**Stockholm Convention on Persistent Organic Pollutants**

**POPs Review Committee**

**PENTACHLOROPHENOL AND ITS SALTS AND ESTERS**

**SUPPORTING DOCUMENT FOR THE DRAFT RISK PROFILE**

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

under the POPs Review Committee

of the Stockholm Convention

**Second draft**

**16 April 2013**

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# Introduction

1. Pentachlorophenol (PCP) is an aromatic hydrocarbon of the chlorophenol family. PCP was first introduced for use as wood preservative in the 1930’s. Since its introduction PCP has had a variety of other applications (biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent, wood preservative and on the production of pentachlorophenyl laurate). The salt sodium pentachlorophenate (Na-PCP) was used for similar purposes as PCP and readily degrades to PCP. The ester pentachlorophenyl laurate (PCPL) is used in textiles. PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst.
2. The European Community and its member States that are Parties to the Convention submitted a proposal to list pentachlorophenol and its salts and esters in Annex A, B and/or C of the Convention at its seventh meeting held October 10-14, 2011 in Geneva (UNEP/POPS/POPRC.7-4). In this proposal, the reasons for concern were that Pentachlorophenol (PCP) and its related compounds (sodium pentachlorophenate, pentachloropheyl laurate and pentachloroanisole, a transformation product of PCP) are persistent in the environment and are frequently found in environmental compartments in remote areas. Information indicates that these substances are highly toxic to wildlife and humans, have the potential for long range transport and the potential to bioaccumulate. In addition, contaminants including hexachlorobenzene, dioxins and furans are produced in the manufacturing process, although these chemicals should already be controlled as they are listed in the Convention.
3. At its seventh meeting, the Committee agreed to defer decision considering the proposal pending the receipt of additional information on the transformation of pentachlorophenol to pentachloroanisole and the proposal by Japan to fill information gaps concerning the conversion of PCP to 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 was 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 soil which is considered to be the most relevant PCP-contaminated compartment in the environment.
4. At its eight meeting, held from the 15th to the 19th of October 2012 in Geneva, both the literature review and preliminary results from the laboratory studies conducted by Japan were presented. The Committee had before it a note by the Secretariat on a proposal to list pentachlorophenol and its salts and esters in Annexes A, B and/or C to the Convention (UNEP/POPS/POPRC.8/5) and additional information on the substances collected since the Committee’s seventh meeting (UNEP/POPS/POPRC.8/INF/7). The Committee adopted decision POPRC-8/4, on pentachlorophenol and its salts and esters.
5. Having examined the proposal by the European Union and its member States, parties to the Stockholm Convention on Persistent Organic Pollutants to list pentachlorophenol and its salts and esters in Annexes A, B and/or C of the Convention and having applied the screening criteria specified in Annex D to the Convention,
6. *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. The conclusion in the annex to decision POPRC-8/4 was: While the pentachlorophenol molecule does not meet all the screening criteria specified in Annex D, the Committee concluded, taking into account its transformation product pentachloroanisole, that pentachlorophenol and its salts and esters meet the screening criteria specified in Annex D;
7. *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;
8. *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.
9. 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 production, uses, 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.

# Summary

1. PCP was first introduced as a wood preservative in the 1930’s and has a variety of other applications (biocide, pesticide, disinfectant, defoliant, anti-sapstain agent, anti-microbial agent, wood preservative and textiles). PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst. Contaminants including hexachlorobenzene, dioxins and furans are produced during the manufacturing process.
2. Under aerobic conditions, large numbers of PCP-degrading bacteria have been identified and there are several transformation pathways and transformation products identified for PCP, depending on the experimental or environmental conditions. PCA, a persistent transformation product, can be generated from the biomethylation of PCP in the presence of some soil organisms (e.g., white rot fungi) under aerobic conditions. Since the formation of PCA involves the addition of a methyl group to PCP, PCA can be converted to PCP through demethylation. PCA itself is not industrially produced.
3. Both of these substances are highly toxic to wildlife and humans and have the potential to bioaccumulate. Even though reported laboratory half-lives indicate that these substances are not expected to be persistent, their detection in remote areas indicates that degradation may not be rapid in all compartments. Given the potential for interconversion between these two substances and their presence in remote areas, both PCP and PCA are relevant to the Annex E risk profile.
4. Historically, production has been estimated to be as high as 90 000 tonnes of PCP per year. Many sites contaminated from the historical use of PCP and from improper practices (e.g., spills from industrial holding ponds from wood treatment facilities prior to the implementation of strict regulations) continue to be sources of PCP in the environment.
5. PCP has either no uses or is banned in all E.U. member states, India, Indonesia, New Zealand, Russia and Switzerland. PCP is only allowed for wood preservation with additional restrictions and/or regulations in Belize, Canada, China, Mexico and the United States. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico. Other uses include ready-for-use products in Nigeria; and snail elimination to control the spread of schistosmoniasis in China.
6. The only manufacturing site for North America is in Mexico which the Wood Preservation Industry indicated produced 7 257 tonnes/year in 2009 for the United States, Canada and Mexico. The Mexican Government reported similar production information for 2009 (6 610 tonnes) and also supplied import/export information. Mexico reported that 734-6472 tonnes were exported yearly between 2007 and 2011 to the United States, Colombia and Peru. Mexico reported importing PCP from the United States, China and Germany between 1997 and 2011. The highest importation estimate was for 2007 where 100 kg of PCP (2 695 000 dollars) was imported into Mexico.
7. There are several potential sources of PCP and PCA in the environment, including from:

* PCP manufacturing sites
* current registered uses for PCP (wood preservation, agricultural uses);
* contaminated sites from historical uses (e.g., improper handling and storage practices);
* revolatilisation from adsorbed residues;
* metabolism of other organochlorines such as HCB and PCNB which are global pollutants and are detected in remote locations.

1. PCP is rapidly degraded in the environment with environmental half-life estimates of less than 4 weeks in water; less than 20 weeks in sediment and less than 10 weeks in soil. There are several pathways for degradation of PCP under aerobic conditions, depending on experimental or environmental conditions. Under anaerobic conditions, reductive dechlorination is likely to be the major degradation pathway of PCP. PCA can be generated from PCP as a result of methylation of PCP under aerobic conditions in the presence of somes bacterial and fungal species (e.g., white-rot fungi).
2. For PCA, the primary route of transformation in both soil and sediment is demethylation to PCP and incorporation into other PCP degradation pathways. Reported half-life estimates for PCA in the literature are often confounded by having PCP present in the test system thereby behaving as a constant source (i.e., formation and degradation of PCA would be occurring simultaneously), PCA concentrations are often reported at low or trace levels and there is some uncertainty regarding whether or not PCA was lost to volatilisation. Half-lives from these studies were estimated to be between 20-35 days. No biotransformation studies were found using PCA as a starting material.
3. In aquatic systems, PCA is expected to partition to sediment and air based on its physical-chemical properties. One study examining 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 and dissipated over time. There was also evidence that PCP transformed to lower chlorinated phenols (tetrachlorophenols and trichlorophenols).
4. The estimated log Kow estimates indicate the potential for the bioaccumulation of PCP, however, BCF values are below 5000 and the rapid biotransformation indicates that these BCF values are probably overestimations. Laboratory data for PCA also indicates the potential for bioaccumulation with estimated BCF values above 5000, however, additional laboratory information indicates that PCA is also metabolised and depurated in various species including fish, earthworms and mammals but at a slower rate than PCP.
5. PCP is a relatively volatile compound. Based on its Henry’s law constant, volatilisation from moist soils and aquatic systems is expected. In the atmosphere, volatilized PCP may undergo photolysis or may react with photochemically produced hydroxyl radicals. Laboratory derived photolysis half-lives in air are 12-44 hours which is considerably faster than the half-life for PCP in air based on modelling calculations. PCP has been detected in particulate matter in air. The average half-life for particles in the atmosphere is estimated to be about 3.9-10 days. Atmospheric PCP associated with particulate matter or moisture will be subject to wet and dry deposition.
6. The Henry’s law constant of PCA indicates that PCA will likely volatize rapidly from water which has been observed under laboratory conditions. Based on the predicted half-life estimates in air and its detection in air and snow in the arctic, there is sufficient evidence indicating that PCA is persistent in air and can be transported to remote locations.
7. Modelling calculations predict both PCP and PCA can be transported over considerable distances, however, PCA is more widely reported than PCP in air in remote areas. Both PCP and PCA have been detected in air monitoring programs close to potential sources (urban areas, proximity to historical use sites).
8. Overall, monitoring data for both PCP and PCA in abiotic matrices (air, soil and water) show a decreasing trend probably due to global regulatory actions (bans, restrictions and regulations) that have been put in place.
9. There are limitations using biomonitoring data as evidence of effects, as it only serves as a direct indicator of exposure to either PCP or other chemicals which are metabolized to PCP. Information is needed to relate external exposure levels to potential adverse affects.
10. Monitoring data for PCA in biota is not as prevalent as for PCP. PCA is detected in biota in remote areas at low levels. Several studies report lower levels of PCA residues in predators than prey in aquatic environments (Vorkamp et al., 2004; U.S. EPA, 2009) thereby contradicting the typical pattern of biomagnification. Compared with the results for chlorobenzenes and chlorinated pesticides, the concentrations of PCA are considered to be low. The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 2009) detected PCA in both bottom feeding and predator fish, however, the detection frequency was lower in the predators.
11. It is expected that PCA, untransformed within an organism, is likely less toxic than PCP because this methylated version of PCP loses its phenolic functionality. However, on an acute basis, both PCP and PCA are very highly toxic to aquatic organisms. Sublethal effects to aquatic organisms were reported in the µg/L range. The similarity of effects thresholds in the aquatic environment between the two substances likely represents biotransformation of PCA to PCP within test organisms. PCP is highly toxic to mammals and birds.
12. Reported environmental concentrations are generally lower than those levels expected to cause an environmental effect. Particularly in remote areas where PCP and PCA were seldom detected in water samples, very little risk to the aquatic environment is expected.
13. However, given the high detection frequency in air and that residues have been measured in biota, potential environmental effects were also considered using a critical body burden estimate.
14. Comparing the highest measured concentration in biota from the continenal U.S. in 1988 (Schmitt et al., 1988) to critical body burden estimates for PCA indicates a 3-fold margin of exposure lower than the internal toxicity threshold. Tissue residues reported for Arctic biota were much lower than those reported in the U.S. study and margins of exposure are expected to be much larger.
15. Regarding the potential adverse effects in humans, PCP is a pesticide and has a complete toxicological database. PCA is not expected to be a greater toxicological concern than PCP in humans. Currently available occupational risk assessments for the pesticidal use (wood treatment, in countries with current registrations, i.e. Canada, U.S.) do not include dietary risk assessments because there are no registered food uses for PCP. For people in the northern regions, well characterized traditional diets with residue data for PCP and PCA are not generally available or robust. Occupational exposures in wood treatment plants are expected to be much larger than incidental environmental or current dietary exposures to PCP based on general population exposure information available in ATSDR (2001).
16. Reported environmental concentrations of PCA in biota from remote areas (e.g., fish and seals) are low. However, since the toxicology database is deficient, and a well characterized northern traditional diet is generally not available, and residue data for PCA in traditional foods is not sufficiently characterized, a robust estimate of dietary human health risk cannot be developed.

**Table 3‑1:** Comparison of PCP and PCA properties to Stockholm Convention Criteria for PBT Characteristics

|  |  |  |  |
| --- | --- | --- | --- |
| **Persistence criteria as defined in Annex D** | **PCP** | **PCA** | **Remark** |
| Soil  (6 months-aerobic) | <30 d | <30 – 35 d | Below criteria threshold |
|  | EPISuite estimates: 37.5 to 62 d | Below criteria threshold |
| Sediments and flooded soil (6 months) | DT50: 14 days  t1/2: 4.9 days. | ca. 20 days. | Below criteria threshold |
| Water  (2 months) | Phys-chem properties indicate that neither PCP or PCA will remain in water and persistence in sediment will be indicative of aquatic systems | | Below criteria threshold |
| Bioaccumulation  (log Kow >5) | 5.12 and 5.18 | 5.45 | Meets criteria threshold |
| Bioaccumulation  (BCF or BAF >5000) | | | |
| Fish | BCF:5-4,900 | BCF: 15,000-20,000 | PCP: Below criteria threshold  PCA: Above criteria threshold |
| t1/2: 6 – 96 h | t1/2 : 1 - 23 d |  |
|  |  |  |
| Aquatic Invertebrates | BCF: 19-830 |  | Below criteria threshold |
| Aquatic and Terrestrial worms | BAF: ≤ 3,830 | BAF: 5-40 | Below criteria threshold |
| Mice |  | -Following injection half-lives ranging from 5-10 hours (Vodnick et al. 1980) | Likely below criteria threshold |
| Rats |  | -Ikeda et al. (1994) determined in the rat, metabolites were eliminated in both urine and feces, with blood elimination half-lives ranging from 6-15 hours.  -Bioavailability of PCA was low in both rats and mice and was sex independent. PCA is not expected to bioaccumulate in humans due to its rapid metabolism (demethylation) to PCP, which is subsequently rapidly metabolised. |  |
| **Field residues in biota in remote areas** | | | |
| Birds eggs | ND-1350 pg/g ww |  | Both compounds have been detected in biota from remote areas.  Hoekstra et al. (2003) noted that PCP was the most abundant halogenated phenolic compound found in bowhead plasma. It was suggested that the PCP originated from either HCB or PCA.  Vorkamp et al. (2004) noted the concentrations in top predatory marine mammals (harp seal, narwhal and beluga) do not exceed the concentrations in marine fish, contradicting the typical pattern of bioaccumulation in the food chain.  Higher range of concentrations reported in polar bears than seals In Bentzen et al. 2008. Polar bears were sampled within a 10 year span from different parts of the Arctic. |
| Ringed Seals | 1.0 ± 0.4 ng/g lw | n.d.-0.82 ng/g lw |
| Blue mussel, cod liver | Not detected |  |
| Pine Needles | 0.42-2.08 ng/g | 0.42-2.08 ng/g |
| Terrestrial animals  Marine invertebrates  Marine fish  Marine mammals |  | nd.- 0.36 ng/g lw  n.d -0.74 ng/g lw  n.d-2.3 ng/g lw  n.d.-0.86 ng/g lw |
| Polar bear (fat) | BMF: 1.5 (Letcher et al., 2009) | n.d.-42 ng/g lw |
| Fish |  | ND-6.5 ng/g lw |
| **Long-Range Transport** | | | |
| **Air** | T1/2: 12-44 h; clear weather conditions, mid-day) (Slooff et al. (1989)  t1/2:3.6-10 days (particle half-life) | T1/2: 9.8 days, with an atmospheric (OH) concentration of 1.56 x 106 OH/cm3 (AopWin v1.96 in U.S. EPA, 2011). | PCP: Below half-life criteria threshold  PCA: Meets criteria threshold. |
| **Monitoring Information** | | | |
| **Air** | 0.43 to 3680 pg/m3  Not detected in Su et al., 1998 (remote) | nd - 4.9 pg/m3 (remote)  Vapour pressure < 1,000 Pa | PCP is generally detected at low levels (or not detected) in air in remote areas, but is measured at higher concentrations closer to point sources. PCA is detected in air in remote areas and is generally detected more frequently and at higher concentrations than PCP. PCA concentrations have decreasedin the Canadian Arctic since 2003. |
| **Water** | ND-170 ng/L  None reported or not detected (remote) | nd - 0.6 ng/L  nd or none reported (remote) | PCP and PCA are generally not detected in water in remote areas. PCP concentrations in areas where PCP was used have decreased in areas where use has been restricted. |
| **Fine particles (brown snow)** | None reported or not detected | Snow: 1442 pg/L (remote)  Particles: 4.3 mg/g (remote) | PCA has been detected in remote areas transported by fine particles. |
| **Sediment** | ND – 40 μg/kg dw  None reported for remote areas | nd – 7.4 ng/g (Yantze River, industrial river)  generally <1, but found up to 4.52 ng/g dw (remote) | PCP concentrations are generally higher than PCA in soil, sediment and sludge. PCA is detected in sediment in remote areas. |
| **Biomonitoring Information** |  |  | PCP is often a dominant organochlorine substance in biomonitoring studies. |
| **Toxicity** | | | |
| **Terrestrial Organisms** | | | |
| Mice Oral LC50: | 117-177 mg/kg | 318-331 mg/kg | PCP: highly toxic |
| Rat Oral LC50  Oral LD50: | 80-120 mg/kg  50-220 mg/kg | ≥ 500 mg/kg | PCP:moderately toxic  PCA: slightly toxic |
| Birds (dietary LC50) | > 5139 mg/kg |  | PCP: practically non-toxic |
| Birds (oral LD50) | 380 - 504 mg/kg |  | PCP: highly to Moderately toxic |
| Earthworms |  | ≥500 µg/g |  |
| **Aquatic Organisms** | | | |
| Invertebrates (EC50, LC50, MAT) | 240 – 2000 µg /L | 10-27.2 µg/L | PCP : Moderately to Highly toxic  PCA: Very highly toxic |
| Fish LC50: | 34-600 μg/L | 650 µg/L | PCP: Highly to Very Highly Toxic  PCA: Highly Toxic |
| Aquatic plants EC50 | 80-7000 µg /L |  | No classification scheme |
| **Human Health** | | | |
| Mice Oral LC50: | 117-177 mg/kg | 318-331 mg/kg | PCP: highly toxic |
| Rat Oral LC50  Oral LD50: | 80-120 mg/kg  50-220 mg/kg | ≥ 500 mg/kg | PCP:moderately toxic  PCA: slightly toxic |
| Chronic | Oral RfD 0.005 mg/kg bw/day based on hepatotoxicity in a 1 year dog study | No RfD established. | The liver is the primary target for noncancer effects of oral exposure to PCP. Numerous short- and long-term oral studies show that PCP is toxic to the liver of rats, mice, and dogs. This reference dose is protective of all shorter term exposures, including reproductive, developmental and endocrine effects. |
| Developmental toxicity | No developmental toxicity. Observed toxic effects were not teratogenic in nature, but rather embryo- or fetotoxic. | Reproductive toxicity in rats is manifested as decreased corpora lutea and increased embryolethality. Reductions in male fetal body weight and crown rump length of males were noted. | PCP: Non-reproductive or developmental toxicity study oral NOAELs based on liver effects are protective of reproduction and developmental effects. |
| Reproductive toxicity | Observed toxic effects were not teratogenic in nature, but rather embryo- or fetotoxic. | Reproductive toxicity in rats is manifested as decreased corpora lutea and increased embryolethality. Reductions in male fetal body weight and crown rump length of males were noted. | PCP: Non-reproductive or developmental toxicity study oral NOAELs based on liver effects are protective of reproduction and developmental effects. |
| Endocrine | Affects thyroid hormone system. | Unknown |  |
| Neurotoxicity | Can produce histopathological and biochemical changes in nervous tissue. Behavioural changes are also produced. | Unknown |  |
| Immunotoxicity | Impairs humoral and cellular immune reponses | Unknown |  |
| Mutagenicity | Overall non-mutagenic | Overall mutagenic |  |
| Carcinogenicity | Carcinogenic  Based on male mouse tumour data (liver and adrenal gland tumors are relevant to humans)  Note that the degree of influence of PCP contaminants on the cancer potency cannot be determined. The most sensitive cancer risk estimate was used.  Oral cancer risk estimate slope factor of 4 × 10 -1 (mg/kg-day) -1. The recommended slope factor should not be used with exposures >0.3 mg/kg-day  An inhalation unit risk was not derived. | Some evidence for carcinogenicity.  A cancer risk slope factor has not been developed. | PCP: The available epidemiologic studies support an association between PCP exposure and development of specific cancers, i.e., non-Hodgkin’s lymphoma, multiple myeloma, soft tissue sarcoma, and liver cancer. However, the lack of an exposure estimate that allows for an absolute rather than a relative level of exposure renders these studies unsuitable for deriving cancer risk estimates for PCP via the oral or inhalation routes. |

# Summary of Information Relevant to the Risk Profile

## Data sources

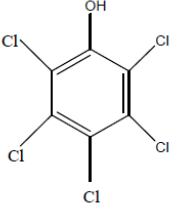
1. The primary source of information for the preparation of this risk profile was the proposal submitted by the European Community and its member States that are Parties to the Convention, contained in document UNEP/POPS/POPRC.7/4, UNEP/POPS/POPRC.7/INF/5, UNEP/POPS/PORC.7/INF/5/Add.1; additional information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

* 2012. Government of Canada. PCA monograph.
* September 2008 – US EPA’s Reregistration Eligibility Decision (RED) for PCP.
* September 2010 – US EPA’s Integrated Risk Information System (IRIS) Summary for PCP (EPA-635-R-09-004F)
* February 16, 2008 – US EPA memo – Environmental Fate and Transport Assessment of PCP for Reregistration Eligibility Decision (RED) (EPA-HQ-OPP-2004-0402-0066)

1. In addition the following parties and observers have answered the request for information specified in Annex E of the Convention: Canada, Croatia, Estonia, Mexico, Nigeria, Romania, Slovakia, Sri Lanka, Sweden, Thailand (Annex D), United States of America, joint submission of IPEN and Alaska Community Action on Toxics (ACAT) and Wood Preservation Canada. See Appendix I for a more elaborate summary.

## Chemical Identity

**Technical Pentachlorophenol**



**Molecular Structure of Pentachlorophenol (PCP)**

**Common name:** Pentachlorophenol

**Chemical name:** 2,3,4,5,6-pentachlorophenol

**Chemical family:** Aromatic Hydrocarbon Chlorophenol

**Empirical formula:** C6H Cl5O and C6Cl5OH

**CAS Registry No.:** 87-86-5

**Case number:** 2505

**OPP Chemical Code:** 063001

**RTECS Number:** SM6300000

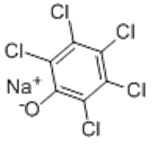
**UN Number:** UN2671, UN2762, UN2995, NA2020

**Molecular weight:** 266.34 g/mol

**Other names:** Pentachlorophenol is abbreviated as PCP. Product names include Acutox,Block Penta,Chem-Penta, chem-Tol, Chlon, Chlorophen, Cryptogil Oil, Cryptogil OL, Dirotox, Dow Pentachlorophenol DP-2 Antimicrobial, Dowcide 7, Dowcide 7/EC-7/G, Dowicide 6, Dowicide 7, Dowicide 7 Antimicrobial, Dowicide EC-7, Dowicide G, Dura TreetII, Durotox, EP 30, Forpen-50 Wood Preservative, Fungifen, GlazdPenta, Grundier Arbezol, 1-hydroxypentachlorobenzene, Lautor A, Lauxtol, Lauxtol A, Lauxtrol A, Liroprem, OnTrack We Herbicide, Ortho Triox Liquid Vegetation Killer, Osmose Wood Preserving Compound, Penchlorol, Penta, Penta C 30, Penta Concentrate, Penta Plus 40, Penta Pres 1-10, Penta Ready, Penta WR, Penta WR1-5 Penwar, Pentachlorophenate, 2, 3, 4, 5-pentachlorophenol, Pentachlorophenol DP-2, Pentachloropheno, Pentachlorphenol, Pentacon, Penta-kil, Pentasol, Pentchloral, Penwar, Peratox, Permacide, Permagard, Permasan, Permatox, Permatox DP-2, Permatox Penta, Permite, Persasan, Prevenol, Priltox, Santobrite, Santophen, Santophen 20, Sautox, Sinithuo, Sinituho, Term-I-Trol, Thompson's Wood Fix, Watershed Wood Preservative, Weed and Brush Killer, Weedone, Witophen P, Woodtreat, Woodtreat A.

**Basic manufacturer:** KMG-Bernuth, Inc., Vulcan Materials Co., (Chemicals Div.), Birmingham, Alabama, USA. In the 1980s produced by Ameco, Canada; National Product Co., China; Chapman Chemicals; Pola Quimia SA de CB, Mexico city, Mexico; Preservation Products, Matamoros, Mexico; Melchemie, Holland; Rhône-Poulanc, Lyon, France.

**Sodium Pentachlorophenate**

****

**Molecular Structure of Sodium Pentachlorophenate**

**Empirical formula:** C6Cl5ONa and C6Cl5ONa x H2O (as monohydrate)

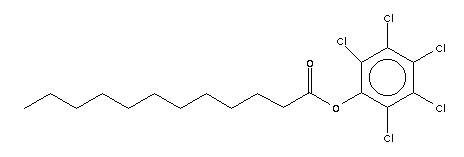
**CAS Registry No.:** 131-52-2 and 27735-64-4 (as monohydrate)

**RTECS Number:** SM6490000

**Molecular weight:** 288.32 g/mol

**Other names:** Penta-ate, Pentachlorophenate sodium, Pentachlorophenol sodium salt, Pentachlorophenoxy sodium, Pentaphenate, Phenol pentachloro- sodium derivative monohydrate, Sodium PCP, Sodium pentachlorophenolate, Sodium pentachlorophenoxide.

**Pentachlorophenyl laurate**

****

**Molecular Structure of Pentachlorophenyl laurate**

**Empirical formula:** C18H23Cl5O2

**CAS Registry No.:** 3772-94-9

**Molecular weight:** 448.64 g/mol

**Other names: pentachlorophenyl dodecanoate, 2,3,4,5,6-pentachlorophenyl dodecanoate**

**Pentachloroanisole**



**Molecular Structure of Pentachloroanisole (PCA)**

**Formula:** C7H3Cl5O

**CAS registry number:** 1825-21-4

**CAS chemical name:** Pentachloroanisole

**IUPAC name:** 1,2,3,4,5-Pentachloro-6-methoxybenzene

**SMILES notation:** COC1=C(C(=C(C(=C1Cl)Cl)Cl)Cl)Cl

**Synonyms:** 1,2,3,4,5-Pentachloro-6-methoxy-benzene; 2,3,4,5,6-pentachloro-anisol; Benzene, pentachloromethoxy-; ether,methylpentachlorophenyl; Methyl pentachlorophenate; Methyl pentachlorophenyl ester; Methyl pentachlorophenyl ether, PCA

**Molecular weight:** 280.362 g/mol

## Contaminants

1. PCP is produced by reacting chlorine with phenol at high temperatures in the presence of a catalyst. Contaminants including hexachlorobenzene, chlorinated dibenzodioxins and chlorinated dibenzofurans are produced as impurities during the 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 pentachlorophenol. Concentrations of dioxin and furans, present as impurities, decreased after legal measures were taken in the U.S. and Europe between 1987 and 1999. Since 1992 in the European Union, PCP has not been allowed to contain more than 4 ppm of total hexachlorodibenzo-p-dioxin (HCDD).This was further reduced to 2 ppm in 2000. Current levels in Canada technical products are found in Appendix II. Based on the maximum levels detected as reported in Appendix 1, the total HCDD and total dioxins/furans are 0.4 ppm and 0.8 ppm, respectively (TEQ calculated as per Van den Berg et al. 2006).

