms (Supporting document Table 5-18 and Table 5-19). Acute LC50 values for fish ranged from 20 µg/L to 600 µg/L PCP and 650 µg/L and >1.2 mg/L for PCA. Acute LC50 values for invertebrates ranged from 240 µg/L to 2,000 µg/L PCP and 10 to 27 µg/L for PCA. Sublethal effects to aquatic organisms were reported in the 10-100 µg/L range for PCP (Supporting document Table 5-18). The sublethal effects observed include reproduction, survival, growth, and effects on T3 activity. The lowest sublethal endpoint is reported by Orton et al. 2009 who noted slight elevations in plasma progesterone levels and degenerative ovarian features in adult *Xenopus laevis* at 0.1 and 1µg/L.
2. The similarity of effects thresholds in the aquatic environment between the two substances likely represents biotransformation of PCA to PCP within test organisms. PCA, untransformed within an organism, is likely less toxic than PCP because this methylated version of PCP loses its phenolic functionality.

###### 2.6.2 Adverse effects on terrestrial organisms

1. PCP is highly toxic to mammals, practically non-toxic to highly toxic to birds. Acute oral LD50 values for rat were 50-220 mg/kg bw and acute oral LC50 values were 80-177 mg/kg bw for both rats and mice. In birds, the 5-day dietary LC50 value in Japanese quail is greater than 5139 mg/kg (Hill *et al.* 1975). LC50 values reported by Hill *et al.* (1975) for northern bobwhite, pheasant and mallard duck varied between 3400 and 4500 mg/kg food. Reported acute oral LD50 values for PCP are 380 mg/kg BW in mallard duck and 504 mg/kg BW in pheasant (Hudson *et al.* 1984). Sublethal effects such as a reduction in hatching of eggs were noted at feeding rates ≥50 mg PCP/kg diet (Stedman et al. 1980;Dorrestein and Zelle 1979). Effects on thyroxine levels in mink and sheep fed 1-2 mg/kg bw were also reported (Beard and Rawlings 1998; Rawlings et al. 1998; Beard and Rawlings 1999: Beard et al. 1999: Beard et al. 1999a) (Supporting document Table-5-17)
2. PCA is also toxic to mammals. Acute oral LD50 values were 318-331 mg/kg bw in mice. The intraperitoneal LD50 values were 281 (m) and 293 (f) mg/kg for PCA (Renner et al., 1986).The LC50 value was ≥ 500 µg/g for earthworms (Supporting document Table 5-18). No information was found on the toxicity of PCA to birds and no information on the chronic toxicity of PCA was found. Given that PCA biotransforms to PCP within rats, mice, rabbits and fish and that the principal route of PCA metabolism is demethylation to PCP (Vodnick et al., 1980; Ikeda et al., 1994; Glickman et al., 1977), effects thresholds are expected to be similar.

