

**Stockholm Convention
on Persistent Organic
Pollutants**

Persistent Organic Pollutants Review Committee
Ninth meeting
Rome, 14–18 October 2013

**Report of the Persistent Organic Pollutants Review Committee
on the work of its ninth meeting****Addendum****Risk profile on pentachlorophenol and its salts and esters**

At its ninth meeting, by its decision POPRC-9/3, the Persistent Organic Pollutants Review Committee adopted a risk profile for pentachlorophenol and its salts and esters on the basis of the draft contained in document UNEP/POPS/POPRC.9/6. The text of the risk profile, as amended, is set out in the annex to the present addendum. It has not been formally edited.

Annex

**PENTACHLOROPHENOL AND ITS SALTS
AND ESTERS**

RISK PROFILE

Prepared by the ad hoc working group on pentachlorophenol and its salts
and esters

Persistent Organic Pollutants Review Committee

18 October 2013

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Executive summary

1. Pentachlorophenol (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 (e.g., biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent and is used in the production of pentachlorophenyl laurate. The salt sodium pentachlorophenate was used for similar purposes as PCP and readily dissociates to PCP. The ester pentachlorophenyl laurate was used in textiles. PCA is not used as a commercial chemical or pesticide and is not intentionally released directly into the environment. It can be produced through the transformation of PCP. PCA may result from the degradation of other structurally related chlorinated hydrocarbons, such as hexachlorobenzene (HCB), lindane (HCH), and pentachloronitrobenzene (PCNB).
2. PCP has either no uses or is banned in all E.U. member states, China, India, Indonesia, New Zealand, Russia and Switzerland. PCP is only allowed for wood preservation with additional restrictions and/or regulations in Canada, Mexico and the United States. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico. It is currently produced in Mexico and the U.S.
3. Considering the complex degradation and metabolic pathways of PCP and PCA both in the environment and in the biota, they should be considered together in the risk profile.
4. PCP is moderately mobile in lower pH soils and mobile in higher pH soils. It partitions to sediment and soil. PCP degradation may occur by photolysis, which is the fastest pathway, as well as by biodegradation. Under typical environmental conditions, half-lives are <4 weeks (water), <20 weeks (sediment) and <10 weeks (soil). However, PCP can persist for many years at contaminated sites where the levels of PCP exceed the toxicity threshold of soil microorganisms or in cold northern climates.
5. PCA is sparingly soluble in water and is likely to be immobile to slightly mobile in soils and partition to sediment in aquatic systems. It is expected to be volatile from moist soil and aquatic systems based on Henry's law constant but under laboratory conditions, volatility was observed from water, but not from soil. PCA meets the Annex D criteria for bioaccumulation. 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 and snow in remote locations.
6. PCP and PCA are detected in air, water, soil and biota throughout the world, including in remote regions. PCA is more dominant than PCP in air whereas PCP is found in higher concentrations than PCA in soil, sediment and sludge. In biota, PCA and PCP concentrations are comparable. Where long-term monitoring data exists, concentrations of PCP and PCA are decreasing in air and biota.
7. PCP is detected in the blood, urine, seminal fluid, breast milk and adipose tissue of humans. Biomonitoring information shows similar levels of PCP in humans from remote and more populated areas. It also demonstrates exposure, and therefore potential hazard, to fetuses, infants and adults. Compared to other chlorinated compounds, PCP is one of the most dominant contaminants measured in blood plasma.
8. PCP and PCA are hepatotoxic, carcinogenic, immunotoxic, neurotoxic and toxic to the reproduction. It should be noted that some of these hazards can be induced by an endocrine mode of action and there is a lack of scientific consensus related to the existence of a threshold for this mode of action. Due to the concentration of PCP/PCA observed in humans, adverse effects for human health related to the toxicities listed above cannot be excluded.
9. PCP and PCA are very highly toxic to aquatic organisms. Reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect particularly in remote areas. However, given the widespread distribution of PCP/PCA, that measurable levels of PCP/PCA are frequently found in biota and that PCP and PCA have an endocrine mode of action, environmental effects cannot be excluded.
10. In addition to the PCP and PCA interaction, these can also have toxic interactions with other POPs.
11. PCP and PCA levels in remote areas, as well as their toxicological parameters (NOEC and NOAEL), were compared with endosulfan and lindane. This approach in the risk profile showed that PCP, PCA, lindane and endosulfan are found in comparable concentrations in biota and in human populations from remote areas. PCP and PCA were also considered to have similar toxicity than endosulfan and lindane.
12. PCP and PCA are likely, as a result of their long-range environmental transport, to lead to significant adverse human health and/or environmental effects, such that global action is warranted.

1. Introduction

13. The European Community and its member States 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 Persistent Organic Pollutants Review Committee (POPRC) at its seventh meeting held in 2011 October.¹ 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, pentachlorobenzene, dioxins and furans are produced in the manufacturing process, although these chemicals should already be controlled as they are listed in the Convention.

14. At its seventh meeting, the Committee agreed to defer the decision concerning the proposal, pending the receipt of additional information on the transformation of PCP to pentachloroanisole (PCA) and the proposal by Japan to fill information gaps concerning the conversion of PCP to 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.

15. At the eighth meeting of the POPRC, held in October 2012, 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 PCP and its salts and esters in Annexes A, B and/or C to the Convention² and additional information on the substances collected since the Committee's seventh meeting.³ The Committee adopted decision POPRC-8/4, on PCP and its salts and esters, which stated as follows.

“The Persistent Organic Pollutants Review Committee,

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 to the Convention and having applied the screening criteria specified in Annex D to the Convention,

1. *Decides*, 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;

2. *Also decides*, 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;

3. *Invites*, 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 before 11 January 2013.”

16. 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.

17. The risk profile addresses both PCP and PCA (multiple exposure, overall half-lives, and similar toxicity).

1.1 Conclusion of the Committee regarding Annex D information

18. The Committee evaluated Annex D information at its eighth meeting 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

19. The primary source of information for the preparation of this risk profile was the proposal submitted by the European Community and its member States, contained in documents UNEP/POPS/POPRC.7/4,

¹ UNEP/POPS/POPRC.7/4.

² UNEP/POPS/POPRC.8/5.

³ UNEP/POPS/POPRC.8/INF/7.

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:

- (a) 2012, Government of Canada, PCA monograph;
- (b) September 2008, The United States Environmental Protection Agency (USEPA), Re-registration 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;
- (c) September 2010 – USEPA Integrated Risk Information System (IRIS) Summary for PCP (EPA-635-R-09-004F).

20. 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 the International POPs Elimination Network (IPEN) and Alaska Community Action on Toxics (ACAT) and Wood Preservation Canada.

21. A more elaborated summary of the submissions is provided in a supporting document (UNEP/POPS/POPRC.9/INF/7).

1.3 Status of the chemical under international conventions

22. PCP is subject to a number of regulations and action plans as highlighted below:

- (a) Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade;
- (b) OSPAR List of Chemicals for Priority Action (1998) of the Convention for the Protection of the Marine Environment of the North-East Atlantic;
- (c) Annex 1A (List of Priority Hazardous Substances) in the Third North Sea Conference;
- (d) Nominated as candidate for inclusion in Annex I of Long-range Transboundary Air Pollution (LRTAP) Protocol on POPs of the United Nations Economic Commission for Europe.

1.4 Chemical identity

23. 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 (e.g., biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent and is used in the production of pentachlorophenyl laurate PCP-L). The salt sodium pentachlorophenate (Na-PCP) was used for similar purposes as PCP and readily dissociates to PCP. The ester pentachlorophenyl laurate was used in textiles. PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst. Contaminants including hexachlorobenzene, pentachlorobenzene, dioxins and furans are produced during the manufacturing process. These compounds are inherently toxic, as well as environmentally persistent and their presence may increase the ecological risk associated with the use of PCP. This information is captured in Table 1-1(a), (b) and Table 1-2.

24. PCA is not used as a commercial chemical or pesticide and is not intentionally released directly into the environment. It can be produced through the transformation of PCP. PCA may result from the degradation of structurally related chlorinated hydrocarbons, such as PCP, hexachlorobenzene (HCB), lindane (HCH), and pentachloronitrobenzene (PCNB).