## Status of the chemical under international conventions

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

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

## Prohibitions, bans and restrictions

1. Prohibitions or bans as reported in UNEP/POPS/POPRC.7/INF/5; UNEP/POPS/POPRC.7/INF/6; and Annex E forms:
2. For all E.U. member states the use of PCP was restricted in 1991 by Council Directive 91/173/EEC and all uses including wood preservation officially terminated at the end of 2008 (according to Commission Directive 1999/51/EC). With the Biocides Directive 98/8/EC also biocidal product types where PCP and its salts and esters were covered. Only those products which contain active substances listed on the Annex I of the directive can be authorised.
3. The Integrated Pollution Prevention and Control Directive (IPPC Directive) 96/61/EC and the new Directive 2010/75/EU on industrial emissions (integrated pollution prevention and control) cover emissions and discharge of installations dealing with treatment of PCP containing material. In addition, there are national regulations in the EU Member States which may also cover emissions and discharge from formulation and use of these substances and requires mandatory inspection and reporting.
4. The European Regulation (EC) No. 1907/2006 of the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH), Annex XVII concerning the Restrictions on the manufacture, placing on the market and use of certain dangerous substances, preparations and articles, has taken up the general limit of 0.1% (1000 ppm) for PCP.
5. The Slovak Republic reports that PCP-based pesticides have been prohibited since 1982.
6. Other countries reporting bans include India, Indonesia, Russia and Switzerland.
7. Thailand reports that PCP has been banned for agricultural use since 1995, public health uses since 2000 and industrial use since 2001.
8. In Belize as of 1985, PCP use was severely restricted and can only be used for wood preservation purposes by approved and certified establishments and personnel.
9. In Canada as of 1990, PCP is only used as a heavy duty wood preservative to treat primarily electrical utility poles and crossarms. It is also used on posts and industrial construction timbers. PCP can only be used in specialised facilities compliant with the “Technical Guidance for the Design and Operation of Wood Preservation Facilities, 2004”. Out-of-use treated wood and treated wood waste is disposed as per the “Industrial Treated Wood Users Guidance Document, 2004”. The TRDs) take an all encompassing approach to environmental and health safety by considering all factors that affect worker health and environmental safety and providing recommendations to minimize these (Cooper and Radivojevic, 2012).
10. In China as of 1982, PCP has been banned for registration and production, sale and use as a pesticide. Uses were restricted to wood preservation. Areas and methods of approved application as stated in the “Bulletin of Pesticide Registration” should be observed. However, as stated below its use as a molluscide has increased in recent years.
11. In New Zealand as of 1991, all uses and products are banned. There is an agreement in principle to permit re-introduction in closed timber treatment systems at approved sites with specific conditions on disposal of waste. These conditions have not been met and therefore no products registered, no use permitted and no imports allowed.
12. In the United States, PCP is restricted for the treatment of utility poles, lumber and timbers (construction). In the USA, the industry is heavily regulated and US states have a well-developed approach to managing treated wood and other wastes. They identify different well-defined landfill types with prescriptions of whether treated wood waste can be disposed there and generally provision for incineration for energy subject to meeting air emission requirements. The USA EPA addresses design and operation considerations through various federal and state acts. (Cooper and Radivojevic, 2010)
13. UNEP (1994) has also developed guidance on best practices for the wood preservation.

## Sources and Uses

1. There are several sources of PCP in the environment including the release of PCP when used in accordance with currently registered uses (i.e., manufacturing process, wood treatment process, volatilisation or leaching from in-use treated wood, disposal of treated wood). PCP is a general biocide which has been used extensively as a fungicide, bactericide, herbicide, molluscicide, algaecide and insecticide by agriculture and other industries including textiles, paints, oil drilling and forestry.
2. Many sites contaminated from the historical use of PCP and from improper practices (e.g., spills from industrial holding ponds from wood treatment facilities prior to the implementation of strict regulations) continue to be sources of PCP in the environment. Releases to the environment may also occur through revolatilisation from adsorbed residues of PCP/PCA.
3. PCP is also a transformation product of other organochlorines such as HCB and, PCNB (quintozene) (Murthy and Kaufman, 1978 and U.S. EPA RED for PCNB, 2006) and lindane (Engst et al. 1979). These organochlorine substances are global pollutants and have been detected in remote locations. PCA resulting from the use of PCP and other organochlorine such as HCB may contribute to the occurrence of PCA/PCP in biota remote areas. In all likelihood it is the combination of all potential precursors that contribute.
4. PCA is not a registered substance and is not released directly into the environment. Pentrachloroanisole (PCA) is introduced into the environment through the methylation of pentachlorophenol (PCP) by soil or sediment micro-organisms. This occurs primarily in the aerobic environment. Biomethylation (PCP → PCA) is a ubiquitous reaction, but generally not the major route of PCP degradation (Valo and Salkinoja-Salonen, 1986). PCA is produced from PCP under aerobic conditions in the presence of some species of fungi. PCA is not produced from PCP through abiotic transformation processes such as hydrolysis and photolysis (soil, water and air).

**Historical uses:**

1. Historically, according to the data profile of IRPTC (1983), 90 000 tonnes of PCP per year were produced globally. The Economist Intelligence Unit (1981) estimated world production to be of the order of 50 000-60 000 tonnes per year, based on the North American and European Community output (UNEP/POPRC.7/INF/5). By the 1990s, the widespread use was discontinued in most counties.
2. In Europe, historical uses included use in the remedial treatment of timber and as a surface biocide for masonry. It was used in the preservation of textiles (wool cotton, flax and jute fabrics and yarns used in covers, tarpaulins, awnings, tents, webbing and netting and also sisal and manila ropes). It was also used as a preservative in oil-based paint, glues, adhesives, as an intermediate in the synthesis of pharmaceuticals, as an intermediate product in colouring substances, in mushroom farms, in slime control in pulp and paper as well as an agricultural chemical in weed control.
3. In Australia, historical uses include uses as an antisapstain fungicide and timber preservative.
4. In Canada, all sapstain and specialty applications (paints, stains, wood joinery products, industrial water treatment products, oil field biocides and material preservatives) were discontinued in 1990 (CCME, 1990).
5. In Sweden, PCP was used in large quantities mainly as a wood preservative and in pulp production. An important, but minor use of PCP was in the protection of textiles.
6. In the U.S., PCP was used in rice and sugar production, in water treatment, as a pre-harvest defoliant in cotton and as a general pre-emergence herbicide. It has also been utilised in numerous products including adhesives, construction materials, leather and paper.

**Current uses:**

1. Currently, PCP has either no uses or is banned in all E.U. member states, Australia, India, Indonesia, New Zealand, Russia and Switzerland. PCP is currently only allowed for wood preservation with additional restrictions and/or regulations in the following countries: Belize, Canada, China, Mexico and the United States. In Canada and the U.S. the wood preservation uses are for heavy duty wood preservation uses only. This type of wood preservation is applied through high pressure impregnation in a treatment cylinder, or retort, at treatment facilities. Registered uses on adhesives, tannery, paper and textile were also reported for Mexico; ready-for-use products in Nigeria; and snail elimination to control the spread of schistosmoniasis in China.
2. In the U.S., pentachlorophenol is currently classified as a Restricted Use Product (RUP) when used as a heavy duty wood preservative and is predominately used to treat utility poles and cross arms.
3. Although all uses were restricted to wood preservation in China, the production and use of PCP for snail elimination and schistosomiasis control has increased due to re-emergence of this disease (Liu et al., 2003 as cited in Zheng et al., 2012).
4. In Nigeria, PCP is imported for marketing and formulations of ready-for-use products or those that required some dilution, dissolution or addition of other (active) substances. Nigeria reports use as alagaecides, bactericides, fungicides, herbicides, insecticides, molluscides, defoliants and germicides as well as use as a preservative. Use in peteroleum drilling and production, paints, paper, cooling tower water, textile treatments were also reported.
5. The following parties indicated that there was no information available on uses: Croatia, Romania and Latvia



Figure 4‑1 Examples of several sources of PCP in the environment.

## Production and trade

1. Historically, according to the data profile of IRPTC (1983), 90 000 tonnes of PCP per year were produced globally. The Economist Intelligence Unit (1981) estimated world production to be of the order of 50 000-60 000 tonnes per year, based on the North American and European Community output. (UNEP/POPRC.7/INF/5). Global production of PCP during the 1990’s was 8,500 – 50,000 tons/year (UNECE 2008 cited in Hoferkamp et al. 2010).
2. Currently, PCP has either no uses or is banned in all E.U. member states, India, Indonesia, New Zealand, Russia and Switzerland. PCP is only allowed for wood preservation with additional restrictions and/or regulations in Belize, Canada, China and the United States. Other uses include agricultural, urban and industrial uses in Mexico, ready-for-use products in Nigeria; and snail elimination to control the spread of schistosmoniasis in China.
3. The only manufacturing site for North America is in Mexico which the Wood Preservation Industry indicated produced 7 257 tonnes/year in 2009 for the United States, Canada and Mexico.
4. 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., Columbia and Peru. Mexico reported importing PCP from the United States, China and Germany between 1997 and 2011. The highest importation estimate was for 2007 where 100 kg of PCP (2 695 000 dollars) was imported into Mexico. See Table 4-1.

Table 4‑1: Production, importation and exportation information from Mexico

|  |  |  |  |
| --- | --- | --- | --- |
| **Year** | **Production**  **(tonnes)** | **Import**  **(dollars)** | **Export**  **(tonnes)** |
| 1997 |  | 35 000 |  |
| 1998 |  | 5 000 |  |
| 1999 |  | 36 000 |  |
| 2007 |  | 2 695 000 (100 kg) | 3,670.689 |
| 2008 |  |  | 734.066 |
| 2009 | 6,610 |  | 6,471.932 |
| 2010 |  |  | 6,220.988 |
| 2011 |  | 1 410 000 | 6,414.246 |
| 2012 |  |  |  |

1. The U.S. Government reported that in 2002, approximately 4 990- 5 444 tonnes were used for utility poles, lumber and timbers. The U.S. reported that about 4 083 tonnes were imported and that 1361-1815 tonnes were produced domestically.
2. Canada estimated that approximately 2-8% of the PCP produced for North America is used in Canada. The amount of PCP used in Canada as estimated by sales information is provided in .

Table 4‑2: Sales information in Canada

|  |  |  |
| --- | --- | --- |
| **Year** | **Production**  **(pounds)** | **Production**  **(metric tonnes)** |
| 2008 | 819 190 | 372 |
| 2009 | 823 979 | 374 |
| 2010 | 1 096 418 | 497 |
| 2011 | 1 151 835 | 522 |
| 2012 | 1 183 903 | 537 |

1. In China in 2003, the annual national output reached approximately 3000 tonnes in 2003 (CESE, 2004; Tan et al., 2008 as cited in Zheng et al., 2012).
2. The following parties reported that they had no information on production: Croatia, Romania, Latvia,
3. The following parties reported that they did not produce PCP: Canada, Nigeria, Sri Lanka, Slovak Republic, Kiribati.
4. World import and export information was obtained from FAOSTAT from Mongabay 2013a, Mongabay 2013b, Mongabay 2013c and Mongabay 2013d are provided in , , and .

Table 4‑3: Quantities of Pentachlorophenol Salts Exported by Top 35 Countries. (Data comes from the [U.N. Food and Agriculture Organization's FAOSTAT database](http://faostat.fao.org/) and has been displayed with the permission of FAO. The data was downloaded on 02/16/2012.)

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

**Table 4‑4:** Quantities of Pentachlorophenol Salts Imported by Top 62 countries. (Data comes from the U.N. Food and Agriculture Organization's FAOSTAT database and has been displayed with the permission of FAO. The data was downloaded on 02/16/2012.)

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

Table 4‑5: Quantities of Pentachlorophenol Exported by Top 35 Countries. (Data comes from the [U.N. Food and Agriculture Organization's FAOSTAT database](http://faostat.fao.org/) and has been displayed with the permission of FAO. The data was downloaded on 02/16/2012.)

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

Table 4‑6: Quantities of Pentachlorophenol Imported by Top 35 Countries. (Data comes from the U.N. Food and Agriculture Organization's FAOSTAT database and has been displayed with the permission of FAO. The data was downloaded on 02/16/2012.)

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

## Releases

1. The following potential releases to the environment were identified in OSPAR (2004):

* Production of PCPL from PCP (manufacturing process);
* Treatment of wood (sapstain control agent);
* Impregnation of heavy-duty textiles and fibers;
* Use and disposal of PCP treated wood and textiles;
* Contaminated sites (former PCP production and wood preservation plants) and treatment of contaminated soil and groundwater;
* Natural sources or burning process.

1. Based on the current bans and restrictions in place, the releases listed above are no longer relevant to all countries and all situations. Specifically, the following information should be noted:

* Registered use on textiles was only reported for Mexico. However, the production information submitted by Mexico indicated that all PCP produced is used for wood preservation. No other country reported textile uses;
* Countries such as the United States and Canada have implemented regulatory programs to minimise worker and environmental releases for heavy duty wood preservation for treatment facilities and disposal. Burning of treated wood is not permitted in either country;

1. The United States compiled the following releases from their Toxic Release Inventory (TRI) On-site and Off-site disposed or otherwise released for 2011:

* Total On- and Off-site Disposal and other releases were reported to be 96,050 lbs (43,567 kg) – of which 89,970 lbs (40,809 kg) were reported as “on-site releases” and 6,080 lbs (2,757 kg) were reported as “off-site releases”.
* The majority of on-site releases reported (89,200 lbs) (40,460 kg) were classified as disposal to an on-site hazardous waste landfill (i.e., a RCRA Subtitle C landfill).
* The majority of off-site releases were classified as “unknown” (2,427 lbs) (1,100 kg), “RCRA Subtitle C landfill” (1,840 lbs) (834 kg), and “other landfills” (1,688 lbs) (765 kg).

1. Mexico reported the following losses: 17 776 kg (incineration), emissions to air were 38 kg in each year between 2006 and 2009 and emissions to soil were estimated to be 0.0029 kg (2005).
2. The following parties reported that they had no information on releases: Romania, Slovak Republic

# Fate and Behaviour of Pentachlorophenol and Pentachloroanisole

## Physical and chemical properties relevant to the environment

1. Pentachlorophenol is light brown to tan (Pure pentachlorophenol, however, is white needle-like crystals). It is a solid with a phenolic odor that is pungent when heated. It decomposes on heating in the presence of water, forming corrosive fumes (hydrochloric acid). Pentachlorophenol is nonflammable and noncorrosive. Sodium pentachlorophenate can be in the form of tan powder, pellets or briquettes with phenolic odour. It decomposes on heating, forming toxic fumes (chlorides and sodium oxide).
2. In the environment, NaPCP and PCPL dissociate/degrade to PCP.
3. The solubility of PCP in water varies with temperature (5-35 mg/L). The KOC values of 293 to 4000 indicate that PCP is likely to be slightly to moderately mobile when in soil as per the McCall et al., (1981) classification scheme. The variable KOC value indicates that in aquatic systems, the extent of partitioning to sediment will depend on environmental conditions. The recommended logKOW values of 5.12 and 5.18 indicate that PCP has the potential for bioaccumulation. The Henry’s law constant is estimated as 2.45 x 10-6, indicating the potential to volatilise from moist soils and water surfaces. Vapour pressure of 2 mPa indicates that PCP has intermediate to high volatility. Combined, these values indicate that aerial transport is possible.
4. PCA is sparingly soluble in water (0.24 mg/L). The Koc estimate (KOCWIN v.2.0 in EPA 2011) of 2474 (MCI method) and 13800 (Kow method) indicate that PCA is likely to be immobile or have slight mobility when in soils as per the McCall et al. (1981) classification scheme. The high KOC also indicate that in aquatic systems, PCA is likely to partition to sediment. The estimated log Kow is 5.30, (derived from KOWWIN v1.68, U.S. EPA, 2011) and the experimentally determined KOW is 5.45 (Opperhiuzen and Voors 1987) indicating PCA is very hydrophobic and has the potential to bioaccumulate. The Henry’s law constant for PCA is estimated as 1.94x 10-3 atm-m3/mole, using a group estimation method and 7.12 x 10-5 atm-m3/mole, using a bond estimation method (HENRYWIN v.3.2, U.S. EPA, 2011), indicating the potential to volatilise from moist soils and water surfaces. The modelled vapour pressure of 0.0458 Pa, using the modified Grain method (MPBPVP v1.43, U.S. EPA, 2011) and the vapour pressure of 0.0933 Pa (Dobbs and Grant 1980) indicates the potential for very high volatility. Combined, these values indicate that aerial transport is possible.

See Table 5‑1 for the summary of physical and chemical properties.

Table 5‑1: Comparative data for the identification and physico-chemical properties of PCP and PCA

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

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

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

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

**QSAR Predictions and Estimates for Persistence:**

1. QSAR estimates were derived from EPI (Estimation Program Interface) Suite 4.1 (U.S. EPA 2011) for both PCP and PCA. The output summaries are presented in Appendix II.
2. The half-life estimation most relevant for persistence classification is the primary biodegradation (Biowin 4: transformation of a parent compound to an initial metabolite). A summary of the model outputs for the BioWin QSAR estimates are presented in Table 5‑2. The results from the BioWin 4.0 output are shaded.
3. The Biowin model predicts similar numbers for both PCP and PCA. The Biowin output for PCP and PCA indicate that at the screening level, exposure estimates indicate that both chemicals would not undergo rapid aerobic or rapid anaerobic biodegradation. The half-life estimation for the primary biodegradation (Biowin 4: transformation of a parent compound to an initial metabolite) and the results indicate that the estimated half-life for both PCP and PCA is weeks-months. This classification is estimated at 37.5 days (alternate estimate of 61.8 days) by the EPISuite model for Fugacity modelling.

Table 5‑2: EPI Suite 4.1 predictions of rapid aerobic and anaerobic biodegradation of PCA and PCP

| **Model** | **Endpoint** | | **Remark** |
| --- | --- | --- | --- |
| **PCP** | **PCA** |
| **Probability of Rapid Biodegradation:** | | | |
| Biowin 1 (linear model) | -0.1755 | -0.1661 | Does not biodegrade fast (probability) |
| Biowin 2 (non-linear model) | 0.000 | 0.0002 | Does not biodegrade fast (probability) |
| **Expert Survey Biodegradation Results** | | | |
| Biowin 3 (ultimate biodegradation\*) | 1.6340 | 1.4885 | Recalcitrant (Months-longer)  (estimate: 180 days) |
| Biowin 4 (primary biodegradation\*\*) | 2.6765 | 2.6937 | Weeks-months  (estimate: 37.5 days (U.S. EPA default); 61.75 days (alternative default) |
| **MITI Biodegradation Probability** | | | |
| Biowin 5 (MITI linear model) | 0.0149 | 0.1046 | Not readily degradable |
| Biowin 6 (MITI non-linear model) | 0.0031 | 0.0049 | Not readily degradable |
| **Anearobic Biodegradation Probability:** | | | |
| Biowin 7 (anaerobic biodegradation potential) | -1.0946 | -1.0768 | Does Not Biodegrade Fast |
| **Ready Biodegradability Prediction:** | **NO** | **NO** |  |

\*Ultimate biodegradation is the transformation of a parent compound to carbon dioxide and water, mineral oxides of any other elements present in the test compound, and new cell material.

\*\*Primary biodegradation is the transformation of a parent compound to an initial metabolite.

Shaded row indicates the output value that most closely matches the soil persistence classification.

## **Abiotic transformation**

### **Soil (hydrolysis and phototransformation)**

1. PCP is hydrolytically stable at pH 4-9, precluding hydrolysis as a major transformation pathway in soil. Information from the mobility and metabolism studies indicate that phototransformation may increase the overall rate of PCP transformation in soil, however, it is not expected to be a major route of transformation, particularly in situations where biotransformation and binding to soil are the dominant processes. A summary of the abiotic radiolabelled studies are provided in .
2. Since PCA is formed via biomethylation, it is not expected to be formed from PCP via phototransformation or hydrolysis. If introduced onto the soil, it would not be expected to hydrolyse based on its chemical structure (Lyman et al 1982 in U.S. EPA 1992). No other information on the hydrolysis and phototransformation of PCA was found.

### Water

1. PCP is hydrolytically stable in water at pH 4-9, precluding hydrolysis as a major transformation pathway in the aquatic environment. Abiotic transformation of PCP in water will occur mainly through phototransformation.
2. PCP phototransformed rapidly with half-lives of 13-20 minutes in sterile aqueous pH 5, 7 and 9 buffer solutions irradiated with natural sunlight. In surface water, PCP is expected to undergo photoreductive dechlorination to produce tetrachlorophenols (2,3,4,6- and 2,3,5,6-) and trichlorophenols. The tetrachlorophenols further react (with the addition of a hydroxyl group) to form tetrachlorobenzenediols (tetrachlororesorcinol at pH 5, 7 and 9; and tetrachlorohydroquinone and tetrachlorocatechol at pH 5 and 7), which are quickly oxidised and may undergo further dechlorination and/or ring cleavage to eventually fomr chloranil, hydroxyquinones and the major transformation product, 2,3-dichloromaleic acid (DCM). Although the photolysis of DCM is less rapid than that of PCP and the intermediate transformation products, the acid will eventually transform to CO2, HCl and small organic fragments (Wong and Crosby, 1981). A summary of the abiotic radiolabelled studies are provided in.
3. The irradiation of PCP at high concentrations (1000 ppm) in water has been observed to produce octachlorodibenzo-p-dioxin (OCDD); the highly toxic compound, 2,3,7,8-tetrachlorodibenzo-p-dioxin was not detected (Crosby et al., 1978).
4. In a study conducted by Baker and Hites (2000), PCP in a pH 5.5 solution was irradiated (wavelengths above 290 nm). PCP was converted to PCDDs and PCDFs at the following concentrations: 0.1% OCDD (TEQ-0.0003), 0.01% for HpCDD (TEQ 0.01).
5. Liu et al. (2002) also investigated the phototransformation of PCP on soil under UV light. Only OCDD, and H7CDD were detected. H7CDD was reported to be 1% of the OCDD produced. Total PCDD reached their maxima for all soil conditions between 100 and 125 minutes, followed by subsequent rapid degradation. See Figure 5‑1.

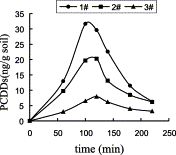


Figure 5‑2: Phototransformation of PCDDs by photolysis of PCP on soils surface (the amount of PCDDs was actually the total amount of OCDD and H7CDD) (Liu et al., 2002)

1. PCA was not detected in either abiotic study conducted with PCP. However, if introduced into an aquatic system, PCA would not be expected to hydrolyse based on its chemical structure (Lyman et al 1982 in U.S. EPA 1992). No other information on the hydrolysis and phototransformation of PCA was found.

### **Air**

1. The Henry’s law constant for PCP is estimated as 2.45 x 10-6 atm-m3/mole, indicating the potential to volatilise from moist soils and water surfaces. In the atmosphere, volatilized PCP may undergo photolysis or may react with photochemically produced hydroxyl radicals. For PCP, the rate of photolysis is estimated to range from 1.4% per h in winter to 6% per h in summer (half-lives 12-44 h; clear weather conditions, mid-day) (Slooff et al. (1991)). This is considerably faster than the half-life for PCP in air based on reactions with the OH- radicals which is 19.43 days with an atmospheric (OH) concentration of 1.56 x 106 OH/cm3as calculated by the AOPWin v1.96 in US EPA, 2011.
2. The Henry’s law constant for PCA is estimated as 1.94x 10-3 atm-m3/mole, using a group estimation method and 7.12x 10-5 atm-m3/mole, using a bond estimation method (HENRYWIN v.3.20 in U.S. EPA 2011). This value indicates that that PCA has the potential to volatilize from water or moist soil. Based on this value for Henry's law constant, 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. 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 to be 6.9 days (Estimated by Group SAR Method in U.S. EPA 2011).
3. PCA can be photo-oxidised in the atmosphere through reactions with hydroxyl (OH) radicals. The calculated half-life for PCA based on this reaction is 9.8 days, with an atmospheric (OH) concentration of 1.56 x 106 OH/cm3 (AopWin v1.96 in U.S. EPA, 2011). No experimental data are available on atmospheric degradation.