###### 2.6.3 Adverse effects on human health

1. Humans may be exposed to PCP through dermal, inhalation, and oral routes, including diet. Most available information on human effects is based on occupational exposures and epidemiological data. A very large, high quality laboratory animal study database submitted in support of pesticidal use registration petitions support the identification of potential hazards and risks of PCP exposure in humans. In addition, there is a robust body of independent academic public literature.
2. In humans, high acute exposure to PCP can cause elevated temperature, profuse sweating, dehydration, loss of appetite, decreased body weight, nausea, and neurological effects such as tremors, uncoordinated movement, leg pain, muscle twitching, and coma. Occupational exposure in wood treatment facilities also noted skin irritation/blistering, irritant effects on the eyes and in the airways, loss of appetite and bodyweight, fainting, rapid heart rate, and death. Human studies showed that immune response was impaired in patients who had blood PCP levels >10 μg/L and in particular in those whose levels were >20 μg/L (Daniel et al., 1995; McConnachie and Zahalsky, 1991). Daniel et al. (2001) found immunological abnormalities associated with plasma levels of PCP in individuals with long-term low-dose exposure, including significant associations with cellular and humoral immunodeficiencies. Some studies indicate that PCP may affect the function of the thyroid in humans (Dallaire, et al 2009, Sandau, et al 2002).
3. Human biomonitoring studies have detected PCP in a variety of body tissues, as well as in amniotic fluid, cord blood, and mother’s milk, demonstrating exposure, and therefore potential hazard, to fetuses, infants and adults. However, concentrations of PCP detected in Fréry et al. (2013) and reported in the Human biomonitoring section did not exceed the HBM-I limit when the value was expressed on a creatinine basis (20 µg/g).
4. Epidemiological and industrial health studies have shown an association between PCP exposure and a variety of health effects, many in common with animal studies. A number of epidemiological studies, primarily based on inhalation and dermal exposures, have made associations with a variety of cancers, including non-Hodgekin’s lymphoma, multiple myeloma, soft tissue sarcoma, and liver cancer (U.S. EPA 2010). In laboratory animal studies used for human health risk assessments (U.S. EPA, 2010), the major target organs for PCP are the liver, kidneys, and central nervous system. Some of the effects of exposure to commercial grade PCP are attributable to microcontaminants present in the technical preparation.
5. In acute toxicity studies PCP is moderately toxic via the oral, inhalation, and dermal routes. PCP causes irritation to the mucous membranes, skin, and eyes.
6. In laboratory animals, repeated exposure by all routes and durations results in liver toxicity. The most sensitive endpoints in repeated dose animal toxicity studies relate to liver toxicity, with the most sensitive species being the dog (U.S.EPA, 2010).
7. The majority of developmental toxicity studies on PCP provided no evidence of teratogenic effects in either rats or rabbits. In rats, exposure to PCP causes adverse reproductive effects, including decreased fertility, delayed puberty, testicular effects, decreased litter size, decreased viability, and decreased pup weights.
8. Other important toxic effects in laboratory animals for PCP include disruption of thyroid homeostasis. Available data do not allow for determination of the mechanism involved in the effects on T3, T4, and TSH following exposure to PCP (U.S. EPA 2010). Since thyroid hormones are important in neurodevelopmental processes, the disruption of thyroid homeostasis is a potential hazard for the normal development of the nervous system. PCP may also affect other endocrine systems through interaction with receptors or alteration of non-thyroid hormone levels (U.S. EPA 2010). EFSA has recently (EFSA 2013) concluded that risk assessment makes the best use of available information and, provided certain criteria are met, substances which affect endocrine systems can be treated like most other substances of concern for human health; i.e., be subject to threshold based risk assessment and not only hazard assessment, an approach which aligns with other international regulatory authorities.
9. PCP has been shown to adversely affect the immune system in several animal species. Neurotoxic effects have also been reported in *in vitro* systems, as *in vivo* changes in brain tissue, and from physical tests in animals.
10. PCP is considered non-mutagenic, although the tetrachlorhydroquinone (TCHQ) metabolite of PCP showed positive mutagenic effects is some tests. PCP is considered carcinogenic by all routes of exposure. Various countries and organizations have classified PCP carcinogenicity as follows: (IARC (1991): possibly carcinogenic to humans (Group 2B); European Union (EC, 1993) classification R40: limited evidence of a carcinogenic effect; U.S. classification: likely to be carcinogenic to humans).

**PCA**

1. Orally administered PCA is rapidly demethylated to PCP in rats, mice and rabbits. Metabolites were eliminated in both urine and feces, with blood elimination half-lives ranging from 6-15 hours. Metabolites included TCHQ, free PCP and conjugated PCP. Bioavailability of PCA was low in both rats and mice and was sex independent. PCA is not expected to bioaccumulate in humans due to its rapid metabolism (demethylation) to PCP, which is subsequently rapidly metabolised and eliminated.
2. While some assays produced negative results, several others suggested that PCA is genotoxic. PCA was associated with an increased incidence of benign pheochromocytomas (adrenal tumours) in male rats, and increased incidences of benign pheochromocytomas (adrenal tumours) and hemangiosarcomas (rapid invasive growing cancer of the liver) in male mice.
3. Chronic PCA administration was associated with increased incidences of adrenal medulla hyperplasia (increased cell growth) in female rats, and increased incidences of pigmentation in the renal tubule epithelium, olfactory epithelium, and hepatocytes of male and female rats. In addition, there were increased incidences of adrenal medulla hyperplasia and hypertrophy, and hepatocellular mixed cell foci in male mice. In male and female mice, increased incidences of hepatocellular cytologic alteration, Kupffer cell pigmentation, biliary tract hyperplasia, and subacute inflammation were noted.
4. Reproductive toxicity in rats is manifested as decreased corpora lutea and increased embryolethality. Reductions in male fetal body weight and crown rump length of males were noted.