Table 1-1(a): Names and registry numbers

| | | |
|--|--|--|
| Common name | <u>Pentachlorophenol</u> | |
| Chemical name | 2,3,4,5,6-pentachlorophenol | |
| CAS registry numbers | Pentachlorophenol | 87-86-5 |
| | Sodium pentachlorophenate | 131-52-2 and 27735-64-4 (as monohydrate) |
| | Pentachlorophenyl laurate | 3772-94-9 |
| | Pentachloroanisole | 1825-21-4 |
| 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, Dirottox, Dow Pentachlorophenol DP-2 Antimicrobial, Dowcide 7, Dowcide 7/EC-7/G, Dowcide 6, Dowcide 7, Dowcide 7 Antimicrobial, Dowcide EC-7, Dowcide G, Dura TreetII, Dura treat 40, Durotox, EP 30, Forpen-50 Wood Preservative, Fungifen, GlazdPenta, Grundier Arbezol, 1-hydroxypentachlorobenzene, KMG Technical Penta Flakes, KMG Technical Penta Blocks KMG, Penta Blocks, 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, Pentachlorophenol, 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. | |

Table 1-1(b): Structures

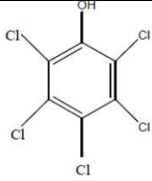
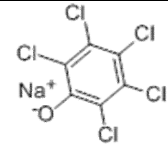
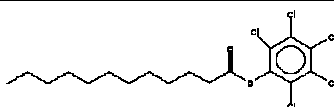
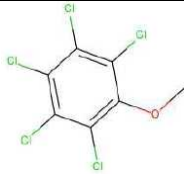
| | Pentachlorophenol | Sodium Pentachlorophenate | Pentachlorophenyl laurate | Pentachloroanisole |
|--|---|---|--|---|
| Molecular formula | C_6HCl_5O and C_6Cl_5OH | C_6Cl_5ONa and $C_6Cl_5ONa \times H_2O$ (as monohydrate) | $C_{18}H_{23}Cl_5O_2$ | $C_7H_3Cl_5O$ |
| Molecular Mass | 266.34 g/mol | 288.32 g/mol | 448.64 g/mol | 280.362 g/mol |
| Structural formulas of the isomers and the main transformation product |  |  |  |  |

Table 1-2: Physical and chemical properties of pentachlorophenol and pentachloroanisole

| Properties | Pentachlorophenol | Pentachloroanisole | |
|--|--|---|--|
| | Value ¹ | Value | Reference |
| Water solubility 25°C | 0.13% (% weight) 5 mg/L at 0°C ^{1,2} 14 mg/L at 20 °C ^{1,2} 35 mg/L at 50 °C ^{1,2} 14 mg/L at 25 °C ^{1,2} | <1 mg/L 0.24 mg/L 0.19 mg/L | http://cameochemicals.noaa.gov/chemical/20850 EVA method logK _{OW} method |
| Vapour pressure (25°C) | 2 mPa (20 °C) 0.0070-0.213 Pa (25 °C) 1.1 x 10 ⁻⁴ mm Hg (25 °C) ² Intermediate volatility | 0.0458 Pa (25 °C) 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.m ³ /mol ² 0.0248 to 0.284 Pa m ³ /mol Potential to volatilise from water or moist soil | 1.94x 10 ⁻³ atm-m ³ /mole (25 °C) (Group method) (1/H = 12.7, K _{AW} = 0.003) 7.12 x 10 ⁻⁵ atm-m ³ /mole (25 °C) (Bond method) Potential to volatilise from water or moist soil | HENRYWIN v3.2 in U.S. EPA 2011 Mackay and Wolkoff, 1973 classification scheme |
| Dissociation constant (pK_a) | pK _a 4.60-5.30 pK _a 4.7 ² 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 (LogK_{ow}) | The measured values are between 1.3 and 5.86 and the value appears to be pH dependent. Generally accepted 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) |
| K_{OC} | 293 to 900 L/Kg(at 0.0125 mg/L) 1000 L/Kg (calculated) 3000 to 4000 L/Kg (measured) 293-4000 L/Kg ² 706-3420 L/Kg (measured) ² Slight mobility to moderate mobility in soil | 2474 L/kg 13800 L/kg Immobile | MCI method, KOCWIN 2.0 K _{OW} method, KOCWIN 2.0 in U.S. EPA (2011) McCall et al., 1981 classification scheme |

Note:

¹ Values 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 Pentachlorophenol 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

25. 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 countries.
26. 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.
27. In Australia, historical uses include uses as an antispain fungicide and timber preservative.
28. In Canada, historical uses include antispain and specialty applications (paints, stains, wood joinery products, industrial water treatment products, oil field biocides and material preservatives) (CCME, 1997). All sapstain control and all other uses (e.g., domestic wood preservatives) were withdrawn in 1990.
29. In Japan as of 1990, registration of all the products containing PCP as agricultural chemicals was withdrawn. In 2003, use of PCP as an agricultural chemical was banned. In Japan PCP was historically used as a herbicide in rice paddy fields (Minomo et. al 2011). It was also used as a fungicide for agricultural use. In 1990, registration of all the products containing PCP as agricultural chemicals was withdrawn.
30. 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.
31. 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.
32. In India, the use of PCP in the tanning industry was prevalent.
33. Currently, PCP has either no uses or is banned in all E.U. member states, Australia, China, India, Indonesia, New Zealand, Russia and Switzerland. For a more comprehensive list of countries with bans and/or severe restrictions (Appendix V of UNEP/POPS/POPRC.9/INF/7).
34. Currently, the only reported world use for PCP is for wood preservation. Information collected showed that every country with wood preservation uses reported also has additional restrictions and/or regulations in place for managing the wood preservation industry, including Belize, Canada, Mexico and the U.S. In Canada and the U.S. the PCP wood preservation uses are for heavy duty wood preservation application and the treated wood products are for industrial use only. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico.
35. PCP is produced in Mexico and the U.S. The Mexican plant produced 7 257 tonnes/year in 2009 for the U.S., Canada and Mexico. 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 U.S., Colombia and Peru. Mexico reported importing PCP from the U.S., China and Germany between 1997 and 2011.
36. The U.S. reported that in 2002, approximately 5 000-5 500 tonnes were used for the treatment of utility poles, lumber and timbers (construction). Of the amount used, 4 083 tonnes were imported and 1 361-1 815 tonnes were produced domestically.
37. Canada reported that 372-537 tonnes of PCP were imported yearly from Mexico between 2008 and 2012 for the treatment of utility poles and crossarms.
38. More detailed information on current uses as informed by countries is provided in Appendix I and Appendix IV of UNEP/POPS/POPRC.7/INF/5 and UNEP/POPS/POPRC.7/INF/6.

2.1.2 Releases to the environment

39. There are several sources of PCP in the environment, including the release of PCP during its production and when it is used in accordance with currently registered uses as a wood preservative, for use in adhesives, tannery, paper and textiles. Other releases are contaminated sites and natural sources or burning processes (OSPAR, 2004).

40. The major worldwide use is as a heavy-duty wood preservative. In the life cycle of treated wood, PCP is potentially released into the environment during the manufacturing process, wood treatment processes, use and disposal of treated wood. Emissions to the environment (air, water and soil) from wood treatment facilities may occur:

- (a) During the treatment process (volatilisation to air);
- (b) During transfer of treated wood from dipping tanks for drying (runoff from wood surface to soil);
- (c) During the drying process (volatilisation to air and leachates to soil);
- (d) From leachates and from outdoor storage of treated wood (runoff from wood surfaces to soil);
- (e) Via evaporation from treated wood products (volatilisation to air);
- (f) As wood waste from sawing and processing of treated wood;
- (g) As solid waste, sludge from the bottom of dipping/treatment tank.

41. Many jurisdictions have implemented measures to minimize releases. An overview of international regulatory instruments is summarized in Cooper and Radivojevic (2012).

42. The following country specific release information was submitted to Annex E:

(a) For the 2011 reporting year, thirty-four PCP release reporting forms (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).

(b) Mexico reported the following releases: Incineration of unspecified 17 776 kg (could be treated material, active ingredient, end-use products) in 2008. Emissions to air were 38 kg in each year between 2006 and 2009. Releases to soil were estimated to be 0.0029 kg (2005).

43. PCP treated wood can be a source of dioxins and furans (Bulle et al. 2010; Fries et al. 2002; Lee et al. 2012; Lorber et al. 2002).

2.1.3 Other sources of PCP

44. PCP is also a transformation product and metabolite of other organochlorines such as HCB (hexachlorobenzene), HCH (lindane) and PCNB (quintozene). The extent of these potential sources cannot be quantified.