Table 5‑3: Abiotic transformation studies conducted with radiolabeled PCP reported in the Annex E information Submitted by the U.S. and Canada, 2013.

| **Fate Study** | **Transformation products identified** | **Major** | **PCA** |
| --- | --- | --- | --- |
| Hydrolysis  pH 4,5  pH 9  pH 7 | Volatilisation of PCP was observed  None (PCP was stable)  None (PCP was stable) | none | Was not detected and is not expected to be produced. |
| Photolysis at pH 5, 7 and 9  (radiolabeled study, recovery >96%) | Tetrachlorohydroquinone, tetrachlorocatechol. Tetrachlorosorcinol and dichloromaleic acid and/or anhydride | dichloromaleic acid and/or anhydride (exceeding 10% and accumulating at study termination-68-100% at study termination) | Was not detected and is not expected to be produced. |
| Photolysis in air | 2,3,5,6-TeCP and 3 additional polar photodegradates. |  | Was not detected and is not expected to be produced. |
| Photolysis in Soil | -3 minor products (<10% and were not identified)  -up to 2.25% were bound residues  -0.032% were volatile T.P.s |  | Was not detected and is not expected to be produced. |

## **Biotransformation**

### **Aerobic Biotransformation**

1. Under aerobic conditions, large numbers of PCP-degrading bacteria have been identified and several degradation pathways that have been identified, depending on the experimental or environmental conditions. The formation of PCA from PCP has been observed primarily under aerobic conditions in the presence of white-rot fungi. The formation of PCP from PCA has been observed at lower levels when tested in mixed-microflora systems. The following is a summary of aerobic biotransformation information conducted with natural or mixed microflora provided at Annex E. A summary of the pathways and transformation products produced under aerobic conditions is found in , . describes the studies where the formation and/or degradation of PCA was observed.
2. In a radiolabeled aerobic soil biotransformation study reviewed by the U.S. EPA (U.S. EPA 2008), PCP degraded in aerobic sandy loam soil with an observed half-life of 7-14 days; the calculated first-order half-life was approximately two months. The major degradation products of PCP were 2,3,4,5-tetrachlorophenol, 2,3,6- and 2,4,6-trichlorophenol and CO2. Bound residues accounted for up to 64% of the radioactivity. Additional analysis of the bound residues indicated that 76%, 21% and 3% of the bound residues were associated with the humin, fulvic and humic fractions, respectively. In a supplemental study in which aerobic soils were subsequently flooded and kept under anaerobic conditions, up to 10% of the bound residues were released as PCP. PCA was not one of the standards used to compare with the observed transformation products, however, all other major transformation products were identified and there were no significant amounts of unidentified radioactivity and overall recoveries were >91.3%. A summary of the results of the biotransformation study are found in .
3. In a radiolabelled aerobic water-sediment study reviewed by the U.S. EPA (U.S. EPA 2008), radiolabeled PCP degraded in aerobic flooded sandy loam soil with an observed half-life of 14 days; the calculated first-order half-life was 4.9 days. The major degradation products of PCP were 3,4-dichlorophenol, various isomers of tetrachlorophenol and trichlorophenol. Bound residues and volatiles accounted for up to 41% and 0.9% of the radioactivity, respectively. PCA was not one of the standards used to compare with the observed transformation products, however, all the major transformation products were identified, there were no significant amounts of unidentified radioactivity and overall recoveries were good (88-117%). A summary of the results of the biotransformation study are found in .
4. Some references available in the open literature, including EHC 71 (1987), Englehardt et al. (1986), Kaufman (1978) and U.S. EPA (2008) cite the Murthy et al. (1979) study as evidence of PCA being formed as a major soil metabolite under aerobic conditions. However, a critical review of the study has shown that this was not the same conclusion reached by the original authors. The study authors concluded that the major degradative pathway of PCP is through reductive dechlorination and that the production of PCA was greater in aerobic than in anaerobic soils. It should be noted that closer examination of the data from the PCP-treated aerobic soil study showed that only 14.7% of the applied radioactivity treated was extractable, 51.5% of which was identified as PCA and 33.4% as PCP. This would result in a maximum concentration of PCA of 7.5% of the total applied radioactivity and not 51.5% as reported in some published literature summarising Murthy et al. (1979). The remaining radioactivity was attributed as follows: 44.6% to bound residues and 40.7% to unaccounted radioactivity. There is also some uncertainty in the results of this study since some samples were methylated prior to analysis thereby possibly converting some of the PCP to PCA. It is expected that the PCA concentrations reported represent a conservative estimate as PCA concentration may have been overestimated.
5. The authors of the Murthy et al. (1979) also speculated that some of the PCA residues may be incorporated into soil components as bound residues, however, this has not been confirmed by any of the studies included in this literature search. The Government of Canada did not find any published literature or registrant-conducted studies where bound residues were released and identified as PCA. In fact, a registrant-conducted study using radiolabeled PCP showed that under anaerobic conditions, small amounts (<10%) of bound residues were released under anaerobic conditions as PCP, not PCA.
6. In a laboratory experiment conducted by Haimi et al. (1993), chloroanisoles (2,3,4,6-tetrachloroanisole; containing approximately 10% PCA) were added to soil with and without earthworms in test vessels. The concentrations of 2,3,4,6-tetrachloroanisole and pentachloroanisole in earthworms and soil decreased in soil with an approximately half-life of 5 weeks in both the soils with and without earthworms. There was evidence of demethylation of PCA to pentachlorophenol and tetrachloroanisole to tetrachlorophenol. The overall rate of disappearance was attributed to degradation, metabolism to unidentified compounds and to the formation of non-extractable compounds. The authors noted that the high rate of metabolism and/or degradation of the chloroanisoles were corroborated by the high respiration activity found with the high concentrations of chloroanisoles.
7. In a study conducted by Kuwatsuka and Igarashi (1975), the transformation of PCP in relationship to soil properties was studied under upland and flooded conditions using 10 different soils collected from rice fields, upland fields and one sample of a subsoil from the forest. The authors reported that transformation of PCP in soils was faster under flooded conditions than upland conditions. The transformation under flooded conditions was more rapid in soils collected from rice fields than in those from adjacent upland fields. The reverse was true under upland conditions. The transformation rate was highly correlated with organic matter content in the soil. The rate was slightly correlated with the clay mineral composition, free ion content, phosphate absorption coefficient and C.E.C., but hardly at all with texture, clay content, degree of saturation, soil pH and available phosphorus content. The transformation products detected included tetrachlorophenol (maximum of 3% of the applied PCP at day 5), trichlorophenol (maximum of 2.5% of the applied PCP at day 5), PCA (maximum of 1% of the applied PCP at day 5 with a decline to 0.5% at 35 days) and various other transformation products detected in trace amounts. Although not reported by the study authors, an observed half-life of approximately 30 days for PCA can be estimated from the graphs provided in the study. This is considered a conservative estimation since PCP was present in the test system over the course of the study and would have behaved as a continuous source of PCA, i.e., formation and degradation would be occurring simultaneously. The authors indicated that PCA formation and subsequent degradation back to PCP was reported previously by Kuwatsuka and Igarashi (1971, 1972 and 1973), however, these studies are only available in Japanese and were therefore not reviewed.
8. Chung and Aust (1995) conducted a degradation study of PCP in soil in a closed system with radiolabelled material with a species of white rot fungi where the production of PCA was observed. Although significant amounts of PCA were produced, no volatile organic compounds, including PCP or PCA were detected in the volatile traps. Degradation half-lives were reported in the study, but are not reported here since the study was conducted with an isolated white rot fungus instead of mixed soil microflora as required under standard protocols.
9. In another closed system experiment, Klowskowski et al. (1981) conducted a study examining the transformation of PCP and ten other substances in soil-plant systems. PCP was found to be non-persistent in both outdoor and laboratory test systems. All volatile products captured in the laboratory test were determined to be carbon dioxide; no other volatile products, including organic volatile products (e.g., PCA), were detected.
10. D'Angelo and Reddy (2000) examined strategies to enhance biotransformation of PCP in a spectrum of wetland soils. Soils were investigated under laboratory conditions, which included manipulations of electron acceptors. Aerobic PCP transformation initially produced small amounts of PCA. However >75% of both chemicals disappeared in 30 d from five soils. Under methanogenic conditions, PCP was reductively dechlorinated to yield a mixture of tetra-, tri-, and dichlorophenols in eight soils, with rates strongly correlated to measures of electron donor supply (total C, N, organic C mineralization rates) and microbial biomass.



Figure 5‑3: Experimental results demonstrating that transformation of PCP is due to microbial transformation (D’Angelo and Reddy 2000).

1. Mardonesa et al. (2009) investigated the uptake of 2,4,6-tribromophenol (TBP), PCP, and its metabolite PCA from contaminated sawdust from the forest industry in horticultural products such as apples, raspberries, and fodder maize for cattle feed. The samples were obtained from Bı´o-Bı´o Province in South Chile between 2002 and 2006. Initial PCP concentration in the soil was 91.5 mg/kg. It was not detected in the harvested plants. PCA was not detected in the soil prior to planting, but was present at the end of the study at low levels (10 µg/kg).
2. Frisbie et al. (1997) investigated PCP and 2,3,4,6-tetrachlorophenol-contaminated soil samples from several depths at a former wood treatment site. Soils were placed under varying conditions in the laboratory to determine the anaerobic and aerobic potential for biodegradation of chlorophenols. PCP degradation occurred in both anaerobic and aerobic soil samples. In aerobic soil samples, PCP concentrations decreased more than 87% within 18 days. Due to the wide variety of metabolites from aerobic degradation, including chlorinated hydroquinones and catechols, no attempt was made to analyze for specific aerobic metabolites. However, no anaerobic metabolites (lesser-chlorinated phenols) were detected. Reductive dechlorination of PCP in anaerobic samples resulted in the accumulation of 3-chlorophenol.

### Anaerobic Biotransformation

1. Under anaerobic conditions, reductive dechlorination is likely the major degradation pathway of PCP. PCA was not produced in significant amounts under anaerobic conditions. The following is a summary of anaerobic biotransformation information conducted with natural or mixed microflora provided at Annex E. A summary of the pathways and transformation products produced under anaerobic conditions is found in . describes the studies where the formation and degradation of PCA was observed.
2. In a flooded sandy loam soil, radiolabeled PCP degraded with a half-life of 1-2 months (calculated first-order half-life of 34 days) (U.S. EPA 2008 and U.S. EPA 2008). Various isomers of tetrachlorophenol and trichlorophenols were formed as major transformation products. Volatiles were negligible (1.4% of the applied radioactivity) and nonextractable residues were 7.8% of the applied radioactivity. PCA was not one of the standards used to compare with the observed transformation products, however, all the major transformation products were identified. There were no significant amounts of unidentified radioactivity and overall recoveries were only slightly outside the acceptable range 78.2-135%. A summary of the results of the biotransformation study are found in .
3. Murthy et al. (1979) observed small amounts of PCA (5.3%) in test soils treated with radiolabeled PCP exposed to anaerobic conditions. The authors concluded that the principal degradation pathway of PCP in anaerobic soil would seem to be by progressive dechlorination to tetra- and tri-chlorophenols, with lesser formation of pentachloroanisole. In addition, Murthy et al. (1979) conducted a supplemental study with soils treated with PCA incubated under both aerobic and anaerobic conditions to assess degradation. The study authors reported that 42% of the applied PCA was transformed to PCP in 24 days. In the anaerobic portion of the study, 98.8% of the radioactivity was recovered in the soil indicating that any losses due to volatilisation were insignificant. Since recovery was good and the successor transformation product (PCP) was identified, the half-life estimate can be attributed to degradation, not dissipation or volatilisation. It should be noted, however, that the analytical sample work-up uses a methylation step prior to analysis thereby possibly converting some of the PCP to PCA. It is expected that the PCA concentrations reported represent a conservative estimate as any additional PCA produced during the sample analysis would contribute to a slight overestimation of PCA concentrations.
4. D’Angelo and Reddy (2000) did not observe any PCP transformation to PCA under anaerobic soil conditions in 10 different soils under various anaerobic conditions.
5. Weiss et al. (1982) found that, one year after application of PCP to flooded soil in the laboratory, most of the radioactivity was found as bound residues. The authors identified conversion products which indicate at least four different reaction mechanisms: reductive dechlorination; methylation; conjugation; incorporation into insoluble macromolecules. Only 0.09% of the applied radioactivity was measured as PCA. Other lesser chlorinated anisoles were also found (tetra- and tri-chloroanisoles).
6. Kuwatsuka and Igarashi (1975) found that PCP was rapidly degraded in soil under flooded conditions. Transformation products included tetrachlorophenol (maximumof 4% of the applied PCP at day 5), trichlorophenol (maximum of 2% of the applied PCP), PCA (maximum of 2% of the applied PCP at day 10 and declined to 1% of the applied PCP at 30 days) and various other transformation products detected in trace amounts. Although the authors did not report half-lives, PCA declined to half of its maximum concentration in approximately 20 days. Since PCP was present in the test system over the course of the study, both formation and degradation of PCA would have been occurring simultaneously, thereby inflating the observed half-life values.
7. Charnay et al., (2000) examined the effect of redox conditions on the behavior of three chemical different organic pollutants (atrazine, 2,4-dichlorophenoxyacetic acid, and PCP) was investigated during laboratory incubation under controlled condition in a loamy clay soil sampled in a wetland located at Grignon (Yvelines, France). The three molecules were 14-labelled which allowed to follow the behavior of the pollutants including their meneralization (measurement of 14C-CO2) and their residue evolution. The soil microflora degraded PCP without complete mineralization and the major degradation products identified was 2,4,5-trichlorophenol. The incubation of soil columns saturated in water showed that strongly reducing conditions increased dechlorination of PCP.The principal degradation pathway of PCP in anaerobic soil is progressive dechlorination to tetra- and tri-chlorophenols. The formation of pentachloroanisole appears to be a minor pathway. When present in anaerobic soil, PCA is likely to convert to PCP and follows the PCP degradation pathway.
8. In Chen et al.(2003), the feasibility and effectiveness of an anaerobic sludge digestion process was evaluated. Two labolatory-scale digesters mimicking the commonly used anaerobic sludge digester in a mumicipal wastewater treatment plant were operated to treat PCP. Results shows that up to 0.98 mM of chemical PCP dissolved in acetone and 0.6 mM soil PCP from a contaminated site were treated at nearly 100 and 97.3 % efficiencies, respectively. PCP dechlorination followed two major pathways; PCP to 2,3,4,5-TeCP to 2,3,5- or 3,4,5-TCP to 3,5-DCP and PCP to 2,3,5,6-TeCP to 2,3,5-TCP to 3,5-DCP to 3-MCP. The 3-MCP was not present until 26 days after the first addition of PCP, which also concluded the end of the sludge acclimation process to PCP. 95% of the added PCP was transformed to 3-MCP, 4.5% to 3,4-DCP, and 0.5% to 3,5-DCP and about 20% of the PCP by-products remained in liquid and the rest absorbed on sludge solids.
9. Chen et al. (2010) examined the metabolism of PCP in batch experiments using coupled sludge granules under various dissolved oxygen concentrations was investigated. Results indicated that the oxygen condition in serum bottles has a significant effect on the microorganism metabolism. A greater degree of mineralization of PCP was achieved under oxygen-limited conditions, producing trichlorophenol (TCP), dichlirophenol (DCP) and monochlorophenol (MCP) as intermediated and chloride as one of the final products. Reductive dechlorination was identified as the primary pathway for the PCP degdradation. Under strictly anaerobic or slightly oxidative conditions, the reductive dechlirination of PCP led to an accumulation of TCP. Under aerobic conditions, PCP degradation was less significant due to the hindered reductive chlorination in the presence of oxygen.
10. No laboratory studies examining the behaviour of PCA in natural sediments were found.

### Formation of PCA from Isolated Fungal and Bacterial Biotransformation Studies under Aerobic Conditions

1. Bioremediation programs have been implemented worldwide to detoxify soil and many studies exist examining conditions to optimise PCP degradation. The following is a summary of the studies reviewed.
2. A large number of aerobic bacteria utilize chlorophenols as carbon and energy sources (e.g., Pseudomonas sp., Mycobacterium sp., Sphingomonas sp., Rhodococcus sp., Flavobacterium sp., Arthrobactorsus sp.). Under aerobic conditions, chlorohydroquinone will be formed as an intermediate in several degradation pathways. Hydroxylation in the para position of PCP occurred by monooxigenases (i.e., PCP 4-monooxygenase) resulting in the formation of p-tetrachlorohydroquinone (TeCHQ). TeCHQ is sequentially dechlorinated and causes metabolites such as 2,6-dichloro-1,4-hydroquinone (TeCHQ). TeCHQ is sequentially dechlorinated and causes metabolites such as 2,6-dichloro-1,4-hydroquiquinone (2,6-CDHQ) (Field and Alvarez, 2007). This information is summarised in .
3. Yang and Lee (2007) showed Sphingomonas chlorophenolica can completely degrade 200 mg/L of PCP within 23.2 hours and release 130.3 mg/L chloride when acclimated with PCP before testing.
4. Zaborina et al. (1997) investigated the fate of PCP in soil under natural conditions. PCP significantly decreased when soil was inoculated by Streptomyces rochei 303, a strain-destructor of chlorophenols. The products of PCP transformation, such as tetra- and trichlorophenols, oentachlorobenzene, chlorinated dioxins, were identified after the first month of the experiment.
5. Nguyen et al. (2003) described the process for the identification of bacteria Enterobacter cloacae, which has recently been discovered to pose the ability to metabolize PCP and proposed a pathway for its metabolism. The reductive dechlorination of PCP by Enterobacter cloacae produced dechlorinated byproducts. In order for the bacteria Enterobacter cloacae, to dechlorinate PCP, it must possess at least one or two types of enzymes. One enzyme (or group of enzymes) recognizes and cleaves the chlorine at the mata- position forming 2,3,4,6-tetrachlorophenol.
6. Pentachloroanisole formation from PCP metabolism is well established in fungi (Cserjesi and Johnson 1972; Lamar and Dietrich 1990; Okeke et al. 1997; Tuomela et al. 1998 and Chung and Aust 1995). PCP methylation to PCA can be mediated by common aerobic fungi such as Trichoderma virgatum, Phanerochaete chrysosporium, Phanerochaete Sordida, Lentinula edodes (Walter et al. 2004; Lamar et al. 1990; Okeke et al. 1997; Cserjesi and Johnson, 1971). Some white-rot fungi are effective at o-methylating chlorinated phenols (Walter et al. 2004). There are two strains of white rot fungi (Phanerochaete spp.), P. chrysoporium and P. sordida, which have been studied extensively for bioremediation purposes.
7. High transformation rates of PCP to PCA were observed in both liquid cultures (Walter et al, 2004; Badkoubi et al., 1996) and soil samples incubated with P. chrysosporium (Pfender et al., 1997). A high transformation rate (85%) of PCP to PCA was also observed in the liquid cultures inoculated with Trichoderma haruanum strain T.h.2023 (Rigot and Matsumura, 2002). In the presence of P. chrysoporium and P. sordida, PCP is rapidly methylated to PCA which is further mineralised. Other white-rot fungi such as Trametes versicolor only converted PCP to PCA in small or trace amounts (Walter et al. 2004; Walter et al. 2005; Ford et al., 2007, Machado et al., 2005).
8. 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).
9. In Rubilar et al.(2007), the degradation of PCP by two white-rot fungi: Bjerkandera adusta and Anthracophyllum discolor was examined in soil slurry culture using different initial PCP concentrations (100, 250 and 350 mg of PCP/kg soil). The highest PCP degradation was attained by A. discolor (95% after 28 days). The main intermediate was PCA and 4-dimethoxybenzaldehyde. The PCP degradation pathway starts with methylation and formation of PCA. The second reaction is hydroxylation to form tetrahydroquinone (TCHQ), which is then methylated to tetrachloro-1,4-dimethoxybenzene, followed by successive dechlorination reactions to form 2,5-dichloro-1,4-dimethoxybenzene and 2-chloro-1,4-dimethoxybenzene, respectively. A series of demethoxylation, carboxylation, reduction, and methylation reactions are conducted to form 3,4-dimethoxybenzaldehyde, and at the end of the pathway, complete degradation of PCP and formation of CO2 is achieved.
10. In Rigot and Matsumura (2002), the rhizospere competency of Trichoderma haruanum strain T.h.2023 in corn roots in liquid culture and in soil. In liquid culture 85% of the PCP was converted to PCA. Under soil conditions, 13.5% of PCA was formed after 14 days. The authors indicated that PCA was not the terminal metabolite in the case of soil corn rhizosphere unlike in the case of the liquid culture. In soil, PCA was either a metabolic intermediate or a temporary bypass reservoir for the purpose of rapidly eliminating PCP.
11. Many differences were noted in the results obtained from various fungal studies depending on the experimental design and the species tested. Some differences existed between the degradative pathways observed in media inoculated with a mixed microflora and those inoculated with isolated microbial cultures; the amount of volatile organics reported in studies conducted with liquid culture versus soil and field-scale bioremediation tests (e.g., Walter et al. 2004; Walter et al. 2005; Rigot and Matsumura, 2002); the amount of volatile organic compounds when volatile traps were placed inside or outside the test vessels (Lamar et al. 1990; Badkoubi et al. 1996). As an example, Lamar et al. (1990) found that despite an apparently superior ability to mineralise PCP in liquid culture, P. sordida 13 did not deplete PCP from a sterile soil as rapidly or to as great an extent as did P. chrysosporium. However, rates and extents of PCP depletion by the two fungi in a contaminated field soil were similar. Also, the loss of PCP via mineralisation in soils inoculated with P.chrysosporium was negligible (i.e., <2%). Since loss via mineralisation is not a major transformation process of PCP depletion in soils inoculated with white-rot fungi, a superior ability to mineralise PCP in liquid culture does not appear to be useful for screening fungi for remediating PCP-contaminated soil. As such, these studies should be interpreted with caution and their relevance to the actual environmental field should be considered.

Liquid Culture:

1. Studies showing the production of PCA as a major volatile product using liquid media inoculated with P. chyrsosporium include Walter et al (2004), Badkoubi et al. (1996), Okeke et al. (1994) and Lamar and Dietrich (1990).
2. In a study to determine growth substrate selection for white-rot fungi, Walter et al. (2004), conducted an additional experiment using liquid medium under laboratory conditions. The authors reported an unusually high amount of volatile in the traps (26-95% of the applied PCP; almost all PCP with traces of PCA). In the P.chrysosporium culture, 75% of the volatilised residues were attributed to PCA. The authors speculated that the large volatile fraction measured in the liquid culture may have been an artefact of the experimental design since the high volatile release seen in the liquid cultures was not observed in the soil microcosms (in the soil microcosms, volatile fractions were reported in the range of 1-2% of the applied).
3. Similar observations were also made by Badkoubi et al. (1996) examining P. chrysosporium in liquid culture. After 12 days, 82% of the PCP was volatilised as PCA. If the fungus is oxygen-limited for lignin peroxidise production, it will convert most of the PCP to the volatile PCA, the only volatile compound detected in the presence of P. chrysosporium in this experiment. When sufficient oxygen was available, the extent of mineralisation was much greater, up to 32%. Higher mineralisation was observed when the volatile transformation products had a chance to equilibrate between the solution and the headspace. Immediate removal of the volatile transformation products reduced PCP mineralisation. Therefore, the PCA remaining in the solution and PCP will be mineralised and produce more 14CO2 over time compared with the case when all PCA is depleted from the liquid and sorbed on the polyurethane volatile trap.

Soil:

1. Chung and Aust (1995) found that both PCP and PCA are readily mineralised in the soil by P.chrysosporium in soil. The rate of degradation increases with increasing concentration of PCA from 50 to 1600 ppm. At 100 ppm in soil, PCP was depleted in 18 days while 40% of the initial 100 ppm PCP was found as PCA. At 800 ppm, about 44% of the PCP remained after 18 days, but only about 10% of it appeared as PCA, and about 9% of the original 800 ppm PCP was mineralised. The rate of mineralisation of PCA increased with increasing concentration of PCA. See Figure 5‑4. Essentially no radioactivity (<0.05%) was found in the volatile organic traps and the aqueous fraction during the mineralisation of either PCP or PCA. It would therefore appear that the rate of methylation is rate-limiting at high levels of PCP, but significant rates of PCA degradation still occur. The authors concluded that P. chrysosporium can degrade PCP efficiently and produce no harmful intermediates during the degradation of PCP in soil and that both PCP and PCA are readily mineralised in soil.

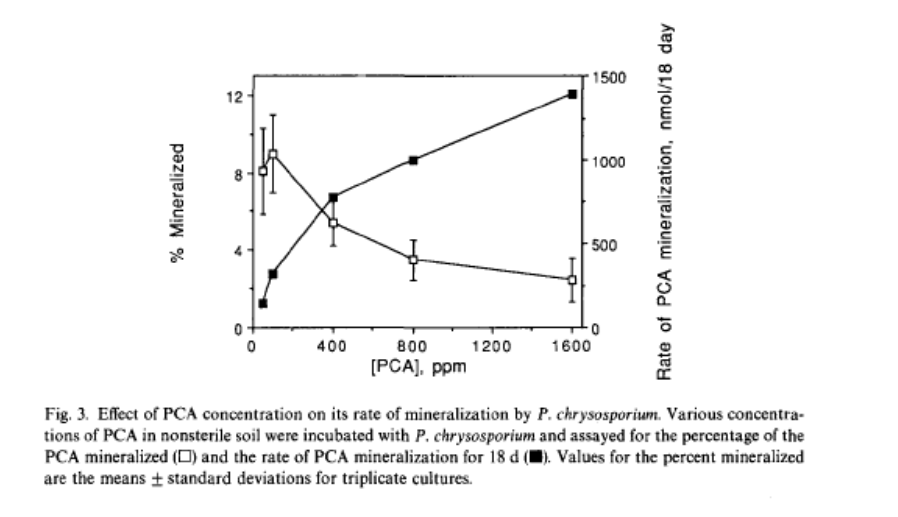


Figure 5‑5: Effect of PCA concentrations on the rate of mineralization by *P. chrysosporium*.

1. In Lamar et al. (1990), the depletion of PCP by P. chrysosporium and P. sordida occurred in two-stages. In the first stage, the rapid deletion of PCP coincided with an accumulation of PCP. At the end of the first stage, 64% and 71% of the PCP was converted to PCA in P. chrysosoporium and P. sordida cultures, respectively. In the second stage, levels of PCP and PCA were reduced by 9.6 and 18%, respectively in soil inoculated with P. chrysosporium and by 3 and 23%, respectively in soil inoculated with P. sordida.
2. In Pfender et al.(1997) the degradation of PCP by two bacteria (Pseudomonas and Flavobacterium) and a fungus (Phanerochaete sordida) in soil was examined with 14C-PCP. Over 65% of the available 14C-CO2 from soil treated with Pseudomonas SR3 or Flavobacterium, and most of the mineralisation occurred in the first 4 days. P. sordida transformed much of the labeled PCP to PCA (62% by day 56). All of the measured PCA was recovered in the soil. Radioactive recovery was good and the authors noted that volatilisation of PCP or any transformation products was low in all treatments (≤0.15% volatilisation was observed in all treatments).

Other organisms:

1. The production of PCA from PCP has been observed in other isolated species of soil microflora. Rott et al. (1979) found PCA was produced in very small yields (<0.005%) in 5 of 10 bacterial strains tested. Haggblom et al. (1988) found that all strains of Rhodoccoccus mycobacterium tested initiated degradation of chlorophenols by para-hydroxylation, producing chlorinated-hydroquinones that were further degraded. Strains also o-methylated the chlorinated phenols, guaiacols, syringols and hydroquinone. This reaction occurred in parallel to the degradation reaction, but was slower. Okeke et al. (1993;1994;1997) found that transformation of PCP to PCA is an important route of PCP depletion during the early stages of PCP biotransformation by L. edodes (shiitake mushroom). Trace or small amounts (<10%) PCA was detected in PCP contaminated soil or liquid culture when inoculated by Trametes versicolor (white-rot fungi). CO2 was detected in these studies, which suggests that mineralisation is likely to occur during the degradation of PCP (Walter et al., 2004, Ford et al., 2007, Machado et al., 2005).

### Summary of Biotransformation:

1. Under aerobic conditions, large numbers of PCP-degrading bacteria have been identified and there are several pathways for degradation of PCP, depending on the experimental or environmental conditions. Under anaerobic conditions, reductive dechlorination is likely the major degradation pathway of PCP. PCA can be generated from PCP as a result of methylation of PCP in the presence of white rot fungi (i.e., Panerochaete chrysosporim) under aerobic conditions.
2. Several studies show evidence of formation, degradation pathways and successor transformation products (Haimi et al. 1993; D’Angelo and Reddy 2000 and Kuwatsuka and Igarashi 1975; Rubilar et al., 2007; Rigot and Matsumura 2002).
3. From these studies, observed half-lives for PCA are between 20-35 days in aerobic soils with mixed microflora, half-life estimates for PCA derived from studies conducted with PCP as a starting material were often confounded by simultaneous formation and degradation and should be considered upper-bound estimates. There is also uncertainty in estimates as this was based on low or trace amounts of PCA produced.
4. In the Haimi et al. (1993), D’Angelo and Reddy (2000), Mardones et al. (2009) and Kuwatsuka and Igarashi (1975) papers, it is unclear whether vessels were securely sealed over the course of the study. Based on the volatility of PCA observed in several laboratory studies conducted with liquid media, additional information was considered to assess whether half-lives could be characterised as representative of transformation/degradation or dissipation/volatilisation.
5. Analysis of the information cited in this document indicates that PCA was only found in the volatile fraction of laboratory studies conducted in liquid culture (e.g., Badkoubi et al. 1996; Walter et al. 2004; Rigot and Matsumura, 2002). PCA was not volatile when under similar test conditions conducted with soil using radiolabeled material in closed test systems with volatile traps (Walter et al. 2005; Chung and Aust 1995; Pfender et al., 1997). Although there is still uncertainty whether some studies were properly sealed, it is unlikely that observed dissipation was due to losses of PCA to volatilisation.
6. The principal degradation pathway of PCP in anaerobic soil and sludge is progressive dechlorination to tetra- and tri-chlorophenols. The formation of pentachloroanisole appears to be a minor pathway. Most studies reviewed showed no formation of PCA under anaerobic conditions.