##### 2.7 Environmental Concentrations and Effects

1. The risk of PCP to the environment has been reviewed by several national regulatory authorities and is well established. Contaminated sites continue to be of concern and there is much effort worldwide to remediate these sites. Strict regulations have been implemented to minimise exposure in countries where PCP is still in use.
2. The environmental fate of PCA indicates that it is expected to be found primarily in soil, sediment, air and biota. This is supported by the information submitted by Sweden (IVL Report B1474, June 2002, report not provided), where samples were collected for analysis of PCP in air, soil, sediment and sludge and biota. The results show that the environmental levels of PCP in Sweden are generally lower than recommended quality limit values (details not provided). Slightly higher concentrations were detected near some potential point sources, but these were still below proposed critical levels. In air, PCA was detected in higher levels than PCP. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge but were comparable in biota.
3. The only water concentration reported for PCA was 0.6 ng/L from an impacted area of the Yangtze River, China. This value is below the most sensitive sublethal endpoint reported for PCP or PCA (0.1 µg/L, Orton et al. 2009). It is also below the WHO provisional drinking water guideline of 9 µg/L for PCP (WHO, 2003). No concentrations for PCP or PCA in water were reported for remote areas, however, concentrations are expected to be lower than in more populated areas.
4. Information was also available on tissue concentrations in biota. Based on the residues measured in animal tissues, potential adverse effects were characterised using a critical body residue method (McCarty and MacKay,1993). In their review of internal toxicity thresholds for baseline narcotic and reactive chemicals, McCarty and MacKay (1993) reference critical body burdens of 0.08 mmol/kg for chronic and 0.3 mmol/kg for acute exposures specifically for PCP. The median internal threshold for narcosis is about 5 mmol/kg.
5. There is a 3-fold margin of exposure between the highest measured historical concentration in fish (1980-84), 100 ng/g (0.028 mmol PCA/kg) (Schmitt et al., 1990), to critical body burden estimates for PCA (0.08 mmol PCA/kg). Tissue residues reported for other sites and in particular, Arctic biota were much lower <1-10 ng/g (0.00028-0.0028 mmol/kg), which indicates there is a 30-fold margin of safety for PCA. This information is reported in the information document (Supporting document, Table 5-21).
6. Although few data are available, reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect, particularly in remote areas.
7. Human biomonitoring studies have detected PCP in a variety of body tissues, as well as in amniotic fluid, cord blood, and mother’s milk, demonstrating exposure, and therefore potential hazard, to fetuses, infants and adults (section 2.5.1). Fréry et al. (2013) reports PCP was detected in 66.2% of urine samples (LOQ: 0.03 to 0.1 µg/L) in the French population. But none of the values exceeded the German toxicological value HBM-II for PCP (30 µg/L (40 µg/g of creatinine)) or HBM-I for PCP (25 µg/L). One person, however, did exceed the HBM-I limit when the value was expressed on a creatinine basis (20 µg/g).
8. When human biomonitoring information for PCP was compared between remote and more populated areas (Parks et al. 2008; Rylander et al 2012; Sandau 2002), levels were found to be similar. Sjödin et al. (2000) determined that in Sweden and Latvia, consumption of fish was not a major source of exposure to PCP; levels of PCP in blood plasma were related to the country where the person lived.
9. A suitable dietary human health risk assessment for PCP and PCA cannot be developed for people that are exposed to these compounds via their diet (traditional Inuit foods) because residues in their foods are not known, their diets are not well characterized and the toxicological database on PCA is lacking. However, the occupational exposures in wood treatment plants evaluated in U.S. EPA (2010) are expected to be much greater than incidental environmental or current dietary exposures to PCP based on general population exposure information available in ATSDR (2001). It should be noted that lifetime cancer risks were evaluated and included in the assessment.