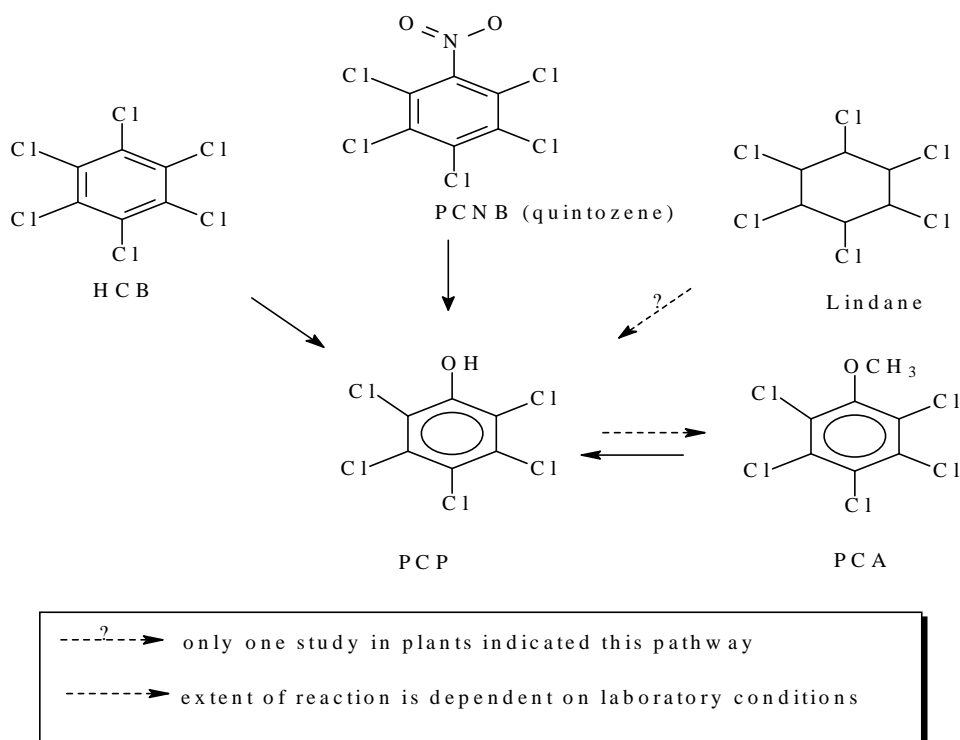


Figure 1: Examples of Sources of PCP in the environment

2.2 Environmental fate of PCP and PCA

45. Photolysis in aqueous solution 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. (UNEP/POPS/POPRC.7/4)
46. Under certain environmental conditions, the microflora can adapt and biodegrade PCP, with half-lives less than 4 weeks in water, less than 20 weeks in the sediment and less than 10 weeks in soil. The half-life in anaerobic sediment ranges from <13 days to <144 days (UNEP/POPS/POPRC.7/INF5 and EPA 2008). The WHO (1987) reports values ranging from 80-192 days, however, this half-life was based on a 28 day study. Many other studies discuss the degradation of PCP in terms of mineralization, with some of them showing a slow rate of mineralization (UNEP/POPS/POPRC.7/4). PCP is moderately mobile in lower pH soils and mobile in higher pH soils.
47. PCP-degrading bacteria are abundant in the environment and there are several pathways for degradation under aerobic conditions, depending on the experimental or environmental conditions. (UNEP/POPS/POPRC.8/5 and UNEP/POPS/PORC.8/INF/7)
48. 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. However, under laboratory conditions, volatility was observed from water, but not from soil where the methods indicated that systems were sealed with volatile traps (Walter et al. 2005; Chung and Aust 1995; Pfender et al., 1997).
49. 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. Under aerobic conditions, 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). 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.
50. The following summarises the results of studies conducted under environmentally relevant conditions. An aerobic transformation study of PCP with sludge and forest soil was conducted by the Japanese government to confirm the transformation of PCP to PCA. In the test with sludge, where secondary effluent of municipal sewage treatment plant was used as inoculum, no transformation of PCP to PCA was observed after 49-day incubation at initial PCP concentrations of 1.0 mg/L and 0.10 mg/L, respectively. In the test with forest soil, transformation of PCP to PCA was observed after 49-day incubation, and transformation rates were estimated to be 14 % and 26 % at initial PCP concentrations of 1.0 mg/kg and 0.10 mg/kg, respectively (CERI 2013). Several studies from the published literature showed 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). In Haimi et al. 1993, demethylation of chloroanisoles back to corresponding chlorophenols was observed. Observed half-lives for PCA are between 20-35 days (Haimi et al. 1993; D'Angelo and Reddy 2000 and Kuwatsuka and Igarashi 1975; Rubilar et al., 2007; Rigot and Matsumura 2002). However, there is uncertainty with these estimates since not all studies indicated whether systems were sealed; studies conducted with PCP as a starting material, resulted in simultaneous formation and degradation of PCA confounding half-life estimates; and these estimates are based on low or trace amounts of PCA. These are not full mineralisation half-lives. PCP (Tables 3.3-1 and 3.3-2 of UNEP/POPS/POPRC.9/INF/7) and PCA (Walter et al. 2005; Chung and Aust 1995; Pfender et al., 1997) were not volatile in the studies conducted with soil with properly sealed test systems and volatile traps .
51. Biotransformation via dechlorination is the principal degradation pathway in anaerobic soil, sludge and aquatic systems once in the sediment. Most studies reviewed showed no formation of PCA under anaerobic conditions (UNEP/POPS/POPRC.8/INF/7*). Under anaerobic conditions, PCA is demethylated to PCP (Murthy et al. 1979). The authors reported that 42% of the applied PCA was transformed to PCP in 24 days. The total radioactivity recovered in the soil was 98.8% indicating that any losses due to volatilisation were insignificant.
52. In aquatic systems, PCA is expected to partition to sediment and volatilise to air over time based on its physico-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 (U.S. EPA 2008).

53. Soils close to sawmill remain highly contaminated. Researchers found that there was no significant decrease of PCP in soil around wood preserving sites up to five years after the last use of technical PCP (Kitunen et al. 1987). Similarly, PCP has been found in urban air in New Zealand, 7 years after it was banned. The New Zealand government concluded that the chlorophenols, especially PCP and tetrachlorophenol, measured in air originate from the historic use of PCP as a timber preservative in New Zealand (Ministry for the Environment, New Zealand, 1998).

2.3 Bioaccumulation

54. In the open literature, a range for the log Kow for PCP varies between 1.3 and 5.86. Mackay, et.al (2006) recommended 5.12 and 5.18 as the values that should be considered in modelling studies. The large variation in log Kow stems from the dissociation of PCP depending on pH (decision POPRC.8/4).

55. In a bioconcentration study conducted on bluegill sunfish, BCF values were 190-790 (U.S. EPA 2008 submitted Annex E information as: *Pentachlorophenol Environmental Fate and Transport Assessment*.⁴). The BCF values in crustaceans, bivalves, aquatic and terrestrial worms and in fish vary between 0.9 – 4 900. (UNEP/POPS/POPRC.7/INF/5). Considering the majority of values are below 5 000 and PCP undergoes rapid biotransformation, PCP does not meet the numeric BCF criteria in Annex D.

56. Letcher et al. (2009) reports BMFs of 1.5 for PCP in polar bear lipids, indicating biomagnification. The proportion of PCP derived from metabolism of HCB (instead of eating contaminated prey) is not known. There is some uncertainty with this BMF estimate for this reason. Because the analytical method contained a methylation step that would not have differentiated between PCP and PCA, the BMF should be considered as an indication of combined biomagnification of PCP and PCA.

57. The log K_{OW} value of 5.45 and the BCF values of 12 000-20 000 for PCA reported by Oliver and Niimi (1985) for fish meets the Annex D numeric BCF criteria of 5 and 5 000, respectively, 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. 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, 1992; 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.

58. 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.

59. 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. PCA was detected at 12% of sites sampling predator fish, at levels higher than dieldrin or lindane (5 – 10% of sites) or endosulfan, mirex, or heptachlor (1 – 5% of sites) or aldrin, endrin, or the alpha isomer of HCH (<1% of sites).