Table 5‑4: Biotransformation studies conducted with radiolabeled PCP reported in the Annex E information Submitted by the U.S. and Canada, 2013

| **Biotransformation Study** | **Soil transformation products** | **Volatile transformation products** | **Bound residues** | **Comment** |
| --- | --- | --- | --- | --- |
| Aerobic Soil  (1 yr, guideline study) | TeCP (2.4%),  TCP (4.5%), both degraded by study termination.  -no PCA standard, but good recovery (91.3%). | Volatile-27.3%  CO2 (26%),  TCP (<1%) | - 64%: 76%-humin  21%-fulvic  3%-humin | Observed half-life of 7-14 days; the calculated first-order half-life was approximately two months; the majority of radioactivity (64%) was bound residues. No chloroanisoles were detected (TeCA, DCA, para- and meta- chloroanisoles). No PCA standard was used for degradate identification. |
| Anearobic Flooded Soil (aerobic, followed by flooding) | TeCP (d), TCP (s), 2-chloro-hydroquinone (i) and mucochloric acid (i) (up to 14% total) | Aerobic:  4.47% [CO2 : 4.06%, PCP: 0.26%, unidentified: 0.15%]  Anaerobic:  1.85% [CO2 : 0.54%, PCP: 0.80%, unidentified: 0.51%] | Up to 44.8% (19 days) and then decreased to 38% (60 days)  52%-humin  38%-humic  11%-fulvic | <10% of bound residues were released under anaerobic conditions (measured as PCP)  Mean mass balance was 96.5% |
| Aerobic Aquatic Biotransformation | TeCP (10%),  TCP(83%) (d), dichlorophenol (37%) (i) | <1% | 40.9% (study termination) | Mass balance:  88.1-117% |
| Anearobic aquatic sediment | TeCP(10%)(d) and TCP(78%)(i) | 2.1% | 7.8% | Mass balance:  78.2-135% |

(i) increasing in concentration by study termination

(d) decreasing in concentration by study termination

(s) stable

Table 5‑5: Summary of PCP aerobic and anaerobic biotransformation information submitted at Annex E

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Study Description** | **Half-life or description of persistence (trend)** | **Transformation Products** | **Remark** | **References** |
| **Aerobic Conditions** | | | | |
| Aerobic soil | 7-14 days | tetrachlorophenol, trichlorophenols  bound residues  CO2 |  | US EPA, 2008 |
| *Spingomonas chloropehnolica*  200 mg/L | Completely degraded within 23.2 h |  |  | Yang and Lee, 2007 |
| *Streptomyces rochei* 303 |  | tetra- and trichlorophenols, oentachlorobenzene, chlorinated dioxins |  | Zaborina et al., 1997 |
| PCP and 2,3,4,6-tetrachlorophenol contaminated soil placed under varying conditions (aerobic) | PCP concentrations decreased 87% within 18 days | chlorinated hydroquinones, catechols. | No attempt was made to analyse for specific aerobic transformation products | Frisbie et al., 1997 |
| Various conditions and species |  | Pentachlorophenol acetate (major);  tetrachlorophenol, pentachloroanisole, tetrachloroanisole, tetrachlorocatechol; tetrachlororesorcinol; tetrachlorohydroquinone dimethylether | Pentachlorophenol is transformed by a number of ubiquitous bacterial strains to different compounds. | Rott et al., 1979 |
| Aerobic soil,-mixed microflora |  | tetrachlorophenols, trichlorophenols dechlorophenols  pentachloroanisole |  | Murthy et al., 1979 |
| Soil, mixed mircroflora |  | tetrachlorophenols  pentachloroanisoles,  tetrachloroanisoles |  | Haimi et al., 1993 |
| Aerobic Aquatic Biotransformation |  | tetrachlorophenols  trichlorophenols  dichlorophenols  bound residues |  | US EPA, 2008 |
| **Anaerobic conditions** | | | | |
| Flooded sandy loam soil | 1-2 months | tetrachlorophenols  Trichlorophenols  2-chloro-hydroquinone and mucochloric acid  CO2 |  | US EPA, 2008 |
| Coupled sludge granules under various dissolved oxygen conditions |  | reductive dechlorination was the primary pathway: Trichlorophenol, dichlorophenol, monochlorophenol | Oxygen condition had a significant effect on metabolism. A greater degree of PCP mineralisation was achieved under oxygen-limited conditions. | Chen et al., 2010 |
| *Enterobacter cloacae* |  | Reductive dechlorination pathway produced dechlorinated byproducts, including tetrachlorophenol |  | Nguyen et al., 2003 |
| Anaerobic sludge  (PCP in acetone and PCP in soil) | Nearly 100% (acetone) and 97.3% (soil) efficiencies | Reductive dechlorination:  Tetrachlorophenol, trichlorophenol (, dichlorophenol  3-monochlorophenol |  | Chen et al., 2003 |
| Loamy clay soil | Degraded PCP without complete mineralisation | 2, 4, 5-trichlorophenol | -Examining redox conditions on behaviour of PCP  -reducing conditions increased the dechlorination | Charnay et al., 2000 |
| PCP and 2,3,4,6-tetrachlorophenol contaminated soil placed under varying conditions (anaerobic) | Degradation occured | Reductive dechlorination:  3-chlorophenol | No anaerobic metabolites (lesser chlorinated phenols) were detected. | Frisbie et al., 1997 |
|  | PCP degraded within a few weeks | Tetrachlorophenols, trichlorophenols, dichlorophenols and 3-chlorophenol |  | Ide et al. 1972 |
| Flooded soil | Rapid degradation of PCP | Tetrachlorophenols, trichlorophenols,  dichlrophenols  pentachloroanisole  Others (trace amounts) |  | Kuwatsuka and Igarashi 1975 |
| Anaerobic aquatic sediment |  | Tetrachlorophenols  Trichlorophenols  Bound residues |  | US EPA, 2008 |

Table 5‑6: Summary of Studies with Information on the Formation and Transformation Rates of PCA observed under laboratory conditions with natural or mixed microflora

| **Study Description** | **Test Substance** | **PCP to PCA** | **Half-Life or description of persistence (trend)\*** | **Successor transformation products** | **Remark** | **References** |
| --- | --- | --- | --- | --- | --- | --- |
| **Aerobic Soil** | | | | | | |
| Laboratory  Aerobic soil (30 days) | PCP | Small amounts | Aerobic PCP transformation initially produced small amounts of PCA. PCA was detected in 7 of the 8 soils tested.  **>75% of both PCP and PCA disappeared in 30 days in five soils.** | Small amounts of PCA.  Authors indicated that PCA was either mineralised to CO2 or bound to soil. | Closed system. However, headspace gases were only analysed for CH4, CO2 and O2. No evidence of loss of PCP or PCA in 2 of the soils where PCA was produced. | D’Angelo and Reddy 2000 |
| Moist aerobic soils (24 d) | PCP | 7.5% | None reported | The principal reaction involved reductive dehalogenation (production of progressively simpler chlorophenols, TeCP, TCP and DCP).  The degradation of PCP and PCA are reductive in anaerobic soils, and oxidative in aerobic soils.  Higher levels of bound residues were observed in aerobic soils than anaerobic. | Limited amount of study details were reported.  Extraction efficiencies were extremely poor for the aerobic soils treated with PCP (14.7%) and a significant amount of radioactivity was lost. Organic volatile compounds and CO2 were not collected. | Murthy et al. 1979 |
| Laboratory aerobic soil |  |  | **5.6% of PCA was converted to PCP in 24 days.** |  | Based on further investigation by Murthy et al., 1979 that was not published but is referred to by Kaufman 1978.  No study details provided | Reported in Kaufman 1978 and Murthy et al. 1979 |
| Field Study  soil | TCA and PCA |  | **Half-life of approximately 5 weeks in both soil and in earthworms.** | Evidence of demethylation to tetrachlorophenol and pentachlorophenol. Rate of disappearance was also attributed to degradation, metabolised to other compounds not measured or were in non-extractable form. | Respiration (CO2 evolution) was measured weekly.  The rate of metabolism and/or degradation of chloroanisoles was high in the aerobic and humus –rich soil (and earthworms) used in our studies. This was corroborated by high respiration activity found with high concentrations of chloroanisoles. | Haimi et al. 1993 |
| Field study 10 upland soils | PCP |  | Rapid degradation of PCP.  PCA reached a maximum of 1% of the applied PCP at day and was 0.5% by day 35.  **Observed PCA half-life of approximately 30 days in upland (aerobic) conditions.** | Transformation products included TeCP (max. 4%), TCP (max 2%) and PCA (max 1%). Others were detected in trace amounts. | Test system was covered with aluminum foil.  The half-lives for PCA were not reported by the study authors, but can be read from the graphs provided. | Kuwatsuka and Igarashi 1975 |
| Soil mixed with PCP contaminate sawdust | PCP | 3.6-7.4  μg/kg |  |  | Degradation of PCP to PCA was observed in the soil. The initial PCP concentration was 91.5 mg/kg. The amount at study termination was <10 µg/kg (extremely low). | Mardonesa et al., 2009 |
| Soil-plant system | PCP |  | PCP was non-persistent in both outdoor and laboratory test systems |  | All volatile products were captured/measured in the laboratory tests. The only volatile substance found was CO2. | Klowskowski et al., 1981 |
| **Anaerobic Soil** | | | | | | |
| Laboratory  Anaerobic soil | PCP and PCA |  | N/A | Trichlorophenol, tetrachlorophenol, with lesser formation of pentachloroanisole (5.1%)  The principal degradation pathway of PCP in anaerobic soil would seem to be by progressive dehydrodehalogentation to tetra- and tri-chlorophenols, with lesser formation of pentachloroanisole.  Conversion of PCA to PCP. | Volatile organic compounds were trapped for the anaerobic portion of the study. Only 0.4-0.5% of the radioactivity was allotted to volatile organic compounds. | Murthy et al. 1979 |
| Anaerobic soil | PCP and PCA | **5.3%** | **42% of PCA was degraded to PCP in 24 days.** |  | Very good recovery of radioactivity. 98.8% of the radioactivity was recovered in the soil. Volatile organic compounds were also captured and only accounted for 0.4-0.5% of the total radioactivity. | Murthy et al. 1979 and reported in Kaufman 1978 |
| Field study (flooded) | PCP |  | Rapid degradation of PCP.  **Observed half-lives for PCA were approximately 25 days in flooded conditions.** | Transformation products included TeCP (max. 4%), TCP (max 2%) and PCA (max 2% at day 10 and was 1% by day 35). Others were detected in trace amounts. | The test system was covered with aluminum foil.  The half-lives for PCA were not reported by the study authors, but can be read from the graphs provided. | Kuwatsuka and Igarashi 1975 |
| Paddy Soil (rice fields) | PCP |  | PCP degraded within a few weeks of application.  No conclusions regarding PCA can be drawn from this study. | TeCP, TCP, DCP and 3-chlorophenol.  Reductive dechlorination was found to occur in paddy soil. | The analytical methods precluded the differentiation between the chlorophenols from the anisoles since the samples were treated with dimethyl sulphate (methylated). The authors allotted all of the residues chlorophenols (not chloroanisoles).  This study is included in Table because it was quoted previously as evidence of the production of PCA. | Ide et al. 1972 |
| Flooded rice soil | PCP |  | No half-lives for PCA can be drawn from this study.  Other lesser chlorinated anisoles were also found (tetra- and tri-chloroanisoles). | Most of the radioactive residues were associated with bound residues.  PCA (0.09%), tetrachloroanisoles, trichloroanisoles | The author states that not only PCP, but its lower chlorinated products may also be methylated in soil, although it must be conceded that tetra- and tri-chloroanisoles could originate as well from PCA by dechlorination. Since the toxic effects of phenols are mainly caused by the hydroxyl group, the methylation may be regarded as an inactivation process.  Limited uptake of residues from soil by rice plants. | Weiss et al. 1982 |
| **Aquatic Field Studies** | | | | | | |
| Contaminated Site-Spill |  |  | Low levels of PCA were observed. Partitioning of PCA to sediment. **Observed half-life of approximately 1.5 months in the sediment at site A.**  **Half-lives at site B, C and D could not be assessed due to continuous formation from PCP.** | Tetrachlorophenols  Pentachloroanisole  Tetrachloroanisoles (small amounts and difficult to analyse) | Any losses of PCA due to volatilisation were not captured.  Evidence of the accumulation of PCP and TeCP and PCA. The concentration in fish decreased as the concentration in the water decreased, but required 6-10 months to reach background concentrations. | Pierce and Victor, 1977 |

\*For those studies conducted where PCP and PCA was tracked, observed half-lives for PCA should be considered conservative, but cannot be considered a true half-life since formation and degradation would be occurring simultaneously. In the cases where formation and degradation is occurring simultaneously, the true half-lives are expected to be shorter.

Table 5‑7: Summary of Formation and Biotransformation of PCA in Studies conducted with Isolated Fungal and Bacterial Strains

| **Study Description and Test Species** | **PCP half-life** | **PCP to PCA** | **Half-Life or description of persistence (trend)\*** | **Successor transformation products** | **Remark** | **References** |
| --- | --- | --- | --- | --- | --- | --- |
| **Liquid** | | | | | | |
| *Trichoderma virgatum* |  | 10-20% | **Approximate (observed) DT50 for PCA is about 33 days.** | 80-90% of the original PCP was recovered neither as the free phenol for the PCA when incubated longer than 10 days. | The formation of PCA is only the first step in the metabolism or a parallel reaction to degradation.  PCA is less toxic to fungi.  Less soluble than PCP (10x).  PCA could not be detected by conventional reagents on TLC. | Cserjesi and Johnson 1972 |
| 9 New Zealand native white-rot fungi  *T. versicolor* |  | Trace |  |  | In liquid culture, very little to no PCA was captured in the volatile fraction of *T. versicolor* isolates, **whereas 75% of the volatile fraction of *P. chrysosporium* consisted of PCA.** | Walter et al. 2004 |
| *P.Chrysoporium* |  | 75%-volatile |  |  | The very large volatile fraction measured in the liquid culture experiments, may be an artefact of the experimental design. The PUF volatile trap in within the culture bottle. It is possible that mass-transfer between solid phase PCP, the solution phase, vapour phase, and the trapped volatiles has occurred.  Whatever the cause of the high volatile release in liquid culture it was not observed in the soil microcosms. It is probably that soil and added organic matter retain PCP sufficiently to significantly reduce volatilization. See Walter et al., 2005. | Walter et al. 2004 |
| O2 limited 12 days  *P. chrysosporium in liquid culture* |  | 82% (volatile) |  | If the fungus is oxygen-limited for lignin peroxidise production, it will convert most of the PCP to the volatile PCA, the only volatile compound detected in the presence of *P. chrysosporium* in this experiment. When sufficient oxygen was available, the extent of mineralisation was much greater, up to 32%. | Higher mineralisation was observed when the volatile transformation products had a chance to equilibrate between the solution and the headspace. Immediate removal of the volatile transformation products reduced PCP mineralisation. Therefore, the PCA remaining in the solution and PCP will be mineralised and product more 14CO2 over time compared with the case when all PCA is depleted from the liquid and sorbed on the polyurethane volatile trap. | Badkoubi et al. 1996 |
| *Trichoderma harzuanum* | 90% in 3 days  100% in 9 days | 85% |  | PCA: terminal transformation product (this was not the terminal transformation product when tested in soil-See Rigot and Matsumura, 2002 below) | Stochiometric formation of PCA was accompantied by the disappearance of PCP | Rigot and Matsumura, 2002 |
| *7 strains of Phanerochaete spp.* |  |  | **PCA was mineralised by both *P.* sordida 12% after 30days was significantly greater than the other P. sordida and**  ***P. chrysosporium* strains tested.** |  | Up to 10% of the total radioactivity in the liquid cultures was captured as volatiles. Volatility was not reported for in the soil | Lamar et al. 1990 |
| Sludge  *Rhodococcus, Mycobacterium* |  | 50%  80% |  | All strains initiated degradation of the chlorophenols by para-hydroxylation, producing chlorinated-hydroquinones, which were then further degraded. Parallel to degradation, strains also O-methylated nearly all chlorinated phenols, guaiacols, syringols and hydroquinones. O-methylation was a slow reaction compared with degradation. The preferred substrates of the O-methylting enzymes were those with the hydroxyl group flanked by two chlorine substituents. | degradation inhibitor was added that favoured the methylation of PCP | Haggblom et al. 1988 |
| **Soil** | | | | | | |
| Field Study  *T. versicolor* in soil/PCP |  |  | Field-Scale bioremediation. |  | PCA is not likely an intermediate transformation product of *T. versicolor* | Walter et al. 2005 |
| *T. versicolor* |  | <0.1% | Soil | Only trace amounts of anisoles such as PCA and TeCA were formed. |  | Tuomela et al., 1999 |
| Non-sterile soil (18 days)  *P. chyrsosporium* | 100% by 18 days (100 ppm)  <100% by 18 days (800 ppm) | 40% (100 ppm) | Beyond 18 days, there was still a linear rate of mineralisation, suggesting that PCA was being mineralised during this time.  800 ppm: both PCP and PCA were detected at 18 d. The rate of mineralisation of PCA increased with increasing concentration (not linear). | PCA (as an intermediate), mineralization. | *P. chyrsosporium* is able to mineralise high concentration of PCP and PCA.  Both PCP and PCA are readily mineralised in soil.  **Essentially no radioactivity (<0.05%) was found in either the volatile organic trap suggesting that PCP, PCA, or other intermediates were not volatile.** | Chung and Aust 1995 |
| *L. edodes* (Shitake mushroom)/PCP Sterilised and non-sterilised soil |  |  | **After 10 weeks:**  **Monocultures of *L. edodes* had eliminated both PCP and the chloroanisoles (including PCA). (sterilised soil)**  **In the mixed culture, PCA, was still detected in soils with mixed microflora after 10 weeks. (non-sterilised soil)** | PCA, TeCA, TeCP were detected, but concentrations were not reported. | PCA was a major transformation product | Okeke et al. 1997 |
| *L. edodes* (Shitake mushroom)/PCP |  |  | PCA was detected as a transformation product of PCP. | PCA was detected but concentrations were not reported. |  | Okeke et al. 1993 |
| *P. chrysosporium* and *L. edodes /*PCP |  |  |  | PCA, TeCA, TeCP were detected but concentrations were not reported. |  | Okeke et al. 1994 |
| Gardone soil (sandy, mixed, frigit Aridic haploxerol  *P. sordida* |  | 62% |  |  |  | Pfender et al., 1997 |
| Contaminate soil (90 days)  *36 fungal strains* |  | ≤ 2.73% | PCP: 58-78% degraded | Chloride ion production  51.7% *(P. cinerea)*  51.3% *(P. perfecra)* | Collected from an area in Sao Vicente, with high concentration of PCP. PCA observed from *P. cinerea, P. castanella and T. villosa* | Machado et al., 2005 |
| Field soil contaminate with PCP (aged PCP residue)  *Trametes versicolr HR 131 Trametes sp. HR577* |  | <10% | PCP: 37-70 degraded |  |  | Ford et al., 2007 |
| Soil slurry (soil collected from a forest site)  *Bjerkandera adusta Anthracophyllum doscolor* |  | ? | ? | 2,5-dichloro-1,4-dimethoxybenzene; 2-chloro-1,4-dimethoxybenzene; 3,4-dimethoxybenzaldehyde |  | Rubilar et al., 2007 |
| *7 strains of Phanerochaete spp.*  *P.chyrosoporium*  *P. sordida* |  | 64%  71% | PCA accumulated initially and was degraded during a second stage.  PCA was reduced by 18% in 55 days  PCA was reduce4d by 23% in 43 days | 64% (9 days) and 71% (21 days) were maxiumum formation values as the authors indicated that PCA was mineralised. | PCA production observed in 2 species  PCA appears to be slightly more persistent in this study.  Up to 13.8% of the total radioactivity in the liquid cultures was captured as volatiles. Volatility was not reported for in the soil | Lamar et al. 1990 |
| *Trichoderma harzuanum* |  | 13.5% | PCP: 0.5% PCP remaining after 14 days |  | The authors indicated that PCA was not the terminal metabolite in the case of soil corn rhizosphere unlike in the case of the liquid culture. In soil, PCA was either a metabolic intermediate or a temporary bypass reservoir for the purpose of rapidly eliminating PCP. | Rigot and Matsumura, 2002 |
| *7 strains of Phanerochaete spp.*  *P. chrysorhiza, P. laevis, P. suguinea, P.filamentosa, P. sordida, Inonotus circinatus, P. chrysosporium* |  |  | PCA accumulated initially and was degraded during a second stage beginning after 9 days of incubation.  PCA was reduced by 18% in soil inoculated with P.chyrosoporium and by 23% in soil inoculated with P. sordida over 56 days.  PCA was mineralised by both P. sordida and P. chrysosporium in liquid culture. |  | *Phanerochaete spp.* were sensitive to PCP. Growth was prevented at 5 ppm. However, P. sordida and P. chrysosporium were able to tolerate higher concentrations of PCP (25 ppm), albeit at greatly decreased mycelial extensions rates.  PCA appears to be slightly more persistent in this study.  Up to 13.8% of the total radioactivity in the liquid cultures was captured as volatiles. | Lamar et al. 1990 |
| In field study  *P. chrysosporium and P. Sordida* | 88%-91% decrease of PCP in 6.5 weeks. | 8-13% | PCA rapidly increased during the first 15 days after inoculation. After day 15, the amount of PCA in the soil inoculated with P. chrysosporium did not change significantly.  In the soil incubated with P. sordida, PCA was 8% at day 22. | PCP and PCA were mineralised by P. Chrysosporium and P. sordida. | PCP was present in the soils at concentrations between 100-400 ppm over the course of the 45 day study. | Lamar and Dietrich 1990 |
| Microbial Degradation  *Various species* |  |  | PCA was produced in very small yields in 5 of the 10 strains.  <0.005% to 0.02%. | Tetrachloroanisoles were formed by two pathways (dechlorination of pentachloroanisole or by methylation of tetrachlorophenoles) |  | Rott et al. 1979 |
| Microbial degradation  *Mycobacterium sp.*  In a mineral salt water medium |  |  |  | Mycobacterium sp. metabolized  PCP mostly through the methylation and at the same time part of PCP was hydroxylated  at ortho and para positions to the hydroxyl group followed by successive methylation. | Maximum rate of the methylation was observed between pH 6.5 and 7.0. The hydroxylation,  however, was dominant below pH 6.0. The addition of nutrients into the incubation  medium resulted in enhancement of the methylation and exclusive formation of PCA as  a sole metabolite. Toxicity of PCA for Mycobacterium sp. and for germinating rice seeds  was significantly low compared with PCP. | Suzuki 1983b |

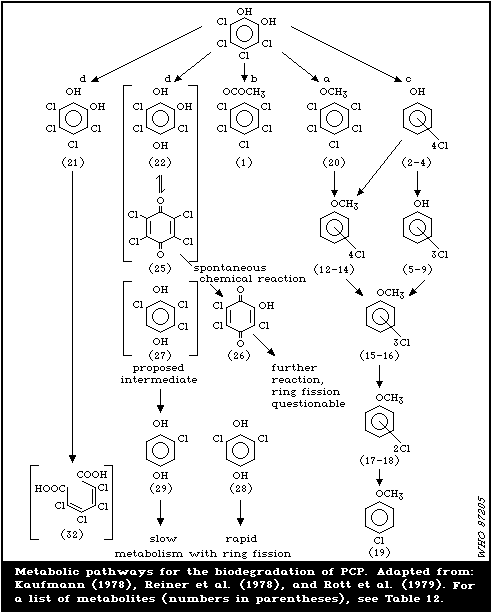


Figure 5‑6. Biotransformation pathways of PCP (EHC 71, 1997)

Table 5‑8: Environmental Transformation Products of PCP as reported in EHC 71(1987) with corrections from Annex E information submitted by Canada, 2013).

| **Number** | **Chemical Name** | **Reference** |
| --- | --- | --- |
| 1 | Pentachlorophenol acetate | Rott et al. 1979 |
| 2 | 2,3,4,5-tetrachlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975; Murthy et al. 1979 |
| 3 | 2,3,5,6-tetrachlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975; Murthy et al. 1979 |
| 4 | 2,3,4,6-tetrachlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975 |
| 5 | 2,4,5-trichlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975 |
| 6 | 2,3,6-trichlorophenol | Kuwatsuka and Igarashi 1975; Murthy et al. 1979 |
| 7 | 2,3,4-trichlorophenol | Kuwatsuka and Igarashi 1975 |
| 8 | 2,3,5-trichlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975 |
| 9 | 2,4,6-trichlorophenol | Kuwatsuka and Igarashi 1975 |
| 10 | 3,4-dichlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975 |
| 11 | 3,5-dichlorophenol | Ide et al. 1972; Kuwatsuka and Igarashi 1975 |
| 12 | 2,3,4,5-tetrachloroanisole (acetate) | Rott et al. 1979b |
| 13 | 2,3,5,6-tetrachloroanisole (acetate) | Rott et al. 1979b |
| 14 | 2,3,4,6-tetrachloroanisole (acetate) | Engel et al. 1966; Rott et al. 1979b; Haimi et al. 1993 |
| 20 | Pentachloroanisole | Cserjesi & Johnson 1972; Kuwatsaka & Igarashi 1975; Murthy et al. 1979; Rott et al. 1979 |
| 21 | Tetrachlorocatechol (diacetate) | Suzuki 1977; Rott et al. 1979b |
| 22 | Tetrachlorohydroquinone | Suzuki 1977 |
| 23 | Tetrachlororesorcinol (diacetate) | Rott et al. 1979b |
| 24 | Tetrachlorohydroquinone dimethylether (diacetate) | Rott et al. 1979 |
| 25 | Trichlorobenzoquinone | Reiner et al. 1978 |
| 26 | Trichlorohydroxybenzoquinone | Reiner et al. 1978 |
| 27 | 2,3,6-trichlorohydroquinone | Reiner et al. 1978 |
| 28 | 2,6-dichlorohydroquinone | Reiner et al. 1978 |
| 29 | 2-chlorohydroquinone | Reiner et al. 1978 |
| 30 | CO2 | Chu and Kirsch 1972; Kirsch and Etzel 1973; Suzuki 1977 |
| 31 | Cl- | Watanabe 1973; Suzuki 1977 |
| 32 | Tetrachloromuconic acid | Lyr 1962 |
| 33 | beta-hydroxytrichloromuconic acid | Lyr 1962 |

### Aquatic Field Study

1. Pierce and Victor (1978) studied the fate of PCP and its transformation products in an aquatic system after an accidental release in 1974 of wood-treating wastes containing PCP in fuel oil and a second spill in 1976. The authors reported that PCP and its transformation products, TeCP and PCA were measured in water and in fish for over six months following the spill. Fish were observed to accumulate PCP and PCP transformation products (PCA and TeCP) rapidly from the water. The concentrations in fish decreased as the concentration in the water decreased, but required six to ten months to reach background levels. Interpretation of the data is complicated by the chronic influx of PCP from the contaminated watershed areas and the possible periodic release of small amount of PCP-containing waste from the industrial holding pond.
2. The average values as reported by Pierce and Victor were plotted and are presented in Figures 2.3-1 to 2.3-10.
3. At all sites, PCP underwent simultaneous degradation and partitioning from the water column to the sediment as is shown by the simultaneous decrease in the water concentrations of PCP and the increase in the sediment concentrations of PCP and its transformation products TeCP and PCA.
4. Concentrations of PCA in the water column remained low during the observation period. Initial low concentrations of PCA increased gradually in the sediment over the four-month period. At these sites, significant amounts of PCP were still available as a source of PCA. The continuous formation of PCA from PCP precluded an assessment of the degradation/persistence of PCA. However, due to the presence of TeCA (tetrachloroanisoles) and tetrachlorophenol, as reported by the authors, it is possible that some transformation of PCA was occurring.
5. Decreased concentrations of PCP, PCA and TeCP were observed at Site A, however, this site was located in a stream and there is too much uncertainty regarding the fate of the sediment to make any conclusion regarding dissipation half-lives (e.g., sediments may have been washed downstream or buried over time).
6. The low solubility of PCA in water (<1 mg/kg) and the high estimated KOC value (2474 and 13800 L/kg) indicate that PCA is likely to partition to sediment in aquatic systems. Given the absence of PCA in the technical product, it is likely that in this aquatic system, PCA was formed through the biomethylation of PCP in the sediment. Once in the sediment, it is likely that simultaneous formation of PCA from PCP and demethylation of PCA back to PCP was occurring as predicted by the laboratory transformation studies. Since significant amounts of PCP were available as a source of PCA over the monitoring period, the continuous formation of PCA from PCP precluded a half-life calculation of PCA. However, the increase in concentration of the tetrachlorophenols indicates that PCP was also undergoing dechlorination to lower chlorinated phenols predicted by the laboratory studies to be the main degradation pathway of PCP.
7. As this was a field study, it was not possible to capture all potential losses including the loss due to volatilisation. The physical and chemical properties of PCA indicate that volatilisation is a potential source of loss from water. The fate dynamics between PCP and PCA in sediment, water and air in the aquatic environment preclude assessing a true degradation half-life from field or monitoring data. However, qualitative information such as the partitioning behaviour of PCA in aquatic systems (i.e., movement from water to sediment) and the degradation of PCP to lower chlorinated phenols is consistent with the biotransformation and mobility observed in the laboratory studies.

