# 3. Other Considerations

1. Historical uses and misuses of PCP have resulted in contaminated sites worldwide. As an example, concentrations of PCP in soil close to sawmills that used PCP heavily are still highly contaminated many years after use was discontinued (Salminen et al 1995). Researchers found that there was no significant decrease of PCP in soil up to five years after the last use; especially in cold northern climates (Kitunen et al. 1987).
2. Contaminated sites may also have high levels of dioxins and furans due to the release of contaminants in PCP products. The Swedish Environmental Protection Agency (2009) did an extensive review of the role of PCP treated wood for emissions of dioxins into the environment based on the widespread uses and high dioxin levels in PCP products in Sweden prior to 1978. Concentrations of dioxin and furans, present as impurities, decreased after legal measures were taken in the U.S. and Europe between 1987 and 1999. In the European Union, a maximum allowable limit of 4 ppm for total hexachlorodibenzo-p-dioxin (HCDD) was set in 1992. This limit was further reduced to 2 ppm in 2000. Current levels in Canadian technical products reported in Appendix II of the Supporting Document indicate that the total HCDD and total dioxins/furans are 0.4 ppm and 0.8 ppm, respectively (TEQ calculated as per the WHO 2005 factors in Van den Berg et al. 2006). Currently, dioxins and furans should be controlled by countries as they are listed in the Convention.
3. PCA is considered a semi-volatile organochlorine substance (SOC) and two studies indicate that oceans may be a major continuous source in air. Schreitmuller and Ballschmiter (1995) indicated that particularly under conditions of a diminishing input of SOCs from continental sources, the air-surface water equilibrium will render the oceanic system to be a global nonpoint source of anthropogenic compounds in marine air. Similarly, Hoferkamp et al. (2010) indicated that with the exception of lindane and α-endosulfan (Weber et al., 2006) there is insufficient data to assess whether air concentrations are resulting in net deposition to Arctic Ocean and lake waters or whether these waters are actually outgassing the currently used pesticides (including PCA) monitored in the Arctic.

# 4. Synthesis of information

1. PCP was first introduced as a wood preservative in the 1930’s and has a variety of other applications (biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent, wood preservative and textiles). PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst. Contaminants including hexachlorobenzene, dioxins and furans are produced during the manufacturing process.
2. Historically, production has been estimated to be as high as 90 000 tonnes of PCP per year. Many sites are contaminated from the historical use of PCP and from improper practices (e.g., spills from industrial holding ponds from wood treatment facilities prior to the implementation of strict regulations).
3. PCP has either no uses or is banned in all E.U. member states, India, Indonesia, New Zealand, Russia and Switzerland. PCP is only allowed for wood preservation with additional restrictions and/or regulations in Belize, Canada, China, Mexico and the United States. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico. Other uses include ready-for-use products in Nigeria; and snail elimination to control the spread of schistosomiasis in China.
4. The only manufacturing site for North America is in Mexico which the Wood Preservation Industry indicated produced 7 257 tonnes/year in 2009 for the United States, Canada and Mexico.
5. There are several sources of PCP in the environment, including the release of PCP when used in accordance with currently registered uses as a wood preservative as well as the contaminated sites from historical uses. PCP is also a transformation product and metabolite of other organochlorines such as HCB (hexachlorobenzene), HCH (lindane) and PCNB (quintozene). The presence of PCP and subsequently PCA in the environment is also as a result of these other sources.
6. Under aerobic conditions, large numbers of PCP-degrading bacteria have been identified and there are several pathways for degradation of PCP, depending on the experimental or environmental conditions. In anaerobic conditions reductive dechlorination is the major pathway of degradation. Laboratory half-lives indicate that PCP is degraded rapidly. However, PCP can persist for many years at contaminated sites where the levels of PCP exceed the toxicity threshold of soil microorganisms.
7. PCA is a transformation product of PCP formed primarily under aerobic conditions in soil and sediment. PCA cannot be formed in abiotic compartments or within biota. In test systems, PCA is constantly formed from PCP and is demethylated back to PCP which can confound half-life estimates and make half-lives look longer than they actually are. Calculated half-lives of transformation products need to be corrected for the rate of formation. Most reported and/or estimated half-lives are below persistence criteria thresholds, but there are uncertainties with these values.
8. PCA is likely subjected to long range transport to remote locations as evidenced by the predicted and observed volatility in laboratory studies, as well as detections in air in remote locations. PCP and PCA can be formed in remote areas by other organochlorine substances such as HCB that are already present in those areas.
9. In laboratory studies, the majority of BCF values for PCP are below 5 000 and PCP undergoes rapid metabolism; PCP does not meet the bioaccumulation criteria.
10. PCA is bioaccumulative, with BCFs >5 000, however, there are uncertainties with reported BCF values, as test concentrations for PCA were highly variable over testing periods and multiple chemicals were tested simultaneously. PCA is demethylated to PCP in biota and is further metabolised and depurated rapidly in various species including, fish, earthworms and mammals. Residues in biota in remote areas are low and no indication of biomagnification up the food chain.
11. Deciphering the environmental monitoring information on PCP and PCA is complicated by their metabolic and degradation pathways. However, where long term data is available, concentrations of PCP and PCA are decreasing in various environmental compartments around the world. These decreases are likely a result of the PCP ban in the EU and the discontinuation of most uses and highly regulating the only use in North America and other markets. But both PCP and PCA are still frequently detected in the environment close to point sources as well as in remote areas.
12. Reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect, particularly in remote areas (Section 2.7). All the water monitoring concentrations cited are below WHO drinking water quality guidelines.
13. A large number of human biomonitoring studies exist for PCP and PCP has been detected in a variety of body tissues, as well as in amniotic fluid, cord blood, and mother’s milk, demonstrating exposure, and therefore potential hazard, to fetuses, infants and adults (section 2.5.1). However, concentrations of PCP in the urine of the French population (Fréry et al. 2013) did not exceed the German toxicological value HBM-II for PCP or HBM-I for PCP. One person, however, did exceed the HBM-I limit when the value was expressed on a creatinine basis..
14. A general decreasing trend was observed in the longer-term human biomonitoring studies. When concentrations of PCP were compared between remote and more populated areas (Parks et al. 2008; Rylander et al 2012; Sandau 2002), levels were found to be similar. Sjödin et al. (2000) determined that in Sweden and Latvia, consumption of fish was not a major source of exposure to PCP; levels of PCP in blood plasma were related to the country where the person lived.
15. PCP has a complete toxicological database, whereas the toxicological information on PCA is deficient. PCA is not expected to be of greater toxicological concern than PCP in humans. Currently available occupational risk assessments for its use as a heavy-duty wood preservative do not include dietary risk assessments because there are no registered food uses for PCP. Hepatotoxicity (toxic effects to the liver) has been observed in various animal species after both short- and long-term exposure to PCP and is the most sensitive non-cancer endpoint. Other effects have been reported, including reproductive and developmental toxicity, kidney toxicity, neurotoxicity, immunotoxicity, and endocrine effects at doses equal to or greater than those doses eliciting liver effects (U.S. EPA 2010).
16. Although there is evidence that PCP can affect thyroid hormones, developmental and reproductive toxicity studies did not demonstrate effects related to thyroid disruption. Current EFSA and North American regulatory policy consider endocrine effects to be threshold effects, i.e., only occurring above a certain level of exposure. Therefore, protecting individuals against liver effects is expected to be protective of the other toxicological effects of PCP, including effects on the endocrine system. PCA has a high BCF value but it is rapidly converted to PCP in mammals. Toxicological risks in addition to bioaccumulation should be assessed.
17. A suitable dietary human health risk assessment for PCP and PCA cannot be developed for people that are exposed to these compounds via their diet (traditional Inuit foods) because residues in their foods are not known, their diets are not well characterized and the toxicological database on PCA is lacking. However, the occupational exposures in wood treatment plants evaluated in U.S. EPA (2010) are expected to be much greater than incidental environmental or current dietary exposures to PCP based on general population exposure information available in ATSDR (2001).