60. PCA residues have been reported in biota in remote locations.

61. Muir (2013) reports residues of PCA in biota from remote Canadian Arctic areas. 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 lipid weight (lw)) than the other marine mammals. A study from Greenland reports measurable residues in varying species from aquatic invertebrates to fish, birds and mammals (Vorkamp et al. 2004). However, the concentrations of PCA found in these different trophic levels show no evidence of biomagnification. 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. However, for both studies, 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.

62. In Swachkhammer et al. 1988, the authors report mean PCA concentrations in fish sampled from Siskiwit Lake a remote lake in Isle Royal in Lake Superior of 3.6 ng/g lw and 6.5 ng/g lw in lake trout and whitefish, respectively.

⁴ <http://chm.pops.int/tabid/3070/Default.aspx>

63. In two studies on earthworms, concentrations of PCA in soil and earthworms were measured at an abandoned sawmill. In the soil, PCA concentrations were reported from 0.06-1.0 µg/g dry soil. PCA accumulated in earthworms and concentrations were reported to be 0.09-9 µg/g lipid (Haimi et al. 1992 and Haimi et al. 1993). Concentrations in earthworms and soil decreased simultaneously with an observed half-life of approximately 5 weeks (Haimi et al. 2003). BAFs estimated from the reported PCA concentrations are 5-40 µg lipid/dry soil (UNEP/POPS/POPRC.7/INF/5).

64. Vodcnik et al. (1980) determined that following the injection of PCA into female mice, elimination of ¹⁴C-PCA-equivalents was rapid with half-lives ranging from 5-10 hours in all tissues except for the liver.

65. Ikeda et al. (1994) determined that in the rat, PCA metabolites were eliminated in both urine and faeces, with blood elimination half-lives ranging from 6-15 hours. Metabolites included tetrachlorohydroquinone (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).

66. Ikeda and Sapienza (1995) reported tissue distribution, excretion and metabolism studies of pentachloroanisole (PCA), an environmental metabolite of pentachlorophenol (PCP), in the beagle dog and miniature pig following single oral doses (25 mg/kg) of radiolabelled PCA. PCA was readily demethylated by both species, with a half-life of 5-8 min. The resultant PCP was the major metabolite in dogs and pigs.

2.4 Potential for long-range environmental transport

67. PCP is a relatively volatile compound, while its sodium salt is non-volatile. Based on Henry's law constant, it has the potential to volatilize from water or moist soils. However, PCP was not detected in the volatile fraction in any of the laboratory studies reported in the UNEP/POPS/POPRC.9/INF/7 (Tables 3.2-1, 3.3-1 and 3.3-2). 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 as cited in UNEP/POPS/POPRC.7/INF/5), PCP has been detected in particulate matter in air. Atmospheric PCP associated with particulate matter or moisture is subject to wet and/or dry deposition.

68. The Henry's law constant of PCA indicates that PCA will likely volatilize 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, volatilization of PCA has been observed in several laboratory studies using liquid medium (Badkoubi et al. 1996, Walter et al. 2004, Lamar et al. 1990). The volatilisation of PCA from soil was not observed in the laboratory studies (Walter et al. 2005; Chung and Aust 1995; Pfender et al., 1997). A QSAR estimate of the phototransformation half-life of PCA in air is estimated to be 9.8 days (U.S. EPA 2011).

69. 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 5 200 m above the sea level with concentrations from 0.25 to 0.93 ng/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 (3-40 pg/m³), a transformation product of PCP, detected at higher levels than PCP (<1-3 pg/m³) (IVL Report B1474, June 2002). 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).

70. There are uncertainties regarding the source(s) of PCP and PCA detected at remote locations, as they may also be a result of the degradation of chlorinated hydrocarbons including PCB, HCB, HCH, and PCNB.

2.5 Exposure

2.5.1 Environmental monitoring data

PCP in Air, Water, Sediment and Soil

71. Borysiewicz (2008) compiled levels of PCP from various European sources. Concentrations of PCP in European river waters have declined since the early 1990s, when marketing and use restrictions were first implemented (UNEP/POPS/POPRC.7/INF/5). Concentrations in rivers from the Netherlands, Germany and Belgium ranged from 0.17 to 0.01 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 in the UK (0.15, 0.20, 0.02 µg/L in 1994, 1995, 1996, respectively), reflecting the use restrictions.

72. 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 compared to 1980-1989 (UNEP/POPS/POPRC.7/INF/5).

73. 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 (UNEP/POPS/POPRC.7/INF/5). 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.

74. 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.

75. Surface water used for drinking water in the U.S.A. contained a range of 0.04 to 1 µg PCP/L (mean ± SD of 0.4052 ± 0.4355 µg/L) (UNEP/POPS/POPRC.7/INF/5). Concentrations of PCP in ground water ranged from 0.04 to 1.64 µg/L (Mean 0.459 ± 0.444 µg/L).

76. Hoferkamp et al. (2010) does not report detections of PCP in water in the Canadian Arctic but PCA was detected (see below).

77. All measurements of PCP in inland surface waters and other surface waters in Estonia were at or below the LOQ 0.4 µg/L.

78. Zheng et al. (2011) in their summary of PCP studies found that 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.

79. 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.

80. Data for PCP in water from the international convention for the Rhine provide annual data. Concentrations of PCP in the Rhine River ranged from 0.1 to 0.006 ng/L between 2000 and 2011.

81. Cessna et al. (1997) monitored PCP/PCA in air near Yellowknife and in Saskatchewan, Canada. In 1994 corresponding concentrations were 0.43 to 3.68 ng/m³ (mean: 1.53 ng/m³) and 0.06 to 0.58 ng/m³ (mean 0.30 ng/m³), in Yellowknife and Saskatchewan, respectively. Because the analytical methodology in this study used diazomethane as a derivatizing agent, the authors could not differentiate between PCP and PCA.

82. 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 1 233 pg/m³ (1995-2001). Concentrations generally ranged though from not detected to 51.5 pg/m³.

83. 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.

84. 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.

85. Sediment in the River Narva, Estonia and two points in Lake Peipus (Estonia) remained below the limit of quantification (0.1 µg/kg dry weight (dw)) and PCP was not detected in coastal sediment (2010 data).

86. 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 approximately 40 µg/kg dw. Studies were from the UK, Netherlands, Germany and France. Temporal patterns are difficult to make as there are insufficient data and comparisons between different regions are not necessarily valid.

87. PCP was not detected in marine sediment from Norway between 2004 and 2008.

88. 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).

89. Soils close to sawmills that used PCP heavily are still highly contaminated with PCP many years after use was discontinued (Salminen et al. 1995). Similarly, soils at a wood preserving plant was heavily contaminated with PCP many years after the decommissioning of its operations (The Clean Environment Commission 1984). In Kitunen et al. (1987) 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.

PCP in Biota

90. PCP has been detected in birds eggs from Norway at concentrations ranging from <LOQ to 1 350 pg/g wet weight (ww) (Berger et al 2004).

91. 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.

92. The US National Study of Chemical Residues in lake fish (U.S. EPA 2009) did not detect PCP during 4 years (2000-2003) of sampling of predatory and bottom dwelling fish from 500 randomly selected lakes. The method detection limit was 555 µg/kg.

93. Dupont et al. 2013 reports levels of PCP in the blood of harbour seals at a median concentration of 134 pg/ml (range 62-293 pg/ml) and a mean concentration of 155 pg/ml ± 16.

94. In 1999, PCP was detected in herring from the Baltic Sea in concentrations ranging from 57-340 ng/g. (IVL Report B1474, June 2002).

95. Letcher et al. (2009) found PCP/PCA at mean concentrations of 1.0±0.4 ng/g lipid weight (lw) in the blubber of ringed seals sampled from East Greenland in 2002. Concentration of PCP/PCA in polar bears was determined via the BMF value of 1.5 resulting in mean concentrations of 1.5 ng/g lw in polar bears actual concentrations for polar bears was not included in Letcher et al. 2009. As discussed previously, there is some uncertainty as to whether the reported concentrations should be interpreted as concentrations of PCP or PCA or both.

96. 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) indicating widespread distribution as an atmospheric pollutant in Saskatchewan and in Europe concentrations ranged from 0.09 to 1.39 ng/g fresh weight (fw) (Eriksson et al. 1989). The analytical methods in these studies included a diazomethane derivatization step, therefore, they were unable to differentiate between PCA/PCP.

97. PCP was not detected in blue mussel and cod liver in Norway between 2004 and 2008 (Norway 2012 submitted information as: Information on pentachlorophenol and its salts and esters⁵).