## Metabolism Information from HCB, lindane and quintozene

1. PCP is a major metabolism product of HCB in a variety of different animals including fungi, caterpillars, fish, rats, birds, monkeys and humans (Sanborn et al. 1977; Frankovic et al. 1995; Kasokat et al. 1989; Metcalf et al. 1973; Mehendale et al. 1975; Koss et al. 1976; Szokolay et al. 1980; Mehendale and Matthews 1973 in Courtney 1979; Lui and Sweeny 1975 in Courtney 1979; Renner 1981; Gomez-Catalane et al. 1987; To-Figueras et al. 1997; Engst et al. 1975; van Ommen et al. 1985; van Ommen et al. 1986; Muller et al. 1978; Deberts et al. 1981).
2. The pathway of metabolism of HCB in mammals was reviewed by Debets and Strik (1979) and by Renner (1988) as reported in Environmental Health Criteria (EHC) 195, IPCS (1997). HCB is metabolised into less chlorinated benzenes, chlorinated phenols, and other minor metabolites via three distinct pathways: i) oxidation giving rise to phenolic metabolites including PCP, tetrachlorohydroquinone and tetrachlorobenzoquinone; ii) glutathione conjugation leading to pentachlorothiophenol, pentachlorothioanixoles and other sulphur-containing metabolites; and iii) a minor pathway that yields lower chlorinated benzenes.
3. PCP is also a metabolic transformation product of PCNB (quintozene) in various mammals including rats and monkeys (EHC 41 1980; Muller et al. 1978; Kogel et al. 1979, Renner 1981). In the environment, the U.S. EPA (2008b) has also reported that PCP was detected as a metabolite of PCNB in several registrant-submitted environmental fate studies and in the published literature, Murthy and Kaufman (1978) reported PCP in anaerobic soils; Begum et al. (1979) reported PCP in onion plants and Torres et al. (1996) reported PCA in formation by a soil micromycetes.
4. There is also literature information on the formation of PCP from lindane. Kujawa et al. (1977) reports PCP as a transformation product of lindane in rats. In the summary report by Engst et al. (1979), the following references are cited as showing PCP formed as a transformation product of lindane: Balba and Saba (1974) reports PCP produced from lindane in plants, Engst et al., 1978a and Gopalaswamy and Aryar (1976) in mammals.
5. The EHC124 (1991) review of lindane, also cites several studies identifying chlorophenols, including PCP, as transformation products of lindane in animals (Grover & Sims 1965; Chadwick & Freal 1972a,b; Freal & Chadwick 1973; Kurihara & Nakajima 1974; Chadwick et al. 1975; Engst et al. 1976; Kujawa et al. 1977; Stein et al. 1977; Tanaka et al. 1977; Engst et al. 1978b; Tanaka et al. 1979; Aiyar 1980; Fitzloff et al. 1982).

## Mobility

### Adsorption / desorption

1. Measured KOC values as reported in UNEP/POPS/PORC.7/INF/5 and in the Annex E Information submitted by the U.S., range from 706 to 4000; indicating that PCP is likely to have slight to moderate mobility in soil as per the McCall et al. (1981) classification scheme.
2. The following additional analysis of mobility is from the Annex E Information submitted by the U.S., 2013.
3. The mobility of PCP in the environment is dependent, among other things, on the pH of the system. PCP is a weakly acid compound (pKa = 4.74) in aqueous solution that will exist in increased proporionts in the ionised form with increasing pH. At pH 6.5 and higher, the majority of the PCP present in a system will occur as the phenolate anion. Because ionised PCP has substantially greater solubility in water than does unionised PCP, the aqueous solubility of PCP increases with increasing pH, indicating a greater mobility when the pH of water/soil is more alkaline. In addition, PCP will be less soluble in organic matter in soils of higher pH. Maximum adsorption has been reported at soil pH values of 4.6-5.1, with no adsorption above pH 6.8 (Choi and Aomine, 1974). As a result of the increased solubility in water and the decreased adsorption to the soil, the mobility of the phenolate anion in the environment at pHs of greater than 6 can be substantial in the absence of transformation. The results of studies in the literature also indicate that desorption of PCP is also greater at higher pHs (Banerji and Wie 1993; You and Liu, 1996); this implies that previously adsorbed PCP transported on particles to environments of higher pH may be desorbed at new sites.
4. Although the mobility of PCP in soils is strongly affected by the soil pH, the soil organic matter content also affects the adsorption of the compound. The concentration of PCP in the soil or in groundwater also has an effect on its mobility; adsorption of PCP to aquifer or soil may decrease as PCP concentrations increase. Also the presence of alchohols or petroleum hydrocarbons in the soil may also increase the mobility of PCP.
5. The estimated soil adsorption coefficient (KOCWIN v2.0 in U.S. EPA 2001) is 2474 L/kg (MCI method) and 13800 L/kg (Kow method) (Appendix II). The Koc estimate of 2474 and 13800 indicate that PCA is likely to be immobile or have slight mobility when in soils as per the McCall et al. (1981) classification scheme. The high KOC also indicate that in aquatic systems, PCA is likely to partition to sediment.

### Aqueous Availability from Treated Wood

1. The following additional analysis of mobility is from the Annex E Information Submitted by the U.S., 2013 [U.S. EPA 2008].
2. Wood treated with PCP may release the compound through volatilisation or leaching. Additionally, PCP may be phototransformed on the wood surface, making transformation products available for leaching. All three processes are affected by the solvent systems/carriers used in the application of the compound. The leaching of PCP out of utility poles may also partially depend on the method of application (pressure or thermal treatment). PCP may be leached from the poles as the compound moves with either aqueous solution (as from rain) or with the solvent down the pole, either at the surface or within the pole. Based on experimental data (Weinberg Group 1997), it was determined that the main mechanism for the leaching of PCP and its mircrocontaminants is the downward migration of the oil carrier along the vertical axis of the pole, designated as “Gravitational Induced Downward Migration of Oil” (GIDMO). Leaching of PCP in aqueous solution from rainwater is not considered to be as important as GIDMO, as the replenishment rate at pole surfaces is a limiting factor with respect to the availability of the compound for leaching. Thus, contamination of subsurface soil found in the vicinity of utility poles may result from the downward movement of PCP within the pole, with subsequent leaching form the bottom part of the pole to the soil surface or to the subsoil near the underground portion of the pole, as well as from the downward movement of PCP from the surface soils to the subsoil. When leaching of PCP from treated poles occurs, the simultaneous leaching of the carrier solvents may affect the mobility of the compound in the soil.
3. Literature information indicates that PCP applied in soil is rapidly transported from the upper portion of the poles to the underground portion during the first few years of use (Cooper, 1991). Laboratory studies (references not specified in U.S. EPA 2008) have also demonstrated a relatively high initial rate of depletion from the wood surface, which became relatively constant with time. Rainwater from treated cedar (oil carrier) maintained a relatively constant PCP concentration of 0.3-0.7 µg/ml over a one-year period (Cserji, 1976). Leaching (via rainfall) from a bundle of 15 utility poles of douglas fir (16- to 27- in diameters) held in storage areas, indicated that the release of PCP was relatively constant throughout a four-month study period. PCP concentrations of 1.57-2.85 mg/L of rainfall were observed (Whiticar, 1994). In a leaching study utilizing class 5 red pine poles which were pressure-treated with PCP, sealed to simulate in-service poles, and subjected to simulated acid rainfall (pH 4.2) for 10 days at 20ºC, the mean PCP loss per leaching event was 23.3 mg/pole section with a total mean loss of 232 mg (range of 159.7-330 mg); the maximum concentration of PCP was 4.4 mg/L (Buchanana, 1991).
4. In a study conducted by the Electric Power Institute (EPRl 1995a), soils adjacent to in-service utility poles (mainly southern pine) in NY were analyzed for PCP and other compounds as a means of examining the release of PCP from treated wood in the environment. The soils were mainly classified as “'lean clay" and "clayey sand" (43% each) and the PCP carrier on 28 of the 31 poles was petroleum. Although soil charactcristics and. levels of PCP contamination (site maximum of 0.35-1900 ppm, with the majority at <100 ppm) varied throughout the sites, a general trend was observed with respect to the changes in concentration of PCP with distance from the poles. Pentachlorophenol was observed to decrease as the distance from the pole increased; a decrease as great as two orders of magnitude, with an average of one order of magnitude, was observed between 3 and 8 inches from the poles. While higher concentrations of PCP were generally observed in survace soils, a general trend with respect to depth was not observed across all soils, nor was there a general trend with repected to the location of the maxium concentration of PCP. Because PCP could reach the subsurface soil through other means than directly leaching fom the soil suface down through the soil, it was concluded that the concentration of PCP in the surface soils would not accurately predict subsurface concentrations. Other means by which PCP could reach subsurface soil include leaching from the below-ground surface of the poles, particularly whena fluctuating water table is present near the bottom of the poles. The soil concentrations of lower-chlorinated phenols were generally lower than the levels of PCP detected. Neither concentrations of PCP in the poles, PCP application rates, nor the age of the poles correlated well with the concentration of PCP detected in the soils near the poles.
5. In a second PRI study (EPRI, 1995b), soils adjacent to in-service utility poles in 16 states throughout the U.S. were analysed for PCP. Results of the study were similar to those of the EPRI study conducted in the NY state: 1) the concentration of PCP decreased as the distance from the pole increased; 2) higher concentrations of PCP were generally observed in surface soils, but a general trend with repect to depth was not observe dacross all soils; and 3) the concentration of PCP in the soils near the poles did not correlate well with pole parameters.
6. Because of the demonstrated tendency for PCP to adsorb to soils and the moderately rapid degradation of the compound in the environment, it is unlikely that contamination of groundwater will result from the use of utility poles. Situations where ground water could potentially be contaminated include: i) when the bottom of the pole is directly in contact with the water table (or with a fluctuating water table) or ii) where leaching occurs from multiple poles in a wood storage or treatment area.

## Potential for Long Range Transport

1. PCP is a relatively volatile compound, while its sodium salt is non-volatile. Based on PCP’s low Henry’s law constant, volatilisation from aqueous systems is not expected to be a significant mode of transport in the environment. 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 (half-lives 12-44 h Slooff et al. (1991)). Atmospheric PCP associated with particulate matter or moisture will be subject to wet and/or dry deposition.
2. Although modelling calculations predict PCP transport over considerable distances, PCP has been reported primarily in air monitoring programs close to potential sources (urban areas (as in Zheng et al 2011), proximity to historical use sites) and has rarely been reporteded in remote areas (see Section 5.8.2).
3. The Henry’s law constant for PCA is estimated as 1.94x 10-3 atm-m3/mole, using a group estimation method and 7.12x 10-5 atm-m3/mole, using a bond estimation method (HENRYWIN v.3.20 in U.S. EPA 2011). This value indicates that that PCA has the potential to volatilize from water or moist soil. Based on this value for Henry's law constant, 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. 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 to be 6.9 days (Estimated by Group SAR Method in U.S. EPA 2011).
4. PCA can be photo-oxidised in the atmosphere through reactions with hydroxyl (OH) radicals. The calculated half-life for PCA based on this reaction is 9.8 days, with an atmospheric (OH) concentration of 1.56 x 106 OH/cm3 (AopWin v1.96 in U.S. EPA, 2011). No experimental data are available on atmospheric degradation.
5. Volatilisation of PCA has also been observed in several laboratory studies using liquid medium (Badkoubi et al. 1996, Walter et al. 2004, Lamar et al. 1990). A QSAR estimate of the phototransformation half-life of PCA in air is estimated to be 9.8 days (U.S. EPA 2011). No other information on half-lives of PCA in air was available. Based on the QSAR estimate for the half-life in air and the detection in air and snow in the arctic, there is evidence indicating the PCA is persistent in air and can be transported to remote locations.
6. Monitoring data collected show the presence of both PCP and PCA in air. PCA is generally found at higher concentrations and more frequently than PCP. Swedish air monitoring information reported that PCA was detected at higher levels than PCP. PCA is monitored and measured in air in remote areas such as the Canadian High Arctic station of Alert, Nunavut, Canada. The research station at Alert is part of Canada’s National Implementation Plan for Arctic Monitoring and Assessment Programme (Hayley Hung, 2013). Monitoring information for PCP and PCA is addressed in Section 5.8.2 and Section 5.9.1. .
7. Based on the QSAR estimate for the half-life in air and the detection in air and snow in the arctic, there is evidence indicating that PCA is persistent in air and can be transported to remote locations. Although the half-life of PCP in air is below the criteria threshold for long range transport, there is information indicating the potential long range of PCP through the sorption to particulate matter.

## Bioaccumulation

### Aquatic Organisms

**PCP**

1. In the open literature, a range for the log Kow for PCP varies between 1.3 and 5.86. However, the recommended values are 5.12 and 5.18. The log KOW is strongly pH dependent . The log Kow may not be a good indicator of bioconcentration as PCP is subject to metabolic transformation (UNEP/POPS/POPRC.7/INF/5).
2. In a radiolabelled guideline bioconcentration study conducted in bluegill sunfish, BCF values of 190-790 were reported (U.S. EPA 2008 submitted Annex E information as: UNEP-POPS-POPRC8CO-SUBM-PCP-USA\_8-20130110.En[1].pdf). All residues were identified as PCP, with the exception of 6-13% which were presented as glucoronide conjugates of PCP in the nonedible tissues. Depuration was rapid with over 97% of the residues eliminated from all tissues by day 14.
3. In addition, measured BCF values for PCP from the published literature for crustaceans, bivalves, aquatic and terrestrial worms and fish are reported in UNEP/POPS/POPRC.7/INF/5.

Table 5‑9: Summary of laboratory BCF and BAFs for PCP in Biota as reported in UNEP/POPS/POPER.7/INF/5 and Annex E information submitted by the U.S.A. 2013.

| **Organism** | **BCF/BAF** |
| --- | --- |
| *Crustacea* | 19-640 |
| bivalves | 0.9-461 |
| worms | 71-830 |
| fish | 5-4900 |

1. Measured BCF data range from 19-640 in crustacean, 0.9-461 in bivalves, 71-3 830 in aquatic and terrestrial worms and 5-4 900 in fish. There is only one study with BCF>5 000 (Gossiaux et al. 1996 in UNEP/POPS/POPRC.7/INF/5). However, only the total radioactivity was determined and parent PCP was not measured, therefore, the measurement included all potential transformation products of PCP as well as PCP. Considering the factors stated above (biotransformation, depuration), PCP does not meet the indicative value of BCF> 5 000.
2. Letcher et al. (2009) reports BMFs>1 for PCP in polar bears, however, the analytical method contained a methylation step with diazomethane that would have made it impossible to differentiate between PCP and PCA. Also, given that PCP is a metabolic product of HCB, a known contaminant of Arctic biota, in various organisms including mammals (See Section 5.4) and the potential that they were measuring PCP and PCA together, this BMF estimate may not be an appropriate indicator of the potential biomagnification of PCP.
3. Hoekstra et al. (2003) noted that PCP was the most abundant halogenated phenolic compound found in bowhead plasma. It was suggested that the PCP originated from either HCB or PCA.
4. Although PCP is detected frequently in Arctic biota, biomagnification of PCP in terrestrial or aquatic food chains has not been observed.

**PCA**

**Laboratory Studies:**

1. The log KOW of PCA of 5.45 indicates the potential for PCA to bioaccumulate.
2. In a bioaccumulation study conducted by Oliver and Niimi (1985), rainbow trout (Oncorhynchus mykiss) were exposed to PCA (and 17 other compounds simultaneously) at average water concentrations of 0.9 ± 0.3 and 10 ± 6.2 ng/L for 96 days. BCFs in the low concentration tank appeared to reach a steady state on day 35. BCFs in the high concentration tank were more variable, but a steady state appeared to have been reached on day 50. Concentrations of PCA in the water were variable over time. No depuration phase was conducted. It should be noted that variation was 33% in the low concentration tank (0.9 ± 0.3 ng/L) and was ca. 66% in the high concentration tank (10 ± 6.2 ng/L). Also, given that there was exposure to multiple chemicals simultaneously, metabolic capacity of the test organisms may have been affected. Average BCFs were well above 5000 (15,000 ± 4,950 and 20,000 ± 13,200 in the low and high concentration tanks, respectively), however, the results should be considered in conjunction with other information.
3. A 7-d static bioaccumulation study conducted on guppies (Poecilia reticulate) by Opperhuizen and Voors (1987) was not considered appropriate for the determination of bioaccumulation factors for classification purposes because the test system was not at equilibrium and recovery rates were extremely low (22.5% for PCA) due to a continuous decrease in aqueous concentrations. However, additional information on the behaviour of PCA in fish can be obtained from the study. The authors concluded that chloroanisoles were eliminated rapidly from the fish. Half-lives for the tetra- and the pentachloroanisoles were between one and four days. Since a rapid decrease of the total amount of test compounds in the aquarium was found for all congeners, the calculation of bioconcentration factors was not possible. Only estimates of the concentration ratio between fish and water are made. Based on the observed high loss of the test compounds from the system and the formation of metabolites, the authors suggested that the total clearance is dominated by transformations of the chloroanisoles. Clearance rates of chlorinated anisoles in fish are much higher than expected from their estimated hydrophobicity. The authors speculated that this may be explained by metabolic hydrolysis of the ether bonds into corresponding hydroxyl groups. Due to these high elimination rates, bioconcentration factors are relatively low, compared to those of chlorobenzenes and other hydrophobic chemicals.
4. In a bioaccumulation study conducted Glickman et al. (1977), rainbow trout were exposed to 14C-PCA at concentrations of 0.024 mg/L. Fish were sampled at 1, 2, 4, 8 and 12 hours in the uptake study and, after transfer to clean water they were sampled at 0, 4, 8, 15, 24, 48, 72, 96, 120, 144 and seven days. Half-lives of PCA in tissues of rainbow trout were 6.3, 9.8, 23 and 6.3 days in blood, liver, fat and muscle, respectively. Corresponding extrapolated half-lives of PCP were 6.2, 9.8, 23 and 6.9 hours. PCP–exposed trout showed no methylation of PCP to PCA in any of the tissues studied. However, the bile of the PCA-exposed trout contained PCP glucuronide as well as PCA, indicating demethylation of PCA in vivo by the rainbow trout.

**Aquatic Field Bioaccumulation:**

1. Pierce and Victor (1978) studied the fate of PCP and its transformation products in an aquatic system after exposure from an accidental release of wood-treating wastes containing PCP in fuel oil in 1974 and a second spill in 1976. The authors reported that PCP and its transformation products, TeCP and PCA persisted in water and in fish for over six months following the spill. Interpretation of the data is complicated by the chronic influx of PCP from the contaminated watershed areas and the possible periodic release of small amount of PCP-containing waste from the industrial holding pond.
2. Fish were observed to accumulate PCP and PCP transformation products (PCA and TeCP) rapidly from the water. The concentrations in fish decreased as the concentration in the water decreased, but required six to ten months to reach background levels.
3. The U.S. Fish and Wildlife Service periodically determines concentrations of organochlorine chemicals in freshwater fish collected from a nationwide network of 112 stations as part of the National Contaminant Biomonitoring Program. Schmitt et al., 1990 analysed samples taken from 1970 up to 1985. PCA was detected in 1980 and 1984 in fish samples from 30% of the stations (Schmitt et al., 1990). The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 2009) detected PCA in both bottom feeding and predator fish, however, the detection frequency was lower in the predators.
4. A study from Greenland shows bioaccumulation of PCA in a range of species varying from aquatic invertebrates to fish, birds and mammals (Vorkamp et al, 2004). However, the concentration of PCA found in these different trophic levels showed no evidence of biomagnification. Vorkamp et al. (2004) noted the concentrations in top predatory marine mammals (harp seal, narwhal and beluga) do not exceed the concentrations in marine fish, contradicting the typical pattern of bioaccumulation in the food chain. Compared with the results for chlorobenzenes and other chlorinated pesticides the concentrations of PCA were considered to be low in biota.
5. Unpublished information (Muir 2013) on residues of PCA in biota from remote Canadian arctic areas are summarized in Table 5‑13. From 2000-2010, the range of concentrations in polar bears, ringed seal, 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-0.35 ng/g lipid and <LOD – 3.85 ng/g lipid, respectively. BAFs cannot be calculated from these data because sampling sites differ between and within species and there is a 10 year span of time when sampling occurred.
6. PCA is detected frequently in arctic biota at low concentrations. In aquatic food webs, concentrations in top predatory animals do not exceed concentrations in lower trophic organisms.

### Terrestrial organisms

**PCP**

1. Measured BCF values for PCP in terrestrial worms were 426-996 and are reported in UNEP/POPS/POPRC.7/INF/5.
2. Biomagnification of PCP in terrestrial food chains has not been observed.

**PCA**

**Laboratory:**

1. BAFs of PCA and TeCA in earthworms exposed to soil from a contaminated site were reported as 5-40 (Haimi et al. 1992, Haimi et al. 1993). In a laboratory experiment, the concentrations of 2,3,4,6-tetrachloroanisole and pentachloroanisole were high in earthworms one week after introduction. Concentrations in earthworms and soil decreased to a low level at a considerable rate (in approximately 5 weeks). Similar degradation rates were observed in the control soils without earthworms and the soils with earthworms. In the earthworms, the concentration of 2,3,4,6-TCA and PCA increased until week 15, after which they decreased.
2. Vodicnik et al. (1980) determined that following 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 the liver. Excretion of 14C was primarily through the urine. However, there was no evidence of parent PCA in either urine or feces. PCP was detected in the urine and feces at approximately 2 and 32% of applied radioactivity, respectively. The majority of the 14C was associated with the PCP conjugate. The authors concluded that PCA must be demethylated prior to conjugation and/or excretion.
3. Other references include Ikeda and Sapienza (1995) and Ikeda et al. (1994). These have not been reviewed, but are provided here as additional references.

The above information on bioaccumulation is summarised in

Table 5‑10.

Table 5‑10: Summary of Laboratory BCF and BAFs for PCA in Biota

| **Organism** | **Exposure Duration**  **(days)** | **Exposure Concentration (ng/L)** | **BCF1** | **Depuration** | **Comments** | **Reference** |
| --- | --- | --- | --- | --- | --- | --- |
| **Laboratory (fish)** | | | | | | |
| *Oncorhyn-chus mykiss* | 35 | 0.9 ± 0.3 | 16000 ± 3500 | Not reported. | Acceptable BCF study. | Oliver and Niimi 1985 |
| 50 | 0.9 ± 0.3 | 14000± 2900 |
| 75 | 0.9 ± 0.3 | 12000± 2400 |
| 96 | 0.9 ± 0.3 | 17000± 7100 |
| 35 | 10 ± 6.2 | 11000± 3100 |
| 50 | 10 ± 6.2 | 20000± 2500 |
| 75 | 10 ± 6.2 | 15000± 2600 |
| 96 | 10 ± 6.2 | 24000± 5400 |
| *Oncorhyn-chus mykiss* | N/A | N/A | N/A | T1/2: 6.3, 9.8, 23 and 6.3 days in blood, liver, fat and muscle, respectively. | Experimental.  Radiolabeled.  Demethylation of PCA to PCP. | Glickman et al. 1977 |
| *Poecilia reticulata*  (Guppy) | 7 | 40 | ~~-~~ | Half-life:1-4 days  Log Kd, oct: 5.45, log Kc: 3.96, K1 (mL/g\*d): 1710 K2(d-1): 0.32 | Not at equilibrium.  22.8% recovery. Continuous decrease in aqueous concentrations as contamination of the water was stopped before the fish were added. BCFs could not be calculated. | Opperhuizen and Voors 1987 |
| **Laboratory (mammals)** | | | | | | |
| Mice |  |  |  | Elimination half-lives of 5-10 hours in all tissues, except liver. The live half-life was 19.3 hours. | Excretion primarily through the urine as PCP-conjugate. PCA must be demethylated prior to excretion. Determined via 14C and no attempt to differentiate between PCA and transformation products | Vodicnik et al. 1980 |
| **Field** | | | | | | |
| Earthworms | 1-20 weeks | 0.06-1.0 µg/g dw  (1.24 – 20.8 µg/g OC) | 5-40 | Half-life of 5 weeks in soil and in earthworms. | BCFs estimated by IEP (2008).  Earthworm reproduction was not affected. | Haimi et al. 1992, Haimi et al. 1993 |

## Monitoring Information for PCP

1. There are several sources of PCP in the environment including the release of PCP when used in accordance with currently registered uses, contaminated sites from the historical use of PCP as an agricultural pesticide and from improper practices of wood treatment plants (e.g., spills from industrial holding ponds from wood treatment facilities prior to current guidelines). Releases may also occur through revolatilisation from adsorbed residues of PCP/PCA.
2. It should be noted that PCP is also a transformation product of other organochlorines such as HCB and, PCNB (quintozene) (Murthy and Kaufman, 1978 and U.S. EPA RED for PCNB, 2006) and lindane (Engst et al. 1979). These organochlorine substances are global pollutants and have been detected in remote locations in both abiotic media and biota. The presence of PCP in remote regions can be due to the transformation of HCB, PCNB or lindane, or a combination of these. Monitoring data should be interpreted with caution.
3. The following countries reported that they had no monitoring information or did not include monitoring information for PCP or PCA: Crotia, Mexico, Nigeria, and Thailand.