# 5. Concluding statement

1. Pentachlorophenol (PCP), its related compounds (sodium pentachlorophenate, pentachlorophenyl laurate and pentachloroanisole, a transformation product of PCP) are being considered for listing in Annex A, B and/or C of the Convention. The Committee evaluated Annex D information at its eighth meeting held in Geneva from 15 to 19 October 2012 and decided that, while the PCP molecule itself does not meet all the screening criteria specified in Annex D, PCP and its salts and esters meet the screening criteria specified in Annex D, taking into account its transformation product PCA.
2. Additional information was submitted by parties and observers at Annex E for the risk profile. This information indicated that worldwide uses and production estimates have been significantly reduced since the 1990’s. Previous national and international evaluations have identified concerns with PCP and as such, countries have implemented measures to reduce both human and environmental exposure such as banning, restricting uses, additional regulatory measures for wood treatment facilities and/or disposal of treated wood and listing under international conventions such as Rotterdam.
3. Where long-term monitoring data exists, concentrations of PCP and PCA are decreasing in various environmental compartments and human biomonitoring studies around the world. These decreases are likely a result of the PCP ban in the EU, the discontinuation of most uses and highly regulating the only use in North America and other markets. Current levels of PCP and PCA in remote areas are expected to be below drinking water quality guidelines and are below levels expected to cause adverse effects to biota. This, coupled with the downward trend in PCA air concentrations, indicate the risk is also likely to continue to decrease with time.
4. Based on considerations of the available information, it is concluded that pentachlorophenol and its transformation product, pentachloroanisole are [unlikely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that no further global action is warranted.]

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