98. PCP and PCA are detected in air, water, soil and biota throughout the world, including in remote regions.

Human Exposure and PCP in Human Biomonitoring Studies

99. PCP is detected in the blood, urine, seminal fluid, breast milk and adipose tissue of humans (Zheng et al. 2011b). 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 foetuses, infants and adults (section 2.5.1). Fréry et al. (2013) reported that PCP was detected in 66.2% of urine samples (LOQ: 0.03 to 0.1 µg/L) in the French population.

100. 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.

101. When human biomonitoring information for PCP was compared between remote and more populated areas levels were found to be similar. 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 ww) (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).

102. In Norwegian women, PCP and p,p'-DDE were the dominating compounds on a wet weight basis and present in considerably higher concentrations than PCBs and other chlorinated pesticides in 311 plasma samples of post-menopausal Norwegian women despite the fact that PCP is not in use in Norway and that the government of

⁵ <http://chm.pops.int/tabid/2543/Default.aspx>

Norway estimates that PCP emissions in Norway have been reduced by 99% in the period from 1995 – 2009 (Rylander et al. 2012).

103. Although a review article by Zheng et al. 2011 indicates a declining trend in humans since the 1970s when regulatory measures began to reduce emissions, PCP remains one of the dominant persistent organic pollutants in human blood on a wet weight basis (Rylander et al. 2012).
104. Concentrations of PCP in umbilical cord blood in Quebec Canada ranged from 628 to 7 680 µg/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 Quebec City.
105. Geometric mean concentrations of PCP in blood plasma from men, women and women of childbearing age among Inuit from Nunavik, Canada in 2004 were 1.0, 0.7, and 0.7 µg/kg lipid weight (lw), respectively. Concentrations ranged from 0.1 to 18 µg/kg lw in all groups (AMAP 2009). The Canadian Health Measures Survey conducted between 2009 and 2011 only detected PCP in urine in 3.45% of the 2551 samples taken from Canadians aged 3-79 years at 18 sites across Canada (Health Canada 2013). The only age group found to contain PCP in urine were the 60-79 years old (range <LOD – 1.1 µg/L; <LOD – 2.2 µg/g creatinine adjusted).
106. In a study carried out in rural and urban areas throughout Saskatchewan, PCP levels were measured in 24-hour urine samples from non-occupationally exposed individuals (Treble & Thompson 1996). Out of a total of 69 male and female participants from 6 to 87 years of age, the average urinary PCP level was 0.75 µg/L and ranged from 0.05 to 3.6 µg/L (Treble & Thompson 1996). It should be noted, however, that the analytical methodology used to determine concentrations of PCP in this study entailed a diazomethane derivatization step that prevented a determination whether the PCP detected was entirely PCP or a combination of PCP and PCA.
107. 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 years old children in urban and rural Spain (means = 6.4 ng/mL and 0.61 ng/mL, respectively).
108. Geometric means of concentrations of PCP in maternal plasma in Norway were 1.1 µg/kg lw in 2004. In Russia concentrations were 1.6 (range 0.8 – 12) and 1.7 (range 1.0 – 3.4) µg/kg lw in Taimyr and Naryan Mar, respectively in 2002 (AMAP 2009). Glynn et. al (2011) found no decline in PCP concentrations measured in the blood serum of Swedish women during pregnancy (range of concentrations 0.6-9.5 ng/g serum).
109. 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. It has been detected in urine in other studies as well (Cooper and Jones 2008, Hill et al. 1989).
110. Bradman et al. (2003) detected PCP in 3 of 20 samples of amniotic fluid from women in California (U.S.) at a geometric mean of 0.23 µg/L (of positive values) (range = 0.15 – 0.54 µg/L) 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 from women in Stockholm, Sweden 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 on average than the sum of OH-PCBs.
111. The U.S. Centers for Disease Control National Health and Nutrition Examination Survey (NHANES) III reported that the 95th percentile of PCP concentrations in urine was 1.0-2.0 µg/L in the 1999-2002 survey (Cooper and Jones, 2008). In the NHANES IV report (NHANES 2013) the 95th percentile concentrations of PCP in urine in all age groups was 3.63 µg/L in the US (n=2 354) with 95% CI of 2.98-4.61 µg/L. In a study of the levels of PCP in the urine of 197 children in Arkansas, researchers found detectable levels in 100% of the samples, with a median concentration of 14 ppb (Hill et al. 1989).
112. 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. 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.
113. 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.

114. Schulz et al. (2007) also shows that geometric mean concentrations of PCP in urine in adults aged 25-69 years old from the former West Germany had decreased during the sampling periods 1985/86, 1990/92, and 1998 from 4.4, 2.7 and 1.1 $\mu\text{g/L}$, 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.

115. Health Canada (2013) reports that food intake is estimated to account for 74 to 89% of total daily intake of PCP (based on Coad and Newhook 1992). Air is estimated to be 10 to 25% of total intake, whereas, water, soil and household dust are considered to be negligible sources. The estimated intakes by all of the populations studied were well below the Acceptable Daily Intake (ADI) of 6 $\mu\text{g/kg bw/day}$. The toxicological endpoint used to determine the ADI for PCP in Canada was 3 mg/kg bw/day from an NOAEL reported in both a subchronic reproductive study and a chronic study and a 500 x uncertainty factor. The estimates were comparable to other references where dietary intake was determined to account for the majority of PCP intake by humans (Hattemer-Frey and Travis 1989). The relative age of these studies may overestimate the current contribution of food to humans. In other studies examining remote populations, consumers of marine mammal fat in Nunavik, Canada had slightly higher concentrations of PCP in blood plasma ($p = 0.05$) than non-consumers (AMAP 2009).

116. Dewailly et al. 2014 reports that PCP was one of the substances showing the highest levels in a health survey of the Inuit population in Nunavik in 2004. PCP was detected in 100% of plasma samples with a median concentration of 822 ng/L and PCA was detected in 1.9% of samples, but could not be measured (LOD was 10 ng/L).

117. Corona et. al. (2013) report concentrations of 0.49 mg/kg PCP in household dust samples in 2010 in an area around a deactivated wood treatment facility.

PCA in Air, Water, Sediment and Soil

118. 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/m^3 , while those in the southern hemisphere (New Zealand) were 2.1 pg/m^3 (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/m^3 (Schreitmuller et al. 1995).

119. PCA has also been reported at several Arctic monitoring stations in Canada, the U.S., 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/m^3 , respectively. These values are comparable to other POPs reported by Hung et al. (2010) α -endosulfan 3-6 pg/m^3 (1993-2005), γ -HCH 4-16 pg/m^3 (1990s) and 1.4-10 pg/m^3 (2000s).

120. More recent information including time trend data are available for PCA in Arctic air (Figure 2). Current monitoring information shows mean concentrations of PCA in air are generally below 5 pg/m^3 in Arctic air at Alert, Nunavut, 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.

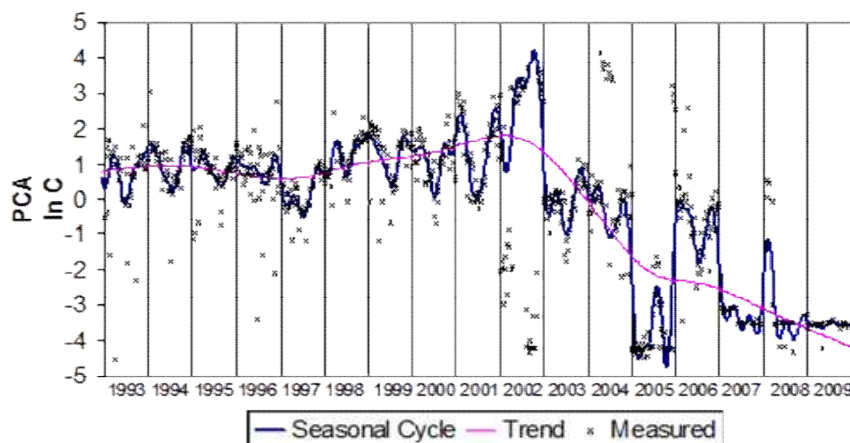


Figure 2: Time trend of PCA (gas+particle phase concentration C (pg/m^3) in air at Alert (1993-2009).

121. 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 with a factor of 200.

122. 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 (Table 5-15 of UNEP/POPS/POPRC.9/INF/7).

123. 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 (approximately 1991-2004) compared to the lower layers.