### Water

1. Borysiewicz (2008) compiled levels of PCP from various European sources. Concentrations of PCP in European river waters have declined sharply since early 1990 when marketing and use restrictions were first implemented (Euro Chlor 1999, in Borysiewicz 2008). Concentrations in rivers from the Netherlands, Germany and Belgium ranged from 0.01 to 0.17 μg/L from 1990 to 1997. The Seine River in France had an average concentration of 0.03 μg/L in 1995. Concentrations in the UK in 1990 to 1992 showed slightly higher concentrations, however, the median levels of PCP were below 1 μg/L; concentrations were higher in industrial areas (one site had a concentration of 40 μg/L. However, between 1994 and 1996 concentrations were considerably lower (0.15, 0.20, 0.02 μg/L in 1994, 1995, 1996, respectively) likely reflecting the restrictions in use.
2. Monitoring data showed that PCP concentrations generally decreased between 1988 and 1993 in the River Elbe. This was attributed to the cessation of PCP production in Germany in 1986 and use ban in 1989. However, an increase was observed in the Rhine River and its tributaries in 1990-1991 compared to 1980- 1989 (Borysiewicz 2008).
3. In the marine environment concentrations of PCP ranged from non-detect to 0.79 μg/L for the period 1983 to 1997 (average/median concentrations were below 1 μg/L) in the North Sea, coastal waters and estuaries of Germany, Netherlands and the UK (Euro Chlor in Borysiewicz 2008). In estuary waters, concentrations generally show a decreasing trend between 1983 and 1997 at all monitoring sites. Between 1983 and 1997 a “typical concentration” for coastal and marine water was estimated to be 0.07 μg/L.
4. Between 1994 and 1998 a median PCP concentration of 0.0706 μg/L (n=2,296 from 85 sample sites) was detected in EU Member States in the context of the EC Water Framework Directive.
5. Surface water used for drinking water in the U.S.A. contained a range of 0.04 to 1 μg PCP/L (mean 0.4052 ± 0.4355) (U.S. EPA 2001a, in Borysiewicz 2008). Concentrations of PCP in ground water ranged from 0.04 to 1.64 μg/L (mean 0.459 ± 0.444 μg/L).
6. Hoferkamp et al. (2010) does not report detections of pentachlorophenol (PCP) in water.
7. Inland surface waters and other surface waters in Estonia had annual average concentrations of PCP of 0.4 μg/L (the LOQ).
8. Zheng et al. (2011) in their summary of PCP studies found that the most recent studies (1991 to 1996) they reviewed for freshwater from various countries (Belgium, Germany, UK, Netherlands and France) had average concentrations ranging from 0.01 to 0.169 μg/L. Average concentrations in marine water between 1993 and 1997 ranged from 0.001 to 0.012 μg/L in samples from the Netherlands and UK. It is difficult to obtain any sort of spatial trends from the data in Zheng et al. (2011) because it is impossible to know if the samples were taken from the same areas over the sampling periods shown.
9. Slovakia has monitoring information in water, but has not submitted.
10. PMRA (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. Water was usually sampled as part of general, year round screening for a number of different items such as, nutrients, bacteria, metals, and organic contaminants. The aim was to determine the effects of agricultural and urban non-point source pollution on aquatic ecosystems. There was no information that indicated that the detections were associated with the registered uses of HDWPs.
11. Concentrations of PCP in the Niagara River and St. Lawrence River in Canada ranged from <0.2 to 21 ng/L ()
12. Data for PCP in water from the international convention for the Rhine provide annual data until 2011 with exemplary levels below 0.05 to 0.006 ng/L upstream and below 0.02 to 0.1 ng/L downstream from 2000 to 2011.

Table 5‑11: Concentrations of PCP (ng/L) in the Niagra River and St. Lawrence River, Canada.

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Location** | **Year** | **Number of Samples** | **Number of Detects** | **Percent Detects** | **MDL** | **Mean** | **SD** | **Median** | **Range** |
| Lower Niagara River\* | 1986-1993 | 308 | 89 | 29 | 0.2 | 0.62 | 1.62 | <0.2 | <0.2-21 |
| Niagara River daily automatic sampling station | 1986-1993 | 334 | 65 | 19 | 0.2 | 0.44 | 1.48 | <0.2 | <0.2-18 |
| Niagara River overall | 1986-1993 | 642 | 154 | 24 | 0.2 | 0.53 | 1.55 | <0.2 | <0.2-21 |
| St. Lawrence River\*\* | 1989-1991 | 21 | 0 | 0 | 0.2 | <0.2 | <0.2 | <0.2 | <0.2 |

\* 2.4 KM-1.5 MI,

\*\* South channel

### Air

1. Concentrations of PCP/PCA in air near Yellowknife, NT, Canada ranged from 0.43 to 3.68 ng/m3 (mean: 1.53 ng/m3) in 1994. In Saskatchewan, Canada concentrations in air ranged from 0.06 to 0.58 ng/m3 (mean 0.30 ng/m3) (Cessna et al. 1997). Because the analytical methodology in this study used diazomethane as a derivatizing agent the authors could not differentiate between PCP and PCA.
2. Zheng et al. (2011) in their summary of PCP studies found that more recent concentrations of PCP in outdoor air from urban areas in Canada and the U.S.A. ranged from ND to 1233 pg/m3 (1995-2001). Concentrations generally ranged though from ND to 51.5 pg/m3. Zheng et al. (2011) suggested that concentrations of PCP had decreased in indoor air between 1979 and 2001, They also found that there were no obvious changes in PCP levels in outdoor air between 1977 and 2001. However, the temporal trend comparison in outdoor air was made with data from Bolivia, Belgium, Switzerland (1977 to 1983), and the data for the comparison years 1995-2001 are from Canada and U.S.A. The validity of comparing across decades from different areas is questionable.
3. Concentrations in two air sampling stations in the mountains of La Paz, Bolivia measured 0.93 and 0.25 μg/1000m3 of air (= 0.25 pg/L). Concentrations in four Belgium samples ranged from 5.7 to 7.8 μg/1000m3 (5.7-7.8 pg/m3). These data were used by Zheng et al. (2011) and are from 1977.
4. 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.
5. Other studies of organochlorine compounds in air samples from remote locations (e.g., Su et al. 2008), do not report detection of PCP.
6. In a Swedish study (IVL Report B1474, June 2002, report not provided), samples were collected for analysis of PCP in air. The results show that the environmental levels of PCP in Sweden are generally lower than recommended quality limit values (details not provided). Slightly higher concentrations were detected near some potential point sources, but these were still below proposed critical levels. In air, PCA was detected in higher levels than PCP. Sweden stated that “it is likely that possible long-range transport of PCP occurs in the form of PCA.”

### Sediment/Soil

1. 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. The concentration units “μg/L” are unusual, however, this report was not submitted and units are shown as written in Borysiewicz 2008.
2. Measurements in the Elbe River taken in the framework of the Intnerational 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.
3. Between April and September 2010 PCP concentrations in sediment in the River Narva, Estonia and two points in Lake Peipus (Estonia) remained below the limit of quantification (0.1 μg/kg dw) and PCP was not detected in coastal sediment
4. Zheng et al. (2011) in their summary of PCP studies found that more recent (1991-1996) average concentrations of PCP in sediment had a range of 0.9 to ca. 40 μg/kg dw. Studies were from the UK, Netherlands, Germany and France. Spatial or temporal patterns and comparisons cannot be made from most data as provided by Zheng et al. (2011) because there is insufficient data or sites were only sampled once. PCP concentrations in the Netherlands in the Rhine/Waal averaged 29.7 μg/kg dw in 1991 and were 22.4 μg/kg dw in 1992-1994. In the Meuse/Maas sampling sites PCP concentrations averaged 25 and 23.4 μg/kg dw in 1991 and 1992-1994, respectively. The southern estuaries in the Netherlands had average concentrations of PCP in sediments of 26.5 and 24 μg/kg in 1991 and 1992-1994, respectively. With only two data points and without some idea of the error around these averages it is impossible to tell definitively if concentrations are truly decreasing.
5. PCP was not detected in marine sediment from Norway between 2004 and 2008.
6. In a Swedish study (IVL Report B1474, June 2002, report not provided), samples were collected for analysis of PCP in soil, sediment, and sludge. The results show that the environmental levels of PCP in Sweden are generally lower than recommended quality limit values (details not provided). Slightly higher concentrations were detected near some potential point sources, but these were still below proposed critical levels. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge. Sweden stated that “it is likely that possible long-range transport of PCP occurs in the form of PCA.”
7. 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).

Table 5‑12: Concentrations of PCP in Water, Air, Sediment and Soil in Monitoring Studies

| **Compartment** | **Location** | **Concentration** | **Comment** | **Reference** |
| --- | --- | --- | --- | --- |
| **Fresh Water** | | | | |
| 1990-1997 | Netherlands, Germany, Belgium | 0.01-0.17 μg/L | PCP concentrations declined sharply after early 1990 | Borysiewicz 2008 |
| 1995 | Seine River, France | 0.03 μg/L | Average concentration |
| 1990-1992 | United Kingdom | <1 μg/L | Median concentrations |
| 1994-1996 | United Kingdom | 0.15, 0.20, 0.02 μg/L | Declined from 1990-1992 likely due to use restrictions |
| 1988-1993 | River Elbe, Germany |  | Concentrations generally decreased 1988-1993 |
| 1990-1991 | Rhine River |  | Concentrations increased compared to 1980-1989 |
| 1997-2010 | River Elbe |  | Yearly averages and monthly variations at different monitoring sites |
| 1994-1998 | EU Member states | Median: 0.071 μg/L |  |
| No date | U.S.A. surface water | 0.04 -1.0 μg/L | Mean: 0.41 |
| No date | U.S.A ground water | 0.04-1.64 μg/L | Mean 0.46 |
| 2004-2005 | Estonia | 0.4 μg/L (average) | LOQ | Estonian submission (Tallinn 2011)? |
| 1991-1996 | UK, Belgium, Germany, Netherlands France | 0.01-0.17 μg/L  0.001-0.012 μg/L | Freshwater  Marine water | Zheng et al. 2011 |
|  | Canada | Not detected |  | Hoferkamp et al. 2010 |
| **Sea Water** | | | | |
| 1983-1997 | North Sea, coastal and estuaries of Germany, Netherlands, UK | ND-0.79 μg/L | “Typical concentration”: 0.07 μg/L; decreasing trends | Borysiewicz 2008 |
| 1993-1997 | UK, Netherlands | 0.001-0.012 μg/L |  | Zheng et al. 2011 |
| **Air** | | | | |
| 1994 | Yellowknife, N, Canada | 0.43-3.68 ng/m3 | Analytical methodology made it impossible to differentiate between PCA and PCP | Cessna et al. 1997 |
| 1994 | Saskatchewan, Canada | 0.06-0.58 ng/m3 |
| 1995-2001 | Canada, U.S.A | ND-1.233 ng/m3 | General range was ND-0.0515 ng/m3 | Zheng et al. 2011 |
| 2000-2003 | Canadian Arctic | Not Detected |  | Su et al. 2008 |
| **Sediment/Soil** | | | | |
| 2004-2005 | River Narva, Lake Peipus, Estonia | <LOQ (0.1 μg/kg dw) |  | Estonian (Tallinn 2011)? |
| 1991-1996 | UK, Netherlands, Germany, France | 0.9- ca. 40 μg/kg dw |  | Zheng et al. 2011 |
| 2004-2008 | Norway | Not detected | Marine sediment | TA 2564/ 2009 in Norwegian information submission to POPRC |

Shaded rows indicate information from remote areas

### Biota

1. The presence of PCP in biota from remote areas may be due in part to the metabolism of organochlorine substances already present in these areas as well as uptake of the parent PCP (see Section 5.4).
2. The first peer-reviewed scientific studies of PCP in sub-arctic wildlife came out in 2004 and showed contamination of the eggs of 4 Norwegian bird-of-prey species – golden eagles, ospreys, peregrine falcons, and white-tailed sea eagles (Berger et al. 2004). Concentrations ranged from <LOQ to 1350 pg/g ww.
3. Hoekstra et al. (2003) determined that PCP was the most abundant halogenated phenolic compound found in the Arctic bowhead whale plasma.
4. The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 2009) did not detect PCP during 4 years of the study (2000-2003).
5. Letcher et al. (2009) found PCP/PCA at mean concentrations of 1.0±0.4 ng/g lw in all ringed seals (n= 15) sampled from East Greenland (collected in 2002). The results of this study should be interpreted with caution given that PCP is major metabolite of HCB in animals.In addition, the analytical method entailed methylation using diazomethane that would have made it impossible to differentiate between PCA and PCP. These confounding factors make the accuracy of these BMFs questionable.
6. Pentachlorophenol was not detected in blue mussel and cod liver in Norway between 2004 and 2008 (Annex E Information submitted by Norway, 2013).
7. Darnerud et al. (2006 in Swedish Annex E submission)) estimated the mean Swedish per capita intake of selected POPs and compared the results with a previous intake estimation from 1999. A variety of contaminant groups including PCP were determined in fish/fish products, meat/meat products, dairy products, egg, fats/oils, cereal products, vegetables and fruits. PCP was only found in fish and meat above the LOQ. Generally, the levels of POPs in food from the 2005 food samples appeared to be lower than in the study from 1999. Sweden did not provide details to determine if PCP was one of the POPs that showed a decrease and a literature search for this information was unsuccessful to confirm these statements.
8. In a Swedish study (IVL Report B1474, June 2002, report not provided), samples were collected for analysis of PCP and PCA in biota. PCA and PCP concentrations were comparable in biota. No data was provided and the report was not submitted by Sweden.
9. In a study of PCP concentrations of pine needles in Saskatchewan, Canada, researchers found PCP in 100% of the samples at concentrations ranging from 0.42-2.08 ng/g, indicating widespread distribution as an atmospheric pollutant (Thompson and Treble, 1995). The extraction method however, included a diazomethane derivatization step, therefore, they are unable to differentiate between PCA/PCP in this study.
10. Erkisson et al. (1989) studied PCP/PCA in pine needles from Europe (Eriksson et al. 1989). Because the analytical technique also included treatment with diazomethane it is impossible to differentiate between PCA and PCP in this study. The samples could have contained all PCP or PCA or some combination, but due to the methylation step they could only measure both as PCA. Eriksson et al. (1989) found that PCP/PCA was found in pine needles across Europe (range = 0.09 to 0.77 ng/g fw), with levels in Swedish samples (0.85 to 1.39 ng/g fw) close to two times that found in the rest of Europe (Eriksson et al. 1989). They noted that atmospheric transport was unlikely from Europe to Sweden due to unfavourable prevailing winds and conclude that “there are no apparent changes in concentration levels over the years…” and that “the source is still present”.

Table 5‑13: Concentrations of PCP in Biota from Monitoring Studies

| **Compartment** | **Location** | **Concentration** | **Comment** | **Reference** |
| --- | --- | --- | --- | --- |
| Birds eggs | Norway | <LOQ to 1350 pg/g ww |  | Berger et al. 2004 |
| Invertebrates and vertebrates | Global locations |  | Potential decreases in concentration | Zheng et al. 2011 |
| Marine vertebrates | Global locations |  | Potential increases in concentration |
| Bowhead Whale plasma | Arctic |  | Most common halogenated phenolic compound | Hoekstra et al. 2003 |
| Fish | U.S.A. | Not detected | 2000-2003 | U.S. EPA 2009 |
| Ringed Seals | East Greenland | 1.0 ± 0.4 ng/g lw | 2002 | Letcher et al. 2009 |
| Polar Bears | East Greenland |  | BMFs were reported, but should be interpreted with great care. PCP is a metabolite of HCB in mammals and is another potentially significant source of PCP in this study. Extraction method used diazmethane so unable to differentiate between PCA and PCP |
| Pine Needles | Saskatchewan, Canada | 0.42 - 2.08 ng/g | Extraction method used diazmethane so unable to differentiate between PCA and PCP | Thompson and Treble 1995 |
| Pine Needles | Europe | 0.09 to 1.39 ng/g fw | Concentrations almost 2 x higher in Sweden compared torest of Europe, unable to differentiate between PCA and PCP due to diazomethane | Eriksson et al. 1989 |
| Blue Mussel and cod liver | Norway | Not Detected | 2004-2008 | UNEP-POPS-POPRC7FU-SUBM-PCP-Norway-120702.En[1]) |

1 BMF values in liver should be viewed carefully relative to lipid and brain considering the liver is primarily responsible for metabolism

### Human Biomonitoring Information for PCP

1. Sandau (2002) reported PCP as the dominant chlorinated phenolic compound in blood samples from Nunavik (Inuit people) and southern Quebec adults in Canada. The researchers noted that PCP may supercede HO-PCBs as the chlorinated compound of highest concern in humans.
2. 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 measured at 642 pg/g plasma.
3. PCP was the dominant organochlorine compound, when compared with PCB and several other phenol compounds, found in the blood serum of pregnant and lactating women in Sweden, analyzed on a wet weight basis up to 3 ng/g serum in early pregnancy. The researchers found that the levels did not change significantly during pregnancy and observed a significant increase from late pregnancy to three weeks after delivery (Larsdotter et al. 2005).
4. In a study of the levels of PCP and other compounds in maternal blood serum, cord blood, and breast milk of women in Sweden, Guvenius et al. (2003) results show that the fetus is likely to be continuously exposed to PCP during development, implicating a risk potential risk for developmental disturbances. PCP was the dominant phenolic compound in the maternal blood plasma, cord blood plasma, and breast milk samples of Swedish women with median levels of 2.83, 1.96, and 0.02 ng/g fresh weight, respectively (n=15), with PCP levels in maternal and cord blood plasma levels 30 and 36 times higher than the sum of OH-PCBs on average. The maternal blood samples had notably high levels.
5. Sjödin et al. (2000) determined PCP in blood plasma in Latvian and Swedish men and compared their consumption of fish as a possible factor influencing uptake of PCP (among other contaminants). Median levels (ng/g lw) (10-90 percentiles) of PCP in the no/low fish consumption group were 610 (240-3400) and 1600 (600-5000), in Latvian men and Swedish men, respectively. Median levels (ng/g lw) (10-90 percentiles) of PCP in the moderate fish consumption group were 420 (170-820) and 720 (460-1400), in Latvian men and Swedish men, respectively. Median levels (ng/g lw) (10-90 percentiles) of PCP in the high fish consumption group were 330 (140-1500) and 1100 (760-1800), in Latvian men and Swedish men, respectively.
6. The PCP level in plasma was inversely related to fish consumption and statistics showed that it was not affected by age, but was strongly correlated with the country in which the subjects lived, with the PCP levels being much lower in Latvia than in Sweden. Consumption of fish is not a major source of exposure to PCP. This conclusion is supported by the low concentration of this compound in fish muscle, in contrast to the higher concentrations seen in fish blood (Asplund et al. 1999). Further, the group with low fish consumption demonstrated levels of PCP that were approximately 50% of the corresponding levels in Swedish women in the early 1980s (Jensen, S. personal communication in Sjödin et al. 2000). HCB is metabolized to PCP to some extent, but this does not explain the high levels of PCP present in blood because the level of HCB was inversely correlated to that of PCP (r2 = –0.29, p < 0.05). The authors concluded that there must be other sources of PCP in the environment which remain to be identified. It can be speculated that exposure to PCP, due to its use as a wood preservative, occurs via indoor air, however, its use was banned in Sweden in 1975.
7. Sweden submitted further information pertaining to the levels of PCP observed in plasma (above study) and hypothesized that Swedish society was still being exposed to PCP through the importation of treated textiles or possibly via PCP formed as a by-product in combustion process and industrial processes where chlorine gas is used. It is not clear however, why Sweden would be exposed to PCP via these mechanisms more so than Latvia.
8. In the first population based study investigating plasma concentrations of pentachlorophenol, Rylander et al. (2012) found that PCP was one of the dominant organic contaminants within a representative population of women in Norway sampled in 2004. 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. “One of the main findings in the current study was that PCP was the second most dominating compound (711 ng/L w.w.) in this group of menopausal women from the general Norwegian population. The levels found were in the same range as samples from Canadian Inuit (801 ng/L, n=567). The authors note that; “High concentrations of PCP in human blood have been reported worldwide (Dirtu et al. 2010; Guvenius et al. 2003) also in Arctic populations (Sandanger et al. 2004)… Despite that, PCP has received little attention over the past years and to the best of our knowledge, this is the first population based study investigating plasma concentrations of PCP and its predictors.” It is interesting to note these findings of PCP in Norwegian women in spite of the fact that PCP is not in use in Norway and that the government of Norway estimates that PCP emissions in Norway have been reduced by 99% in the period from 1995 – 2009.
9. The German Environmental Survey for Children 2003/06 - GerES IV - Human Biomonitoring (Becker et al. 2008) reported on the levels of PCP in urine of children aged 3-14 years. They found that “None of the variables (sex, age, economic status, migrant status, area of residence (East or West Germany), or community size) was seen to have had a significant influence on the percentage of values representing quantifiable PCP levels.” Meaning that no variable was seen to affect the concentrations of PCP in urine of children (ages 3-14). Combining the data, concentrations of PCP in urine ranged from <0.60 to 9.71 μg/L with a detection frequency of 49% and a geometric mean of <0.06.
10. Fréry et al. (2013) report biological concentrations of PCP in a representative sample of the French population. The purpose of this study was to establish baseline levels of contaminants found in the French population. PCP was detected in 66.2% of urine samples (LOQ: 0.03 to 0.1 µg/L). The mean and median concentrations were 0.88 µg/g of creatinine (0.90 µg/L) and 0.90 µg/g of creatinine (0.85 µg/L), respectively. None of the values exceeded the German toxicological value HBM-II for PCP (30 µg/L (40 µg/g of creatinine)) or HBM-I for PCP (25 µg/L). One person, however, did exceed the HBM-I limit when the value was expressed on a creatinine basis (20 µg/g).
11. Schulz et al. (2007) summarized the PCP data for 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.
12. Schulz et al. (2007) also shows that concentrations of PCP in urine in adults aged 25-69 years old from the former West Germany have decreased during the sampling periods 1985/86, 1990/92, and 1998 from 4.4, 2.7 and 1.1 μg/L, (geometric means) respectively. Sampling also took place in the former East Germany during the 1990/92 and 1998 sampling periods, but there were no statistical differences between the two sites.
13. Dirtu et al. (2010) investigated the levels and profiles of PCBs and phenolic compounds in human blood serum and found that PCP accounted for up to 85% of the total quantified phenolics found in Belgian samples and 35% in Romanian samples.
14. In a birth cohort from Slovakia, PCP was more abundant in infant cord blood than any PCB or OH-PCB congener measured (Park et al. 2008). In a study of human milk from 50 women in Bratislava, PCP was the dominant chlorophenol, with a median concentration of 2.21 μg/kg of whole milk (Veningerova et al. 1996).
15. In a study of the levels of PCP in blood serum of 4-year old children born between 1997 and 1999 in urban and rural communities of Spain, mean levels of PCP were found to be 6.4 ng/mL ± SD of 6.0 (with minimum of 1.5 ng/mL and maximum of 35 ng/mL in the urban population; n=66) and 0.61 ng/mL ± SD of 0.69 (with minimum ND, maximum 4.7 ng/mL in the rural population; n=131).
16. In an exposure assessment of 257 children randomly selected from households and daycare centers in Ohio and North Carolina in the US, PCP was found in nearly all of the urine samples at similar levels found nationally in children ages 6-11, with arithmetic mean levels of 0.605 ng/mL with SD of 0.629 in the 128 children from North Carolina and arithmetic mean levels of 1.27 ng/mL with SD of 2.20 in the 126 children from Ohio. Maximum levels in the children’s urine samples were 3.45 ng/mL in North Carolina compared with a maximum level of 23.8 in Ohio. In the same study, PCP was found in more than 50% of the indoor air, outdoor air, and dust samples and children’s exposures were predominantly through inhalation (Wilson et al. 2007).
17. Bradman et al. (2003) detected PCP in amniotic fluid of women in California (USA), indicating direct exposure to the fetus during critical development periods.
18. 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 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 Biomonitoring data serve as a direct indicator of exposure to either PCP or a number of other chemicals which are metabolized to PCP. Due to the fact that PCP is metabolised in mammals, tissue concentrations likely represent recent or ongoing exposure. The information necessary to relate external exposure levels of PCP to internal dose is lacking (US EPA, IRIS, 2010).

## Monitoring Information for PCA

### Air

1. PCA has most often been detected in air compared to other abiotic matrices. It has been detected in potentially contaminated areas as well as remote Canadian and Russian Arctic locations.
2. Mean concentrations of PCA in air are generally below 5 pg/m3 in Arctic air, however a strong seasonal gradient is apparent in longer term studies such as Hung et al. 2003 (unpublished data) (). Hung et al. determined that concentrations of PCA in air at Alert, NU, Canada (a high Arctic site) usually peaked in winter/spring when accumulation in the atmosphere usually takes place over the Arctic due to reduced vertical mixing and low wind speed resulting in more stagnant conditions (Barrie et al 1998 in Hung et al. (pers. comm.). However, the winter/spring maxima appear to have decreased in later years and tend to be more episodic with less seasonal variability (2007-2009).
3. also clearly shows that concentrations of PCA in air at Alert have shown a sharp decline in the years 2003 to 2009 from a period of relatively consistent concentrations (1993 to 2002). It should be noted that most samples collected in 2005 and all samples collected in 2007 and 2009 were below detection limits and are replaced with 2/3 the instrument detection limit (IDL) according to their QA/QC protocol.
4. The actual cause of atmospheric concentration decline and changes in seasonal variability of PCA at Alert is unclear. A warming Arctic with higher temperatures and lower sea ice coverage could potentially enhance the degradation rate and air-surface (water, soil, ice and snow) exchange of PCA. The latter may result in a shift of PCA from air to other environmental media. A more detailed analysis of how different influencing factors may have affected air concentrations of PCA is needed in order to interpret the observed results. Such factors include changing source-receptor relationships, changes in deposition and partitioning properties of PCA due to temperature increase, degradation rates and PCA concentrations in adjacent media etc.
5. PCA has also been reported at several Arctic monitoring stations in Canada, the USA, and Russia with different seasonal profiles (Su et al 2008). Three episodes of elevated PCA concentrations were observed in June–August 2002 at Point Barrow, Alaska. Corresponding back-trajectories indicated that the air masses largely originated from the Eurasian portion of the Arctic Ocean or the Russian Arctic. Overall, mean and median concentrations of PCA measured at the Arctic monitoring stations were 4.9 and 3.8 pg/m3, respectively, which were comparable to those of g-HCH and endosulfan I.
6. 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, however, no details were provided by Sweden. They stated that “it is likely that possible long-range transport of PCP occurs in the form of PCA.”