124. Concentrations of PCA in soil of the Taurus Mountains ranged from a low of 1.44 pg/g dry weight (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).

125. Only two studies report 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 that the source was likely 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).

126. Only one study (Jiang et al. 2000) has reported PCA concentrations in water and this was from an impacted area of the Yangtze River, China.

PCA in Biota

127. In 1999, PCA has been detected in herring from the Baltic Sea in concentrations ranging from 88-260 ng/g lipid weight. (IVL Report B1474, June 2002). A similar range was found for PCP in the same study and over the same sampling period.

128. 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 (Table 5-16 of UNEP/POPS/POPRC.9/INF/7) show low level residues in biota in remote locations.

129. 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). PCA was detected at 12% of sites sampling predator fish, at levels higher than dieldrin or lindane (5 – 10% of sites) or endosulfan, mirex, or heptachlor (1 – 5% of sites) or aldrin, endrin, or the alpha isomer of HCH (<1% of sites).

130. 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 lipid) than the other marine mammals. The animals were sampled from different parts of the Arctic and during a 10-year time span.

131. 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 authors considered concentrations of PCA in biota to be low.

132. PCA was also found in snow crab muscle and liver at 0.66 ng/g lipid and 0.45 ng/g lipid, respectively. Levels of PCA were also found in king eider and thick-billed murre livers at concentrations of 0.36 and 0.22 ng/g lipid respectively. Concentrations in Arctic marine mammals ranged from 0.08 ng/g lipid in harp seal to 0.54 ng/g lipid in narwhal muscle and 1.1 ng/g lipid in beluga muscle. PCA in caribou muscle was found at 0.20 ng/g lipid (Hoferkamp 2010).

Monitoring information of PCP with PCA

133. In a Swedish study (IVL Report B1474, June 2002), samples were collected for analysis of PCP and PCA in multiple media including air, rain, water, sediment, soil, sludge, fish and elk liver. The results show that the environmental levels of PCP in Sweden are generally lower than international quality limit values identified at the time of the study. PCP concentrations are significantly higher than PCA concentrations in soil, sediment and sludge, whereas PCA and PCP concentrations are comparable in biota. In air, however, PCA was detected in higher levels than PCP. Therefore, it is likely that possible long-range transport of PCP occurs in the form of PCA. The results are summarised in Table 2.1.

Table 2.1: IVL Screening report of PCP and PCA in various media in the environment in 2002.

| Medium | PCP concentration | PCA concentration |
|-------------------------------------|------------------------|------------------------|
| Air Background (pg/m ³) | <1-3 pg/m ³ | 3-40 pg/m ³ |
| Air Urban (pg/m ³) | <1-50 | <2-130 |
| Rain (ng/m ² /day) | n.d-3.4 | n.d.-0.16 |
| Water sources (ng/L) | <1.5-19 | <1.5-2.3 |
| Sediment (ng/g dw) | <1-28 | n.d.-1.6 |
| Soil background (ng/g dw) | 0.2-3 | <1-7 |
| Soil urban (ng/g dw) | 2-19 | <1-11 |
| Sludge (ng/g dw) | 7-200 | <1-11 |
| Biota-Herring (ng/g lipid) | 57-340 | 88-260 |
| Elk calf liver (ng/g lipid) | 26-130 | <1-3 |

2.6 Hazard assessment of PCP and PCA

134. PCP acts by uncoupling oxidative phosphorylation, inhibiting ATP pathways important to respiration in both animal and plant cells. Moreland and Hilton (1976) described PCP 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).

135. PCA is not industrially produced and not well studied. There are only limited toxicological data available. 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; Vodnick et al., 1980). Therefore, toxicity information for PCP is considered relevant for PCA.

2.6.1 Adverse effects on aquatic organisms

136. On an acute basis, both PCP and PCA are very highly toxic to aquatic organisms (UNEP/POPS/POPRC.9/INF/7, Table 3.11-1 and Table 3.11-2). In fish, PCP is more acutely toxic, but in invertebrates, PCA is more acutely toxic. 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 chronic effects to aquatic organisms were reported in the 0.1-100 µg/L range for PCP (UNEP/POPS/POPRC.9/INF/7, Table 3.11-1). The sublethal effects observed include reproduction, survival, growth, effects on the thyroid hormone (T3) activity and alteration of larval skeleton. The lowest sublethal endpoint is reported by Orton et al. 2009 in adult *Xenopus laevis* exposed to 0.1 and 1 µg/L. They reported statistically significant elevations in plasma progesterone levels when samples from both concentrations were pooled. Moreover degenerative ovarian features were observed but those were not statistically significant. However these effect levels are underestimations because at the higher concentration the measured value declined by up to 50% of the initial concentration after 48 hours. No measurements were available for the lower concentration level. The second lowest endpoint is a NOEC of 6 µg/L reported by Brooks 2001 in *Pimephales promelas* exposed to 90 days.

2.6.2 Adverse effects on terrestrial organisms

137. PCP is moderately to highly toxic to mammals and practically non-toxic to highly toxic to birds as per the U.S. EPA Ecotoxicity Categories for Terrestrial Organisms. Acute oral LD₅₀ values for rat were 50-220 mg/kg bw and acute oral dietary LC₅₀ values were 80-177 mg/kg diet for both rats and mice. In birds, the 5-day dietary LC₅₀ value in Japanese quail is greater than 5 139 mg/kg (Hill et al. 1975). LC₅₀ values reported by Hill et al. (1975) for northern bobwhite, pheasant and mallard duck varied between 3 400 and 4 500 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 et al. 1997; Beard et al. 1999; Beard et al. 1999a).

138. PCA is also toxic to mammals. Renner et al. 1986 reported acute oral LD₅₀ values of 318-331 mg/kg bw in mice; intraperitoneal LD₅₀ values of 281 (males) and 293 (females) mg/kg. The LC₅₀ value was ≥ 500 µg/g for earthworms (Salminen and Haimi 1991 as cited in Haimi et al. 1993). No information was found on the toxicity of PCA to birds and no information on the chronic toxicity of PCA was found.

2.6.3 Adverse effects on human health

139. 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.
140. 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.
141. The most sensitive endpoints are hepatotoxicity and endocrine effects (U.S.EPA, 2010). In addition to those references reviewed by the U.S. EPA, the following references indicate potential endocrine modulation, cytotoxicity and genotoxicity (Schurr 1998; Li 2010; Shan et al. 2013; Chen et al. 2013; Guo et al. 2013; Ma et al. 2011). For hepatotoxicity, the most sensitive endpoint is the dog, dosed orally at 1.5 mg/kg/day. For endocrine effects, these were observed in ram lambs, ewes, and mink exposed orally via different timing regimens at the dosage of 1 mg/kg/day (Beard et al. 1997; Beard et al. 1998; Beard et al. 1999a; Beard et al. 1999b; Rawlings et al. 1998).
142. 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. (U.S. EPA, 2010).
143. Other important toxic effects in laboratory animals for PCP include disruption of thyroid homeostasis. Although there is evidence that PCP can affect thyroid hormones, developmental and reproductive toxicity studies in rats and mice reviewed by U.S. EPA (2010) did not demonstrate effects related to thyroid disruption. 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).
144. 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 neurofunctional tests in animals.
145. PCP is considered non-mutagenic, although the tetrachlorhydroquinone (TCHQ) metabolite of PCP showed positive mutagenic effects in some tests. PCP is considered carcinogenic by all routes of exposure in laboratory animals. U.S EPA has recently classified PCP as likely to be carcinogenic to humans (U.S.EPA 2010).
146. 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 body weight, 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). In a study of pregnant Inuit women, maternal PCP levels were associated with lowered free T4 (fT4) levels in umbilical cord blood. However, the association between either maternal or neonatal PCP and fT4 was not apparent at a neonatal age of 7 months. This result suggests PCP may reduce the transfer of maternal T4 to the fetus by inhibiting T4 binding to transthyretin (TTR). There is also evidence that PCP contained in indoor dust contributes to thyroid hormone disruption. Suzuki et al. (2008) used chemical fractionation with *in vitro* competitive human TTR-binding assay and GC-MS to analyze the transthyretin (TTR) binding compounds in a sulfuric-acid-treated dust extract. As a result, they found that PCP was one of the potent TTR-binding compounds in all dust samples, and PCP contributed strongly to the TTR-binding potency of house dust. The concentrations of PCP in house and office dusts were 23-680 ng/g (median 100 ng/g) and 8.60-480 ng/g (median 55 ng/g), respectively. Neurological testing of infants was not conducted to determine if reduced T4 had affected neurological development. Small sample size limits interpretation of these results. (Dallaire, et al 2009a). In a later study, Dallaire (2009b) in the same population of Inuit, adults were within normal thyroid hormone range (>96% fT4, >99% fT3). Adults of this population had measurable concentrations of polyhalogenated compounds, including PCBs and organochlorine pesticides such as pentachlorophenol, and various other compounds, all of which are believed to affect thyroid hormone levels. Pentachlorophenol was not significantly associated with any thyroid parameter. It was concluded by the authors that, since most Inuit had normal thyroid status, it was not clear if the observed effects on thyroid parameters could be associated with increasing disease burden in adults.
147. 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.