Figure ‑: Time trend of PCA (gas+particle phase concentration C (pg/m3 ) in air at Alert (1993-2009).

### Sediment/Soil

1. PCA has been detected in sediment from impacted areas (Mississippi River, U.S.A.; Yangtze River, China; Alexandrian Harbour, Egypt, and the Yellow Sea) as well as remote areas. Concentrations are below 7.4 ng/g in all areas.
2. In remote areas (Canadian Arctic) levels were reported to be generally <1 ng/g dw, but found up to 4.52 ng/g dw.
3. Concentrations of PCA in a dated sediment core from Lake Hazen, NU, Canada (high Arctic) collected in 2006 range from <DL to 0.523 ng/g dw (Derek Muir, personal communication, unpublished data). The sediment core encompasses a time series from 1898 to 2005. Concentrations are much higher in the upper layers compared to the lower. demonstrates that HCB and PCA concentrations track each other quite well in the upper sediment layers (2001-2005), however, that trend was not apparent in deeper sediment layers (prior to 1996).
4. PCA was generally present in sediment profiles in Northern Canada (Yukon and Northern B.C.), at < 1 ng/g dw. The maximum concentration reported was 4.52 ng/g dw for Hanson Lake. (Rawn et al., 2001).
5. Concentrations of PCA in soil of the Taurus Mountains ranged from a low of 1.44 pg/g dw at 121 m to a high of 6.02 pg/g dw at 1881 m (Turgut et al. 2012). There was no correlation with altitude or any other variable that they measured (soil characteristics).

Table 5‑14: Concentrations of PCA and HCB in sediment core from Lake Hazen, NU, Canada

|  |  |  |
| --- | --- | --- |
| **Deposition year** | **PCA ng/g dw** | **HCB ng/g dw** |
| 2005 | 0.523 | 0.366 |
| 2004 | 0.201 | 0.160 |
| 2001 | 0.167 | 0.204 |
| 1996 | 0.320 | 0.352 |
| 1991 | 0.176 | 0.425 |
| 1984 | 0.065 | 0.580 |
| 1974 | 0.005 | 0.996 |
| 1971 | 0.122 | 1.026 |
| 1967 | 0.014 | 0.409 |
| 1963 | 0.081 | 0.272 |
| 1959 | 0.049 | 0.213 |
| 1955 | 0.028 | 0.131 |
| 1951 | 0.076 | 0.167 |
| 1947 | 0.005 | 0.106 |
| 1943 | 0.009 | 0.066 |
| 1939 | 0.050 | 0.061 |
| 1934 | 0.077 | 0.046 |
| 1929 | 0.172 | 0.037 |
| 1925 | 0.000 | 0.031 |
| 1919 | 0.034 | 0.016 |
| 1914 | 0.000 | 0.001 |
| 1909 | 0.000 | 0.000 |
| 1903 | 0.000 | 0.008 |
| 1898 | 0.012 | 0.000 |



Figure 5‑8: Overlay of PCA and HCB concentrations in Lake Hazen Sediment Core

1. Only two studies have shown PCA in soil (Finland and Sweden). In a Swedish study (IVL Report B1474, June 2002 , summary in English), samples were collected for analysis of PCP and PCA in soil, water, sediment, sludge. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge. Actual concentrations were not included in the information submitted by Sweden.

### Snow

1. Only two studies have shown PCA in snow. A brown snow event in the Canadian Arctic had very high concentrations and was a result of 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.

### Water

1. PCA is rarely found in water, in fact, only one study has shown PCA in water and this was from an impacted area of the Yangzte River, China.

Table 5‑15: Concentrations of PCA in various abiotic compartments.

| **Compartment** | **Year** | **Location** | **Concentration** | **Comment** | **Reference** |
| --- | --- | --- | --- | --- | --- |
| **Sediment** | 1998-1999 | Mississippi River, USA | ≤ 7.4 ng/g | PCP was not detected at any site, however, PCA was detected in almost every sample. The authors stated that it is probably present in sediments and converted to PCA. Concentrations were 2-4 times higher in the spring than in summer at every site (1989 and 1990). The main source of PCA in the spring is the Ohio River (highly contaminated industrial river). | Rostad et al. 1999 |
| **Brown Snow** | 1988 | Canadian Arctic | melted snow: 1230 pg/L  particles: 4.3 mg/g  Brown snow: 1442 pg/L | Long Range Transport via fine particulates. Origin of brown snow: Asia. | Welch et al. 1991 |
| **Water** | 1998 | Yangtze River, China | 0.6 ng/L | Sampled from an area with history of intensive use of polychlorinated organic compounds in agriculture and industry (including PCP as an agricultural pesticide) | Jiang et al. 2000 |
| **Sediment** | 1998 | Yangtze River, China | <1 ng/g | Values were taken from a graph and were difficult to read | Jiang et al. 2000 |
| **Soil** | 1989 | Finland | 0.01-1 µg/g (mg/kg) | Soil taken from a contaminated site. Sawmill has been closed for 30 years. | Haimi et al. 1993 |
| **Air** | 1985 | South Pacific Ocean | PCA:9.0 pg/m3  TCHQ-DE: 6.2 pg/m3 | Residues of TCHQ indicate that the source of PCP/PCA is local. TCHQ is unstable and is rapidly oxidized.  TCHQ-DE is a transformation product of PCA. | Atlas et al. 1986 |
| **Air** | 1985 | New Zealand | PCA:2.0 pg/m3  TCHQ-DE: 13.4 pg/m3 | Atlas et al. 1986 |
| **Air** | 2000-2003 | Arctic | Mean: 4.9 pg/m3 | Air concentrations showed strong seasonal/spatial variations. At ALT, air concentrations were lower in summer than in winter. At PTB, Concentrations were higher in summer than in winter. Three episodes of elevated PCA concentrations were observed in June-August 2002 at PTB. Back-trajectories indicate that the air masses largely originated from the Eurasian portion of the arctic Ocean or the Russian Arctic. At KNG and LFL, no patterns were observed. Mean and median concentrations were comparable at ALT, KNG, LFL and PTB. PCA air concentrations were low at VKK in July-September 2002. | Su et al. 2008 |
| **Air** |  | Canadian and Russian Arctic | 2.6 – 4.0 pg/m3 | Greater than 17% of all samples exceeded “breakthrough” (amt of chemical on front PUF/amt on back PUF) > 0.333. | Hung et al. 2005 |
| **Sediment** | Not available | Alexandrian Harbour, Egypt | “Near or below detection limits” | Potentially impacted by wastewater disposal (sewage and industrial) and agricultural run-off. | Barakat et al. 2002 |
| **Air** | 2004-05 | Durban, South Africa | “detected in all samples” max: 20±13 pg/m3 at one site (Nizam) | The elevated levels of PCA at Nizam were twice that measured at other sites. The authors indicated that this may suggest a local source, but uncertainties in measurements must be recognised. | Batterman et al. 2008 |
| **Sediment** | 2000, | Yellow Sea | Sediment: ND-0.04 ng/g dw  Biota: ND-0.95ng/g | Borders with China, North and South Korea. Highly impacted body of water. Industrial wastewater containing major pollutants from port cities, and non-point source contaminants of agricultural origin, oil discharge/exploration | Oh et al. 2005 |
| **Sediment** | 1992-1995 | Seven Yukon Lakes | Generally <1 ng/g dw. Max values in lakes range from 0.33-4.52 ng/g dw with lower concentrations in surface sediment | Concentrations of the OC compounds [HCH-lindane, CHL-chlordane, CBz-chlorinated benzenes (including TCP, TeCP and PCP) and PCA] were similar to concentrations reported for other Arctic lake sediments previously studied, and show little evidence of major local inputs. | Rawn et al. 2001 |
| **Sediment** | 1898-2005 | Lake Hazen | <0.52 ng/g dw |  | Muir, personal communication, 2006 |
| **Snow** | 2005 | Devon Ice-Cap, Canada | Flux = 0.4-0.6 ng.m2/yr | No details given. No trend analysis was possible | Muir 2007 in Hoferkamp et al. 2009-unpublished. |
| **Air** | 1993-1994 | Can. Arctic | 2.3-3.1 pg/m3  <0.01(LOD) – 20.5 pg/m3  Average weekly atmospheric concentrations  Particle: 0.01-0.02 pg/m3  Gas: 1.80-4.12 pg/m3 | Concentrations in Alert, Dunai and Tagish | Macdonald et al. 2000 |
| **Sediment** |  | Taurus Mountains, Turkey | 1.44 – 6.02 pg/g dw |  | Turgut et al. 2012 |

### Biota

1. Despite PCA being ubiquitous in air and found in multiple other media, PCA has not been detected in appreciable levels in biota in the field thereby contradicting the bioaccumulation potential predicted by the aquatic BCF value. Metabolism, depuration, biodegradability and bioavailability of PCA are likely significant factors in reducing the likelihood of bioaccumulation and biomagnification in aquatic systems.
2. 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 PCA has been found in aquatic biota in remote areas as well as in more populated areas.
3. Concentrations of PCA have been detected in fish in Siskiwit lake, a remote lake on Isle Royal in lake Superior (Swackhammer et al., 1988).
4. Four studies (Vorkamp et al. 2004; Bentzen et al. 2008 and Swackhammer et al. 1988 and Muir 2013) show low level residues in biota at or below detection limits in remote locations.
5. The concentrations of PCA in biota from the remote Canadian Arctic are summarized in (Muir 2013). Between 2000-2010, the range of concentrations in polar bears, ringed seal, 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-0.35 ng/g lipid and <LOD – 3.85 ng/g lipid, respectively. However, the animals were samples from different parts of the Arctic and sampling occurred during a 10 year time span so it is diffiults to compare concentrations with these variabilities.
6. It should be noted that Vorkamp et al. (2004) indicated that the concentrations in top predatory marine mammals did not exceed the concentrations in marine fish. Therefore, there is no indication of biomagnification. Of the various tissues analysed, the highest concentrations of PCA in marine mammals were found in the muscle, ranging from 0.08 ng/g lw (lipid weight) in harp seals to 1.1 ng/g lw in beluga. Compared with the results for chlorobenzenes and chlorinated pesticides, the concentrations of PCA are considered to be low.
7. The National Study of Chemical Residues in Lake Fish Tissues (U.S. EPA 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).

Table 5‑16 show the concentrations of PCA detected in biota.

**Table 5‑16:** Summary of Concentrations of PCA in Field Biota

| **Organism** | **Year/Location** | **Tissue** | **Concentration** | **Comments** | **Reference** |
| --- | --- | --- | --- | --- | --- |
| Caribou | 2004/Greenland | Muscle | ≤ 0.2 ng/g lw | The concentrations in marine mammals did not exceed the  concentrations in marine fish. Thus there was no indication of biomagnification.  Compared with the results for  chlorobenzenes and chlorinated  pesticides, the concentrations of PCA are considered to be low.  Of the seabird samples analysed, the only median concentrations above the detection limit were found in king eider and thick-billed murre liver. | Vorkamp et al. 2004 |
|  | Blubber, kidney | Detectable residues |
| Atlantic Cod | Muscle, liver | 2.3 ng/g lw (median) |
| Capelin, Cod | Liver | 2.3 ng/g lw (median) |
| Snow crab | Muscle  Liver | 0.66 ng/g lw  0.45 ng/g lw |
| Atlantic Salmon | Muscle | <0.01 ng/g ww |
| Redfish | Muscle | <0.01 ng/g ww |
| King Eider, thick billed murre | Liver | 0.36 ng/g lw  0.22 ng/g lw |
| Harp Seal  Narwhal  Beluga | Muscle | 0.08 ng/g lw  0.54 ng/g lw  1.10 ng/g lw |
| Marine Invertebrates |  | Residues above the LOD in 6 of 10 samples |
| Fish | 1980-84, Rivers USA | Whole Body | 100 ng/g ww | in 30% of 112 stations | Schmitt et al. 1990 |
| Cassave | 2006, Tanzania | Roots  Leaves | 0.6 ng/g lw  2.1 ng/g lw |  | Marco et al. 2006 |
| Lake trout  White fish | 1988, Siskiwit Lake USA | Whole body | 3.6 ng/g lw  6.5 ng/g lw |  | Swackhammer et al. 1988 |
| Mussels | Finland | Whole body | <1 – 274 ng/g lw |  | Herve et al. 1988 |
| Mango | Tanzania 2002 | Leaves | <0.5 to 3900 lw |  | Marco and Kishimba 2007 |
| Oysters/Mussels | USA coastal waters | Whole body | <0.25-8.99 ng/g dw |  | Wade et al. 1998 |
| Bass  Carp | 2004, USA, Mobile River Basin | Whole Body | 0.06-0.38 ng/g ww  0.72-3.18 ng/g ww |  | Hinck et al. 2008 |
| Carp  Catfish | 2003, Colorado River and tributaries | Whole body | >0.1 ng/g in 46 of 48 samples, carp had >10 ng/g |  | Hinck et al. 2007 |
| *Pseudosciaena crocea*  *Collichthys niveatus* | 2000, Yellow Sea | Muscle  Liver | ND-0.95 ng/g dw  ND-0.02 ng/g dw  ND-0.27 ng/g dw  ND-0.04 ng/g dw |  | Oh et al. 2005 |
| Polar Bears | 2003, Beaufort Sea Coast, Alaska | Fat | <0.1-27 ng/g ww  <0.1-42 ng/g lw |  | Bentzen et al. 2008 |
| Fish | Salton Sea, Calif. | Muscle | 0.15-0.20 ng/g fw |  | Riedel et al. 2002 |
| Fish | U.S.A. | Muscle | <MDL to 9 ng/g | Detection frequency higher in bottom dwellers compared to predators | U.S. EPA 2009 |

Most sites are from impacted sites. Shaded rows indicate remote sites.

Table 5‑17: Summary of concentrations of PCA in biota from the Canadian Arctic (2000-2010 (Muir unpublished data).

| **Location** | **Sample Number** | **Number of Detects** | **% Detects** | **Concentration (ng/g lipid)** | | | |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Mean** | **SD** | **Median** | **Range** |
| **Polar bears (2003) (Alaska; Bentzen et al 2008)** | | | | | | | |
| Beaufort Sea | 57 | Not provided |  | 11 | 10 |  | <0.1-42 |
| **Ringed seal (2000-2009)** | | | | | | | |
| Arctic Bay | 19 | 5 | 26 | 0.21 | 0.15 | 0.16 | <MDL-0.48 |
| Arviat | 49 | 24 | 49 | 0.29 | 0.27 | 0.14 | <MDL-0.82 |
| Gjoa Haven | 22 | 10 | 45 | 0.25 | 0.11 | 0.24 | <MDL-0.48 |
| Grise Fiord | 15 | 0 | 0 | <MDL |  |  |  |
| Holman | 51 | 30 | 59 | 0.07 | 0.10 | 0.011 | <MDL-0.40 |
| Inukjuaq | 10 | 4 | 40 | 0.03 | 0.012 | 0.03 | <MDL-0.05 |
| Kangiksuluaaq | 3 | 0 | 0 | <MDL |  |  |  |
| Pangnirtung | 10 | 10 | 100 | <MDL |  |  |  |
| Resolute | 55 | 26 | 47 | 0.09 | 0.1 | 0.07 | <MDL-0.32 |
| Sachs Harbour | 37 | 26 | 70 | 0.005 | 0.021 | 0.001 | <MDL-0.11 |
| **Overall** | 271 | 135 | 50 | 0.11 | 0.174 | 0.03 | <MDL-0.82 |
| **Sea run Arctic char (2004-2009)** | | | | | | | |
| AR | 10 | 0 | 0 | <MDL |  |  |  |
| Cambridge Bay | 20 | 0 | 0 | <MDL |  |  |  |
| KA | 10 | 0 | 0 | <MDL |  |  |  |
| KK | 8 | 8 | 100 | 0.04 | 0.024 | 0.05 | 0.04-0.10 |
| Nain | 21 | 3 | 14 | 0.04 | 0.0057 | 0.04 | <MDL-0.05 |
| Pond Inlet | 10 | 0 | 0 | <MDL |  |  |  |
| PU | 10 | 0 | 0 | <MDL |  |  |  |
| VR | 5 | 0 | 0 | <MDL |  |  |  |
| **Overall** | 94 | 11 | 12 | 0.05 | 0.02 | 0.05 | <MDL-0.10 |
| **Landlocked char (2000-2009)** | | | | | | | |
| Amituk lake | 25 | 18 | 72 | 0.01 | 0.009 | 0.02 | <MDL-0.03 |
| Char Lake | 32 | 9 | 28 | 0.50 | 0.60 | 0.57 | <MDL-1.83 |
| Lake Hazen | 50 | 13 | 26 | 0.07 | 0.004 | 0.005 | <MDL-0.017 |
| Resolute Lake | 45 | 45 | 100 | 0.14 | 0.07 | 0.12 | 0.04-0.38 |
| **Overall** | 152 | 85 | 56 | 0.13 | 0.24 | 0.07 | <MDL-1.83 |
| **Lake trout (2002-2009)** | | | | | | | |
| Great Bear Lake | 10 | 10 | 100 | 0.04 | 0.026 | 0.04 | 0.02-0.08 |
| Great Slave Lake | 52 | 32 | 62 | 0.08 | 0.087 | 0.04 | <MDL-0.35 |
| **Overall** | 62 | 42 | 68 | 0.07 | 0.079 | 0.04 | <MDL-0.35 |
| **Burbot (2002-2009)** | | | | | | | |
| Great Slave Lake | 41 | 31 | 76 | 1.23 | 1.16 | 0.78 | <MDL-3.85 |
| **Overall** | 41 | 31 | 76 | 1.23 | 1.16 | 0.78 | <MDL-3.85 |

### Human Biomonitoring Information for PCA

1. No information on biomonitoring for PCA in humans was found.

## Summary of Monitoring Informmation

1. In air, PCP has been detected at low levels in remote areas, but is detected at higher concentrations closer to point sources such as urban areas (0.43-3680 pg/m3). PCA has been detected in air in remote areas and is generally detected more frequently and at higher concentrations than PCP. PCA concentrations in the Canadian Arctic have shown a decreasing trend since 2003.
2. PCP and PCA are generally not detected frequently in water. The highest median water concentration reported is 21 ng/L from the Niagara River (Muir, personal communication). Concentrations of PCP in water where PCP was used have decreased since uses were restricted. No concentrations in water for remote areas were reported. Concentrations of PCA were reported up to 0.6 ng/L from an impacted area of the Yantze River in China.
3. PCA has been reported in snow in the Canadian Arctic transported by fine soil particles.
4. Soils close to historical treatment facilities and/or sawmills still have relatively high concentrations of PCP.
5. Information on PCP concentrations in sediments in Europe from the 1990s range from 0.9-40 µg/kg dw. More recent information from Estonia indicated that PCP was not detected in freshwater or coastal sediments. Norway reported no detections of PCP in marine sediments (2004-2008).
6. PCA has been detected in sediments from various impacted sites (Mississipi River, U.S.A; Yangtze River, China, Alexandrian Harbour, Egypt, and the Yellow Sea) and in remote areas of Canada. All concentrations are below 7.4 ng/g.
7. Sweden reported slightly higher concentrations were detected near some potential point sources, but these were still below proposed critical levels. PCP concentrations were significantly higher than PCA concentrations in soil, sediment and sludge.
8. Various monitoring studies were submitted showing the detection of PCP in biota. However, this information is complicated by the fact that PCP is a metabolite of various other organochlorine substances and should be interpreted with caution. See Section 5.4.
9. PCP/PCA was found frequently in pine needles in Canada and in Europe at similar levels (<2.08 ng/g).
10. Although PCP is reported to be frequently detected in biota and was reported to be one of the most abundant halogenated phenolic substance in whale plasma, levels were reported at concentrations <1.35 ng/g in various biota including bird eggs and ringed seals.
11. PCA has also been detected in biota in both remote areas and close to point sources. Recent reported concentrations in aquatic species are below 2.3 ng/g lw. One study reported fish residue in wet weight (<3.18 ng/g ww). Residues in oysters/mussels are slightly higher and were reported to be <0.25-8.99 ng/g dw in U.S. coastal waters. Four studies (Vorkamp et al. 2004; Bentzen et al. 2008 and Swackhammer et al. 1988 and Muir, unpublished) show low level residues in biota at or below detection limits in remote locations. The information shows a slightly higher range of concentrations detected in polar bears (<n.d.- 42 ng/g lw) than the other marine mammals in one study. However, 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.
12. Residue levels in aquatic organisms reported in the 1980s were significantly higher than more recent studies. For example, in fish sampled from U.S. rivers in 1980-84, the highest value reported was 100 ng/g ww (Schmitt et al. 1990) whereas the highest value reported in 2009 U.S. fish survey was 9 ng/g (US EPA 2009). Residues in mussels from Finland ranged from <1-274 ng/g lw.

## Hazard Assessment

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

### Adverse effects on terrestrial organisms

**PCP**

1. PCP is highly toxic to mammals and birds. Some of the acute effects of exposure to commercial PCP are attributable to microcontaminants present in the technical preparation. Numerous studies have described the developmental effects of pentachlorophenol and its dioxin and hexachlorobenzene contaminants. The reported 5-day dietary LC50 value in Japanese quail is greater than 5139 mg/kg (Hill et al. 1975). LC50 values reported by Hill et al. (1975) for northern bobwhite, pheasant and mallard duck varied between 3400 and 4500 mg/kg food. Reported acute oral LD50 values for PCP are 380 mg/kg BW in mallard duck and 504 mg/kg BW in pheasant (Hudson et al. 1984). Vermeer et al. (1974) found 50 dead snail kites (Rostrhamus sociabilis) after extensive application of Na-PCP as a molluscicide in Surinam rice fields. High PCP residues were found in the brain (mean 11.3 mg/kg wet weight), liver (46.6 mg/kg), and kidney (20.3 mg/kg) of dead snail kites which had probably ingested Na-PCP contaminated snails. Effects on thyroxine in effects in mink and sheep fed 1-2 mg/kg (Beard and Rawlings 1998; Rawlings et al. 1998; Beard and Rawlings 1999: Beard et al. 1999: Beard et al. 1999a). These studies are also discussed in Section 5.11.2
2. Nesting of canaries (Serinus canarius) on straw containing 285 mg/kg PCP resulted in reduced hatch, high mortality of young during the first week and non survival to the age of 3 months (Dorrestein and Zelle 1979). Sublethal effects occur at levels lower than those mentioned above.
3. Poultry were fed graded levels (0, 1, 10, 100, and 1000 mg/kg) of pentachlorophenol (PCP) containing less than 0.0023% octachlordibenzo-p-dioxin (OCDD) for 8 weeks. Kidney weights were significantly increased by the 100 mg/kg and 1000 mg/kg PCP diet. Weights of all other organs including the body weights were significantly lowered by the 1000 mg/kg PCP. Hatching of eggs was reduced by 50% at a dose of 50 mg/kg egg and none hatched at 100 mg/kg diet (Stedman et al. 1980).

**PCA**

1. Male Sprague-Dawley rats and New Zealand White Rabbits were administered 14C-labelled PCA in corn oil by gavages as single doses of 25 mg/kg. Peak blood level of radioactivity occurred 6 hr after administration of the dose to rats and between 3 and 4 hr in rabbits; the blood elimination half-life ranged from 8 to 15 hr in rats and averaged 6 hr in rabbits. Rats excreted an average of 54% of the administered radiolabel in the urine and 32% in the feces during the 96 hr. The rabbits excreted an average of 84% and 13% of the radiolabel in the urine and feces, respectively during the 96 hr. Examination of the metabolites in the rat showed that 60% of the urinary radioactivity was attributable to tetrachlorohydroquinone (TCH), 3% to free PCP and 29% to conjugated PCP. Fecal metabolites were PCP (86%), TCH (4%), and polar metabolite(s) (10%). In the rabbits, 58% of the urinary radioactivity was attributable to TCH, 8% to free PCP and 34% to conjugated PCP. Fecal metabolites consisted of PCP and conjugated metabolites (Ikeda et al. 1994).
2. The acute toxicity of PCA was evaluated in mice. PCA was administered orally and intraperitoneally in male and female mice. The oral LD50 values were 318 (m) and 331 (f) mg/kg. The intraperitoneal LD50 values were 281 (m) and 293 (f) mg/kg for PCA (Renner et al., 1986). Based on the oral LD50 values for male and female mice, PCA was highly acutely toxic. In earthworms, Haimi et al. (1993) indicate that PCA was toxic at high concentrations ≥ 500 µg/g soil.

### Adverse effects on aquatic organisms

1. On an acute basis, both PCP and PCA are very highly toxic to aquatic invertebrates and highly toxic to fish. Sublethal effects to aquatic organisms were reported in the µg/L range. The most sensitive species appear to be pelagic organisms with most of the lowest observable effects concentrations and median lethal concentration from acute exposures occurring in the µg/L range ( and ). The median lethal toxicity of PCP to aquatic organisms is also in the µg/L range, for example for the water flea, Daphnia magna.
2. The similarity of effects thresholds in the aquatic environment between the two substances likely represents biotransformation of PCA to PCP within test organisms. PCA, untransformed within an organism, is likely less toxic than PCP because this methylated version of PCP loses its phenolic functionality.  It is well known, however, that chlorinated phenols such as PCP are more toxic than the baseline of narcosis because they have an uncoupling of oxidative phosphorylation mode of toxic action (McCarty and McKay 1993, IPCS 1989; Esher et al. 2011)

**PCP**

1. Toxicity data were provided in a report by Eurochlor for the Marine Environment (Eurochlor 1999). Acute LC50 values for fish ranged from 20 µg/L to 600 µg/L PCP. Acute LC50 values for sensitive invertebrates including species of Daphnia, lymnaeid snails, and oligochaetes ranged from 240 µg/L to 2,000 µg/L. These data indicate that fish are more sensitive than invertebrates to PCP. Acute 96-h EC50 values for aquatic plants ranged from 80 µg/L to 7,000 µg/L. Lowest chronic NOEC’s in the Eurochlor report varied between 2 and < 15 µg/L PCP with freshwater fish showing the lowest value.
2. Eisler (1989) summarized 96-hr LC50 concentrations for aquatic organisms. Table 1 summarizes the lower LC50 values provided by Eisler (1989) for salmonids and centrachids. Salmonids of the genus Oncorhynchus appear most sensitive. In contrast, invertebrate LC50 values are typically above 100 µg/L.
3. Pentachlorophenol can have more subtle effects that are important to the competitiveness of individuals in natural environments and to the sustainability of populations of organisms. Chronic toxicity endpoints involving reproduction and/or growth for PCP are listed in and range from 10-100 µg/L.The most sensitive endpoint in Orton et al. 2009 noted degenerative ovarian features in adult Xenopus laevis at 0.1 and 1µg/L.