148. 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-Hodgkin's lymphoma, multiple myeloma, soft tissue sarcoma, and liver cancer (U.S. EPA 2010). However, major weaknesses in exposure assessment methods often limit the validity of reported findings, either positively or negatively. 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.

149. In an epidemiological prospective cohort study prenatal exposure to organohalogenes, PCP was correlated in worse coordination, less sensory integrity, worse attention, and worse visuomotor integration in children of 5-6 years old in the Netherlands (Roze et al. 2009). Other examples of effects related to prenatal exposure to PCP include effects on motor, cognitive and behavioural performance in school children (Meijer 2012) as well as effects on sexual development (Roze et al. 2009).

150. Zheng et al. (2011) report adverse effects on motor, cognitive, and behavioural outcomes and other health risks correlated to PCP exposure at low environmental levels.

PCA Hazard Assessment

151. 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 metabolised and eliminated.

152. 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.

153. Daily PCA administration in diet over the lifespan of rats (2 years) 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.

154. Reproductive toxicity in rats is manifested as decreased corpora lutea and increased embryoletality. Reductions in male foetal body weight and crown-rump length of males were noted.

2.7 Environmental Concentrations and Effects of PCP and PCA

155. 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 sub-lethal endpoint reported. 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.

156. 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 and its mode of action as a respiratory uncoupler.

157. There is a 3-fold difference 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 minimum 30-fold margin of safety for PCA. This information is reported in the information document (UNEP/POPS/POPRC.9/INF/7, Table 3.12-1).

158. There are some uncertainties with these estimates since the estimate is based on a limited amount of environmental data. In addition, a critical body burden approach may not be applicable for non-threshold effects and there is evidence that PCP elicits endocrine effects at low doses. It should be noted that some of the adverse effects of PCP/PCA can be induced by an endocrine mode of action and there is a lack of scientific consensus related to the existence of a threshold for this mode of action.

159. Additional risk from combined exposure of PCA and PCP should also be considered.

160. PCP/PCA is hepatotoxic, carcinogenic, immunotoxic, toxic to the reproduction and neurotoxic. It should be noted that some of these hazards can be induced by an endocrine mode of action and there is a lack of scientific consensus related to the existence of a threshold for this mode of action. Due to the concentration of PCP/PCA observed in humans, adverse effects for human health related to the toxicities listed above cannot be excluded.

161. PCP and PCA are very highly toxic to aquatic organisms. Reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect particularly in remote areas. However, given the widespread distribution of PCP/PCA, that measurable levels of PCP/PCA are frequently found in biota and that PCP and PCA have an endocrine mode of action, environmental effects cannot be excluded.

3. Other Considerations

162. 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).

163. Contaminated sites may also have high levels of dioxins and furans due to the release of contaminants in PCP products. There is also evidence of higher dioxin exposure to humans living close to PCP contaminated sites (Lee et al., 2006). However, it should be noted that the presence of dioxins as impurities in PCP is not covered by the listing of dioxins in Annex C. 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 1987, the U.S. EPA required that no detectable concentrations of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) (1 pg/kg) be present in PCP. It also required that hexachlorodibenzo-p-dioxin (HCDD) concentrations cannot be above 2 ppm on a monthly average and that no single batch exceed 4 ppm. Between 1987 and 1999, the total dioxins in PCP technical products dropped 3-6 fold. In the European Union, a maximum allowable limit of 4 ppm for total 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 UNEP/POPS/POPRC.9/INF/7 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).

164. 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.

165. In addition to the PCP and PCA interaction, these can also have toxic interactions with other POPs. ATSDR 2013 (<http://www.atsdr.cdc.gov/toxprofiles/tp90.pdf>) showed that PCP co-administered with HCB increased the severity of HCB-induced porphyria. Also it has to be considered that the liver is a target organ not only for PCP but also for all HCH-isomers thereby leaving the risk for additive effects. Moreover the indigenous Arctic population as well as wildlife are exposed to a broad range of POPs including all HCH isomers and other pollutants leading to probably additive effects (UNEP/POPS/POPRC.3/20/Add.8). There is also a report by Shan et al. (2013) showing enhanced cytotoxicity of PCP by PFOS (perfluorooctylsulphonate) in HepG2 liver cells.

166. PCP and PCA levels in remote areas, as well as their toxicological parameters (NOEC and NOAEL), were compared with endosulfan and lindane, two POPs listed in 2009 and 2011 based on the review process provided in Article 8. This approach in the risk profile showed that PCP, PCA, lindane and endosulfan are found in comparable concentrations in biota and in human populations from remote areas (see Table 3.1). PCP and PCA were also considered to have similar toxicity to endosulfan and lindane.

Table 3.1: Comparison of some toxicity values and measured concentrations in biota and humans.

| COMPARISON OF THE TOXICITY OF ENDOSULFAN, LINDANE and PCP/PCA | | | | | |
|--|---|--|--|---------------------------------------|-------------------------------------|
| TOXICITY TO AQUATIC ORGANISMS | Lowest aquatic NOEC (fish) | Endosulfan : 0.05 µg/l (Knacker et al., 1991) | Lindane: 2.9 µg/l (lindane risk profile) | PCP 2 and < 15 µg/L (Euro Chlor 1999) | PCA no data |
| TOXICITY TO MAMMALS | Lowest NOAEL for mammals | Endosulfan 0.6 mg/kg bw day Rats (Ruckman et al., 1989) Dogs (Brunk 1989-1990) | Lindane: 0.8 mg/kg bw day Rabbit (lindane risk profile) | PCP 1 mg/kg-day (Demidenko, NM. 1969) | PCA no data |
| COMPARISON OF MEASURED CONCENTRATIONS IN BIOTA (for endosulfan: $\Sigma = \alpha$ -endosulfan+ β -endosulfan + endosulfansulfate; sum of indicated isomers in other cases) | | | | | |
| Reference & Location | Organism (tissue) | Endosulfan Mean (range) | Lindane Mean (range) | PCP Mean (range) | PCA Mean (range) |
| Bengtson Nash et al 2008. Antarctica | Invertebrate: Antarctic krill | Σ 419 (<LOQ-451) pg/g lw | 127 (<LOQ-127) pg/g lw | | |
| Herve et al. 1988 | MusselsFinland | | | | <1 – 274 ng/g lw |
| Wade et al. 1998 | Mussels US | | | | <0.25-8.99 ng/g dw |
| EPA 910-R-01-003. 2003. Alaska | Fish: Chinook salmon Fish: Chum salmon Fish: Sockeye salmon | Σ (<273-780) ng/kg Σ (<273)ng/kg Σ (<273-1610) ng/kg | (<124-203) ng/kg (<124-186) ng/kg (<124-793) ng/kg | | |
| Hinck et al. 2008. US mobile river bassin | BassCarp | | | | 60-380ng/kg ww 720-3.180ng/kg ww |
| Swackhammer et al. 1988. Siskiwit Lake US | Lake trout White fish | | | | 3.600 ng/kg lw 6.5 ng/g lw |
| Vorkamp et al. 2004 | Atlantic Cod | | | | 2.300ng/kg lw (median) |
| Canada unpublished | Lake trout (2002-2009) | | | | 70(<MDL-350) ng/kg lipid |
| Bentzen et al 2008, Alaska | Mammal: Polar Bear (fat) | α + β 8 ng/g lw | 8 ng/g lw | | |
| Bentzen et al 2008. Alaska | Polar bear | | | | 11 (<0.1-42)ng/g lw |
| Roseau et al. 2008. Alaska | Bird: Common murre (eggs) | Σ 3.15 ng/g ww | 0.19 ng/g ww | | |
| Berger et al. 2004. Norway | Bird (eggs) | | | <LOQ to 1.35 ng/g ww | |

| | | | | | |
|---------------------------------------|---|--|--|---|---|
| Miranda-Filho et al. 2007. Antarctica | Marine mammals: elephants seals: Adult males Adult females Juveniles Pups | \sum 3.02 ng/g lw \sum 2.68 ng/g lw \sum 1.99 ng/g lw \sum 0.90 ng/g lw | 1.04 ng/g lw 0.65 ng/g lw 0.34 ng/g lw 0.28 ng/g lw | | |
| Letcher et al. 2009. East Greenland | Ringed Seals | | | 1.0 ± 0.4 ng/g lw | 0.11 (<MDL-0.82) ng/g lipid |
| Hobbs et al 2003. North Atlantic | Marine Mammals minke whales (blubber) | α (<1 -33.6) ng/g lw | (<1 - 86.6) ng/g lw | | 1.10 ng/g lw Harp Seal, narwhal, beluga Vorkamp et al. 2004 |
| Hoekstra et al. 2003. Alaska, US | Marine mammals, bowhead, plasma | | | Mean=1.55±0.19 Range 0.16-3.48 (n=19) ng/g ww Bowheadwhale | |

COMPARISON OF MEASURED CONCENTRATIONS IN HUMANS

 (for endosulfan: \sum = α -endosulfan+ β -endosulfan + endosulfansulfate; sum of indicated isomers in other cases)

| Reference & Location | | Endosulfan Mean (range) | Lindane Mean (range) | PCP Mean (range) | PCA Mean (range) |
|--|-------------|-------------------------|----------------------|---|------------------|
| WHO Europe 2003 (NL, DE, UK, CD) | milk | | 1-100 ng/g lipid | 100 ng/g lipid | |
| Sandanger et al. (2004) Russian Arctic | plasma | | | 642ng/kg | |
| Sandauet al 2002 Nunavik Gulf of St. Lawrence and southern Québec urban center | Cord plasma | | | 1,670 ng/kg, (628-7,680) ng/kg ww | |
| WHO Europe 2003 (NL) | blood | | 100-200 ng/l | | |
| Roze et al. (2009) (NL) | blood | | 1 pg/g serum | 1,018 (297-8,532) fresh weight basis (pg/g serum) | |
| Rylander 2012 Women of the general population in Norway (n=311) | plasma | | | Range: Less than level of detection to 7686 ng/L ww Median: 711 ng/L ww; Arithmetic mean: 958 ng/L | |
| Dallaire 2009, Canadian Inuit | plasma | | | 801 ng/L, n=567 | |
| Guvenius 2003 Sweden | plasma | | | 2830 ng/L, n=15 | |
| Dewailley et al. 2004, Inuit of Nunivik | plasma | | | Mean= 1115.7 ng/g (n=925) | |

4. Synthesis of information

167. 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, pentachlorobenzene, dioxins and furans are produced during the manufacturing process. It should be noted that the presence of dioxins as impurities in PCP is not covered by the listing of dioxins in Annex C.

168. PCP is manufactured in India, Mexico and the U.S. 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, Mexico and the United States. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico. Uses in other countries are not known.

169. PCA is not produced commercially and, as such, it is not intentionally released in the environment. PCA is a metabolite that may be formed in soil and sediment from the biodegradation of PCP under aerobic conditions by certain microorganisms.

170. Considering the complex degradation and metabolic pathways of PCP and PCA both in the environment and in the biota, they should be considered together in the risk profile.

171. There are several sources of PCP in the environment, including the release of PCP when used in accordance with currently registered uses as well as the contaminated sites from previous use. PCP and consequently PCA can also be a transformation product and metabolite of other organochlorines such as hexachlorobenzene, lindane and quintozone. The extent of these potential sources of PCP/PCA in the environment cannot be quantified.

172. PCP is moderately mobile in lower pH soils and mobile in higher pH soils. It partitions to sediment and soil. PCP degradation may occur by photolysis, which is the fastest pathway, as well as by biodegradation. Under typical environmental conditions, half-lives are <4 weeks (water), <20 weeks (sediment) and <10 weeks (soil). However, PCP can persist for many years at contaminated sites where the levels of PCP exceed the toxicity threshold of soil microorganisms or in cold northern climates.

173. PCA is sparingly soluble in water and is likely to be immobile to slightly mobile in soils and partition to sediment in aquatic systems. It is expected to be volatile from moist soil and aquatic systems based on Henry's law constant but under laboratory conditions, volatility was observed from water, but not from soil.

174. The generally accepted log Kow values for PCP are 5.12 and 5.18, exceeding the screening criterion for bioaccumulation. BCF values in crustaceans, bivalves, aquatic and terrestrial worms and in fish do not meet screening criteria of 5000. A BMF value of 1.5 for PCP in polar bear lipids has been reported. PCA has a log Kow value of 5.45 that exceeds the screening criteria of 5.

175. 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 and snow in remote locations. PCP and PCA can also be formed in remote areas by other organochlorine substances such as HCB that are already present in those areas. The relative importance of the contribution of the two pathways (local transformation of other organochlorine compounds and actual long-range transport) is unknown.

176. PCP and PCA are detected in air, water, soil and biota throughout the world, including in remote regions. PCA is more dominant than PCP in air whereas PCP is found in higher concentrations than PCA in soil, sediment and sludge. In biota, PCA and PCP concentrations are comparable. Where long-term monitoring data exists, concentrations of PCP and PCA are decreasing in air and biota.

177. PCP is detected in the blood, urine, seminal fluid, breast milk and adipose tissue of humans. Biomonitoring information shows similar levels of PCP in humans from remote and more populated areas. It also demonstrates exposure, and therefore potential hazard, to the foetus, infants and adults. Compared to other chlorinated compounds, PCP is one of the most dominant contaminants measured in blood plasma.

178. PCP, but not PCA, uncouples oxidative phosphorylation. Since PCA is demethylated back to PCP in several species, e.g., mice, rats, rabbits and fish, toxicity information for PCP is considered significant for PCA.

179. PCP and PCA are hepatotoxic, carcinogenic, immunotoxic, neurotoxic and toxic to the reproduction. It should be noted that some of these hazards can be induced by an endocrine mode of action and there is a lack of scientific consensus related to the existence of a threshold for this mode of action. Due to the concentration of PCP/PCA observed in humans, adverse effects for human health related to the toxicities listed above cannot be excluded.

180. PCP and PCA are very highly toxic to aquatic organisms. Reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect particularly in remote areas. However, given the widespread distribution of PCP/PCA, that measurable levels of PCP/PCA are frequently found in biota, that PCP and PCA have an endocrine mode of action, environmental effects cannot be excluded.

181. In addition to the PCP and PCA interaction, these can also have toxic interactions with other POPs. PCP co-administered with HCB increased the severity of HCB-induced porphyria. Also it has to be considered that the liver is a target organ not only for PCP but also for all HCH-isomers thereby leaving the risk for additive effects.

182. PCP and PCA levels in remote areas, as well as their toxicological parameters (NOEC and NOAEL), were compared with endosulfan and lindane, two POPs listed in 2009 and 2011 based on the review process provided in Article 8. This approach in the risk profile showed that PCP, PCA, lindane and endosulfan are found in comparable concentrations in biota and in human populations from remote areas. PCP and PCA were also considered to have similar toxicity than endosulfan and lindane.

5. Concluding statement

183. Pentachlorophenol (PCP) and its related compounds that contribute to the presence of PCP in the environment (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.

184. 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. However, production and use information is lacking for a number of countries. 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 the Rotterdam Convention.

185. Both PCP and PCA are still frequently detected in the environment close to point sources as well as in remote areas. PCA concentrations in air are comparable to those of some other POPs. Human biomonitoring studies show PCP is consistently measured in populations around the world. Both PCP and PCA present high acute and chronic toxicity for both humans and the environment. PCP and PCA are found in biota and in human populations at similar concentrations in remote areas as previously listed POPs having similar levels of toxicity to PCP and PCA. In addition to the PCP and PCA interaction, these can also have toxic interactions with other POPs.

186. Based on the inherent properties of PCP and PCA, and given their widespread occurrence in environmental compartments and biota/humans in remote areas, it is concluded that PCP and its transformation product, PCA, are likely, as a result of their long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted.

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