**PCA**

1. On an acute basis, PCA appears to be very highly toxic to aquatic invertebrates and highly toxic to fish (EC50 values ranged from 27 µg/L to >1.2 mg/L). Acute LC50 values for fish and invertebrates ranged from 350 µg/Lto >1.2 mg/L and 10 to 27 µg/L, respectively. The most sensitive species appear to be pelagic organisms with most of the lowest observable effects concentrations and median lethal concentration from acute exposures occuring in the microgram per litre range . No information is available on the chronic toxicity of PCA.
2. The median lethal toxicity of PCP to aquatic organisms is also in the microgram per litre range, for example for the water flea, Daphnia magna. The similarity of effects thresholds likely represents biotransformation of PCA to PCP within test organisms. One referenced study suggests that PCA is approximately 1000x less toxic to fish than PCP, however, this was a non-standard toxicity test and details of the study were not available for review. PCA, untransformed within an organism, is likely less toxic than PCP because this methylated version of PCP is a neutral narcotic compound (i.e., looses its phenolic functionality).  It is well known, however, that chlorinated phenols such as PCP are more toxic than the baseline of narcosis because they have a uncoupling of oxidative phosphorylation mode of toxic action (McCarty and McKay 1993; IPCS, 1989)
3. PCA likely facilitates partitioning of PCP into aquatic and benthic organisms due to increased hydrophobicity. In addition, PCA is expected to have a longer half-life in aquatic organisms than PCP with an estimated half-life in the order of a few days vs a few hours for PCP (Glickman et al. 1977).  Therefore, at steady-state within an organism one would expect exposure and toxicity from both PCA and PCP, but the greatest toxic potency observed via PCP.  In addition, when assessing the toxicological potential of PCA it should be considered that PCA is demethylated back to PCP in living organisms as part of Phase I and II elimination processes.

Table 5‑18: Toxicity of PCP to various terrestrial and aquatic species.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Organism** | **Exposure** | **Endpoint value** | **Degree of toxicity** | **Reference** |
| **Terrestrial Organisms** | | | | |
| Rat |  | Oral LC50:  80-120 mg/kg | Highly toxic | St. Omer and Gadusek cited in ATSDR 2001; Borzelleca *et al.*cited in WHO 1987;Renner *et al.* cited in ATSDR 2001 |
| Rat |  | Oral LD50:  50-220 mg/kg | Moderately –highly toxic | St. Omer and Gadusek cited in ATSDR 2001 |
| Mice |  | Oral LC50:  117-177 mg/kg | Highly toxic | St. Omer and Gadusek cited in ATSDR 2001; Borzelleca *et al.*cited in WHO 1987;Renner *et al.* cited in ATSDR 2001 |
| *Coturnix japonica*  (Japanese quail) | 5 –d | Dietary LC50:  > 5139 mg/kg | Practically non-toxic | Hill *et al.* 1975 |
| *Anas platyrhynchos*  (Mallard duck) | 14-d | Oral LD50:  380 mg/kg | Moderately toxic | Hudson *et al.*1984 |
| *Phasianus colchicus*  (Pheasant) | 14-d | Oral LD50:  504 mg/kg | Slightly toxic | Hudson *et al.*1984 |
| Mink, sheep | 7-8 days | 1 mg/kg/day  Effects ont thyroxine (T4) |  | Beard and Rawlings 1998; Rawlings et al. 1998; Beard and Rawlings 1999: Beard et al. 1999:  Beard et al. 1999a |
| **Aquatic Organisms** | | | | |
| Aquatic plants | 96-h | EC50:  80-7000 µg /L |  | Eurochlor 1999 |
| Daphnia, lymnaeid snails, oligochaetes | 48-h | LC50:  240 – 2000 µg /L | Moderately – highly toxic | Eurochlor 1999 |
| *Oncorhynchus mykiss* (rainbow trout) | 96-h | LC50:  34 – 121 µg /L | Very highly toxic | Eisler (1989) |
| *Oncorhynchus tshawytscha (Chinook salmon)* | 96-h | LC50:  68 – 78 µg /L | Very highly toxic | Eisler (1989) |
| *Oncorhynchus nerka* (sockeye salmon) | 96-h | LC50:  63 – 68 µg /L | Very highly toxic | Eisler (1989) |
| *Salvelinus fontinalis* (brook trout) | 96-h | LC50:  128 µg /L | Highly toxic | Eisler (1989) |
| *Lepomis macrochirus* (bluegill) | 96-h | LC50:  120 – 350 µg /L | Highly toxic | Eisler (1989) |
| *Micropterus salmoides* (largemouth bass) | 96-h | LC50:  136 – 287 µg /L | Highly toxic | Eisler (1989) |
| *Salmo salar* (Atlantic salmon) | 96-h | LC50:  500 µg /L | Highly toxic | Eisler (1989) |
| Lamprey ammocoetes | 96-h | LC50: 31 µg/L | Very highly toxic | Andersen et al., 2010 |
| Marine Fish | 96-h | LC50:  20-600 µg /L | Highly –very highly toxic | Eurochlor 1999 |
| *Lymnaea stagnalis* | 16-d | NOEL: (no. viable eggs)  50 µg /L |  | Brooks 2001 |
| American Flagfish | 10-28 d | NOEL: (larval survival and reproduction )  55 µg /L |  | Brooks 2001 |
| *Oncorhynchus mykiss* | > 28 d | NOEL: (biomass and mortality )  10.9 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 32-d | NOEL: (hatchability, survival,growth )  16.5 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 90-d | NOEL: (survival,growth fry and juveniles )  6 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 90-d | NOEL: (survival,growth fry and juveniles )  36 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 90-d | NOEL: (survival,growth fry and juveniles )  > 130 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 32-d | NOEL: (hatchability, survival,growth )  44.9 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 32-d | NOEL: (hatchability, survival,growth )  63.7 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 32-d | NOEL: (hatchability, survival,growth )  27.6 µg /L |  | Brooks 2001 |
| *Pimephales promelas* | 32-d | NOEL: (hatchability, survival,growth )  32 µg /L |  | Brooks 2001 |
| *Chaetogammarus* | 56-d | NOEL: (growth )  100 µg /L |  | Brooks 2001 |
| *Oncorhynchus mykiss* | 18-d | NOEL: (no. of viable oocytes )  11 µg /L |  | Brooks 2001 |
| *Oncorhynchus mykiss* | 18-d | NOEL: (no. of viable oocytes )  12 µg /L |  | Brooks 2001 |
| *Daphnia magna* | 21-d | NOEL: (reproduction )  180 µg /L |  | Brooks 2001 |
| Mediterranean sea urchin embryos  *Paracentrotus lividus* | 48-50 h | NOEC: 30 µg /L  Altered deposition of larval skeleton at low concentrations. Modified the cytoskeleton assembly at high concentrations |  | Buono et al. 2011 |
| *Daphnia magna* | chronic | LOEC: 0.25 mg/L  NOEC: 0.12 mg/L  (reproduction) |  | Parks and LeBlanc 1996 |
| *Daphnia magna* | 48 h | LOEC: 62µg/L  (inhibition of glucose conjugates of testosterone) |  | Parks and LeBlanc 1996 |
| *Chironomus prasinus* | 16 days | Adult emergence and oviposition were tested. Slight effects noted at 10 mg/kg (0.75 mg/L) and 20 mg/kg (1.95 mg/L). Male/female ratio was statistically greater for the treatment group  NOEC (not reported): 5 mg/kg |  | Sanchez et al. 2005 |
| *Daphnia magna* | 48 h  16 days | Some mortality reported at 0.746-1.95 mg/L  Some effects on reproduction reported, but not related to endpoints |  | Sanchez et al. 2005 |
| *Xenopus laevis* | 5d | * + 1. µM (27 µg/L)   T3-antagonist activity |  | Sugiyama et al. 2005 |
| *Freshwater rotifer* | 96 h | NOEC>10 mg/L |  | Preston et al 2000 |
| carp | 7 and 15 days | EROD (liver microsome ethoxyresorufin O-deethylase) and GST (glutathione S-tranferases) activity in carp increased in 20 µg/L and 41 µg/L |  | Zhang et al. 2008 |
| Japanese medaka | 28 d | NOEC: 20 µg/L  The lowest observed effect was at 50 µg/L (testis-ova of male fish and kidney/liver lesions). Hatching rates and time to hatch were significantly affect in F1 generations when exposed to 200 µg/L. |  | Zha et al. 2006 |

Table 5‑19: Toxicity of Pentachloroanisole (PCA) to various terrestrial and aquatic species

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Organism** | **Exposure** | **Endpoint value** | **Degree of toxicity** | **Reference** |
| **Terrestrial Organisms** | | | | |
| Mice | N/A | Oral LD50:  318±22 (m)  331±22 (f) mg/kg | - | Renner et al. 1986 |
| Rat |  | Oral LD50:  ≥ 500 mg/kg | - | Dieke et al. 1947 |
| Earthworms |  | ≥500 µg/g | acutely toxic at high concentrations. | Salminen and Haimi 1991 in Haimi et al. 1993 |
| **Aquatic Organisms** | | | | |
| *Daphnia magna* | Acute (48 h) | 48-h EC50:  27.2 µg/L | Very highly toxic | Brooke 1991 |
| *Cladocera* | Acute (time not specified) | LC50: 27 µg/L | Very highly toxic | Sanchez-Bayo 2006 |
| *Hydra attenuate* | Acute (96 h) | MAC: 10 µg/L | Slightly to moderately toxic | Mayura et al. 1991 |
| *Pimephales promelas* | Acute (96 h) | LC50: 650 µg/L | Highly toxic | Brooke 1991 |
| LC50: >1.2 mg/L |  |

### Adverse effects on human health

**PCP**

1. The following information was extracted from the 2013 Annex E Information submitted by the U.S., (U.S. EPA 2010)

**Introduction**

1. During manufacture of PCP, the chemical is contaminated with impurities that consist of several congeners of the chlorophenols, chlorinated dibenzo-p-dioxins, and chlorinated dibenzofurans. Of the chlorinated dibenzo-p-dioxin and dibenzofuran contaminants, the higher chlorinated congeners are predominantly found as impurities within tPCP (technical/commercial grade PCP; approximately 90% purity). Use of the aPCP (analytical grade PCP; approximately 98% purity) first requires a purification process to remove the contaminants that are simultaneously created during the manufacturing of PCP.
2. Some toxic effects of exposure to commercial PCP are attributable to microcontaminants present in the technical preparation.

**Toxicity in humans**

1. Instances of PCP poisoning have been documented, indicating the potentially severe consequences of acute, high-dose exposures. Few studies have examined the effects of the lower exposures that occurred in occupational settings or through residential or environmental sources. Many of the available studies are relatively small (<50 participants) (Peper et al., 1999; Triebig et al., 1987; Klemmer et al., 1980; Begley et al., 1977) or may not be representative of the exposed population (Gerhard et al., 1999; Walls et al., 1998). Despite these limitations, there are indications of specific types of neurobehavioral effects seen with chronic exposure to PCP in non-occupational settings (Peper et al., 1999). A larger study of 293 former sawmill workers in New Zealand also suggests neuropsychological effects and respiratory diseases (McLean et al., 2009b). In addition, the results from a large nested cohort study of reproductive outcomes in offspring of sawmill workers (Dimich-Ward et al., 1996) indicate that specific types of birth defects warrant additional research. 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. Lymphocytes that function as natural killer cells capable of killing tumor cells, viral-infected cells, and antibody-coated cells are significantly decreased by exposure to PCP, thus adversely affecting a critical immune defense and perhaps playing a role in the development of cancers associated with PCP exposures (Ndodu and Whalen, 2010).

Meijer et al. (2012) examined the endocrine-disrupting effects from prenatal exposures to PCP and other organohalogens in infant boys and found that PCP levels correlated positively with sex hormone binding globulin (SHBG) (p=0.30) and negatively with inhibin B (-0.43). Prenatal exposure to organohalogens may have effects on sexual maturation in boys up to 18 months of age. A study of women daycare workers exposed to PCP, lindane, and PCDFs from treated wood found a significantly reduced birth weight (p=0.04) and length (p=0.02) among their offspring. The detrimental effect could not be attributed to any substance, but to interactions of PCP, HCH, and PCDDs/PCDFs. (Karmaus and Wolf, 1995).

1. The available epidemiologic studies support an association between PCP exposure and development of specific cancers: non-Hodgkin’s lymphoma, multiple myeloma, soft tissue sarcoma, and liver cancer (limited evidence). These studies used PCP-specific exposure assessment and in some cases, additional assessment of other chlorophenols and potential contaminants. PCP preparations are produced with methods that allow for the formation of contaminants, and degradation products occur naturally in most formulations. However, these contaminants are unlikely to spuriously produce the observed associations seen in the epidemiologic studies, given the difference in the patterns of cancer risk seen in studies of dioxins compared with the studies of PCP, and the relative strengths of the effects of different chemicals (PCP, other chlorophenols, dioxins, and furans) in the studies that examined more than one of these chemicals. Ruder and Yiin (2011) found an excess of cancers among PCP production workers in the U.S. of a priori interest, including trachea, bronchus and lung, non-Hodgkin’s lymphoma and leukemia, providing some support for the carcinogenicity of PCP.

It should be noted that in the epidemiological studies examining the cancer risk associated with exposure to PCP, exposures occurred predominantly via the inhalation and dermal routes.

**Biomonitoring data in humans related to potential effects from exposure to PCP**

1. Bradman et al. (2003) detected PCP in amniotic fluid of women in California (USA), indicating direct exposure to the fetus during critical development periods.
2. Dallaire et al. (2009) found a significant adverse relationship between maternal PCP levels and umbilical cord serum T4 concentrations in Inuit mothers from Nunavik and their infants, consistent with the hypothesis that chlorinated phenolic compounds inhibit T4 binding to TTR in humans. Reduced transfer of maternal fT4 during brain maturation of the fetus can impair neurocognitive function in infants. Sandau et al. (2002) conclude that PCP and possibly other phenolic compounds can alter thyroid hormones in newborn children which could result in neurodevelopmental harm in infant children.
3. Zheng et al. (2012) conducted a systematic review of PCP in the environment and humans in China and concluded that the use of PCP to treat the epidemic of snail-borne schistosomiasis was correlated with the occurrence of PCP in the environment and humans. They further concluded that thyroid-disrupting effects and cancer risk caused by PCP and PCDD/Fs even at low environmental levels in China’s schistosomiasis epidemic areas are of concern.
4. Sweden submitted further information pertaining to the levels of PCP observed in plasma (above study) and hypothesized that Swedish society was still being exposed to PCP through the importation of treated textiles or possibly via PCP formed as a by-product in combustion process and industrial processes where chlorine gas is used. It is not clear however, why Sweden would be exposed to PCP via these mechanisms more so than Latvia.
5. Biomonitoring data serve as a direct indicator of exposure to either PCP or a number of other chemicals which are metabolized to PCP. Due to the fact that PCP is metabolised in mammals, tissue concentrations likely represent recent or ongoing exposure. The information necessary to relate external exposure levels of PCP to internal dose is lacking (US EPA, IRIS, 2010).

**Non-cancer toxicity in animals**

**Oral exposure**

1. The toxicity of PCP in orally exposed animals was investigated in numerous studies in experimental animals. These studies indicate that PCP is toxic to the liver. In chronic studies in rats and dogs, liver toxicity was characterized primarily by increased incidence of chronic inflammation, cytoplasmic vacuolization, pigmentation, and hepatocellular necrosis as well as changes in liver weight (NTP, 1999; Mecler, 1996; Schwetz et al., 1978). Liver toxicity in mice was exhibited as necrosis, cytomegaly, chronic active inflammation, pigmentation, and bile duct lesions (NTP, 1989). The increased severity of liver toxicity observed in mice versus rats could be based, in part, on differences in biotransformation of PCP (Lin et al., 1997), but it is also noted that in the mouse studies, the PCP test material contained higher concentrations of chlorinated dibenzo-p-dioxin or dibenzofuran contaminants, which could contribute to the severity of the liver response. Liver toxicity in the dog (Mecler, 1996) was similar to that of the mouse, but the doses inducing toxicity were lower than those in the mouse (i.e., 1.5 mg/kg-day in the dog versus 17–18 mg/kg-day in the mouse). Studies using domestic or farm animals showed that pigs, but not cattle, exhibited similar liver toxicity as that observed in mice. Pigment deposition was also observed in the proximal convoluted tubules in the kidneys of rats (NTP, 1999).
2. Disruption of thyroid homeostasis has been observed following the administration of PCP. Several studies have reported decreased serum T4 and T3 levels in rats (Jekat et al., 1994) and cattle (Hughes et al., 1985; McConnell et al., 1980). Decreases in serum T4 have been observed in ram and ewe lambs (Beard et al., 1999a, b), mature ewes (Rawlings et al., 1998), and mink (Beard and Rawlings, 1998) after administration of PCP. TSH was unaffected by treatment with 1 mg/kg-day PCP in calves (Hughes et al., 1985) and sheep (Beard et al., 1999b). However, Jekat et al. (1994) reported a decrease in TSH accompanying the decrease in T4 levels in rats administered 3 mg/kg-day tPCP and aPCP. Considering that TSH acts on the thyroid to control production of T4, the concurrent decrease in TSH is in contrast to the expected TSH response to a decrease in T4 (TSH is generally expected to increase in response to a decrease in T4), which led Jekat et al. (1994) to suggest that this was due to interference with thyroid hormone regulation at the hypothalamic/pituitary level and possibly increased peripheral thyroid hormone metabolism. However, the available data do not allow for determination of the mechanism involved in the effects on T3, T4, and TSH following exposure to PCP. The effect of PCP on thyroid hormone homeostasis has been attributed to PCP and not to contaminants. Changes in thyroid hormones have been associated with effects (i.e., delayed myelination, neuronal proliferation, and synapse formation) on neurons. Considering that thyroid hormones may play a role in neurodevelopmental processes, the disruption of thyroid homeostasis that has been observed with PCP indicates a potential concern for critical period of development of the nervous system (CalEPA, 2006). However, the downstream effects associated with PCP and decreased T4 levels have not been explored.
3. In in vitro profiling of endocrine-disrupting effects in a set of recombined yeast cell strains, PCP showed agonistic and antagonistic effects at the estrogen receptor (ERα) and antagonistic effects at the progesterone receptor (Li et al. 2010). Ma et al. found that exposure of human adrenocortical carcinoma cell line (H295R cells) to PCP significantly reduced the production of testosterone and 17 β-estradiol. In H295R cells exposed to PCP, there was a dose-dependent reduction of cellular AMP (cAMP), indicating that PCP “may inhibit steroidogenesis by disrupting cAMP signaling.” PCP exposure resulted in effects on serum levels of reproductive and metabolic hormones, including marked decreases in thyroxin levels, increases in serum levels of insulin, as well as increases in the severity of oviductal intraepithelial cysts in ewes (Rawlings et al. 1998).
4. Elevated blood sugar levels (considered minor by Demidenko, 1969) and increases in organ weights were observed in rats and rabbits exposed to 21–29 mg/m3 PCP by inhalation for 4 months (Ning et al., 1984; Demidenko, 1969). Additional effects included anemia, leukocytosis, eosinophilia, hyperglycemia, and dystrophic processes in the liver. Minor effects were noted on the liver, cholinesterase activity, and blood sugar effects of animals exposed to 2.97 mg/m3 (calculated as 0.3 mg/kg-day PCP by Kunde and Böhme [1978]), a dose that is lower than the lowest NOAELs (1 mg/kg-day) observed in animals orally exposed to 28.9 mg/m3 PCP (Demidenko, 1969). Ning et al. (1984) reported significant increases in organ weights (lung, liver, kidney, and adrenal glands), serum γ-globulin, and blood-glucose levels at 21.4 mg/m3. The spleen weights of mice (NTP, 1989), rats (Bernard et al., 2002), and cattle (Hughes et al., 1985) were decreased following exposure to PCP.

**Inhalation exposure**

1. A Chinese study (Ning et al., 1984; translation) exposed weanling male rats to 3.1 or 21.4 mg/m3 PCP for 4 months. Rats in the 21.4 mg/m3 group exhibited significant increases, compared with control, in lung, kidney, liver, and adrenal gland weight. Additionally, the levels of blood-glucose were elevated in rats exposed to the high concentration of PCP. Ning et al. (1984) also observed statistically significantly increased serum γ-globulin and lung and liver weights in six rabbits (pooled males and females) exposed, in a similar manner, to 21.4 mg/m3. Demidenko (1969) reported results in which anemia, leukocytosis, eosinophilia, hyperglycemia, and dystrophic processes in the liver were observed in rats and rabbits exposed to 28.9 mg/m3 PCP (purity not reported) for 4 months. Animals exposed to the low concentration (2.97 mg/m3) exhibited effects on liver function, cholinesterase activity, and blood sugar that were considered minor and were not observed 1 month following exposure completion. Kunde and Böhme (1978), calculated an estimated dose of 0.3 mg/kg-day PCP based on the 2.97 mg/m3 concentration reported by Demidenko (1969). This calculation assumed 100% pulmonary uptake and absorption.

**Dermal Exposure**

1. A 13-week dermal toxicity study conducted in Sprague-Dawley rats (0, 100, 500, or 1,000 mg/kg-day; tPCP (88.9% purity) ) applied to clipped dorsal skin for 6 hours/day for 91 days (Osheroff et al., 1994). Some degree of skin irritation (acanthosis and chronic inflammation) was observed in both sexes at all doses of tPCP. Chronic inflammation was observed in 10, 80, and 100% of males and 0, 100, and 100% of females treated with 100, 500, and 1,000 mg/kg-day tPCP, respectively. Hepatocellular degeneration was observed in 90 and 100% of males at the mid and high doses, respectively, and in 20, 100, and 100% of females in the low, mid, and high doses, respectively. ALT and AST was statistically significantly increased in males and females in the 500 and 1,000 mg/kg-day dose groups. Relative liver weights were statistically significantly increased over controls in all dose groups for male rats. In females, the relative liver weights in animals of the 500 and 1,000 mg/kg-day dose groups were significantly greater than controls. Additionally, relative kidney weights were increased in 1,000 mg/kg-day males and in 500 and 1,000 mg/kg-day females, respectively. This study showed that PCP is absorbed from the skin at levels that caused liver toxicity. The study authors determined that the LOAEL for this study was 500 mg/kg-day based on dose-related increases in liver toxicity (hepatocellular degeneration, chronic inflammation, and statistically significant increases in hepatic enzyme induction). The NOAEL was 100 mg/kg-day.

**Developmental and Reproductive toxicity**

1. The majority of developmental toxicity studies on PCP provided no evidence of teratogenic effects, but some older studies showed toxic effects of PCP in offspring that occurred at dose levels below those producing maternal toxicity (Welsh et al., 1987; Schwetz et al., 1974a).
2. A two-generation reproductive toxicity study in rats showed that exposure to tPCP is associated with decreased fertility, delayed puberty, testicular effects, decreased litter size, decreased viability, and decreased pup weights at a dose of 30 mg/kg-day (Bernard et al., 2002). These effects occurred at the same doses causing systemic toxicity in parental animals. Studies in mink indicate some reproductive effects following exposure to PCP (Cook et al., 1997), although other studies in mink do not at doses of 1 mg/kg-day PCP (Beard et al., 1997; Beard and Rawlings, 1998). Additionally, no effects on reproduction were noted in sheep (both ewes and rams) at a PCP dose of 1 mg/kg-day (Beard et al., 1999a, b).

**Immunotoxicity**

1. Studies examining the immunotoxic effects of PCP showed that the humoral response and complement activity in mice were impaired by tPCP, but not by aPCP, when administered to adult animals (NTP, 1989; Holsapple et al., 1987; Kerkvliet et al., 1985a, b; 1982a). However, treatment of mice with aPCP from the time of conception to 13 weeks of age resulted in impaired humoral and cell-mediated immunity (Exon and Koller, 1983), suggesting that PCP, and not just the contaminants, induce immunotoxicity. Based on the limited available information, immunotoxic effects of PCP may be elicited, in part, through the presence of the dioxin/furan contaminants within PCP.

**Neurotoxicity**

1. In vitro neurotoxicity studies showed that PCP causes a dose-dependent irreversible reduction in endplate potential at the neuromuscular junction and interferes with axonal conduction in the sciatic nerve from the toad (Montoya and Quevedo, 1990; Montoya et al., 1988). An NTP (1989) study in mice showed only decreased motor activity in rotarod performance in male rats treated with tPCP for 5 weeks and increases in motor activity and startle response in females receiving purified and tPCP for 26 weeks. Another in vivo study showed that treatment of rats with PCP for up to 14 weeks caused biochemical changes in the rat brain (Savolainen and Pekari, 1979). The most definitive study showed that rats receiving PCP in drinking water for at least 90 days had marked morphological changes in sciatic nerves (Villena et al., 1992).

**Mutagenicity**

1. Studies examining the mutagenicity of PCP have shown that in a variety of test systems, PCP is nonmutagenic, with the exception of one study (Gopalaswamy and Nair, 1992) in which PCP exhibited a positive response for mutagenicity in the Ames Salmonella assay. In contrast to data on PCP, data for the TCHQ metabolite of PCP show positive mutagenic effects in CHO cells (Jansson and Jansson, 1991; Carstens et al., 1990; Ehrlich, 1990), an increase in micronuclei using V79 cells (Jansson and Jansson, 1992), covalent binding to DNA (Witte et al., 2000, 1985), and induction of DNA SSBs (Witte et al., 1985).

**Cancer**

1. Animal studies with PCP show evidence of adrenal medullary and hepatocellular tumors in male and female mice, hemangiosarcomas and hemangiomas in female mice, and nasal squamous cell carcinomas and mesotheliomas in male rats. Two well-conducted studies provide data for the carcinogenicity of PCP via the oral route in laboratory animals: one study in B6C3F1 mice (NTP, 1989) and another study in F344 rats (NTP, 1999). Two formulations of PCP (tPCP and EC-7) were carcinogenic in the mouse. Hepatocellular adenomas/carcinomas and adrenal medullary pheochromocytomas developed in male mice treated with tPCP or EC-7, and hepatocellular adenomas/carcinomas and hemangiosarcomas developed in female mice treated with tPCP or EC-7 and adrenal medullary pheochromocytomas developed in female mice treated with EC-7.
2. Several countries consider PCP carcinogenic to humans by all routes of exposure, including the U.S., Canada, and the EU.

**PCA**

1. Pentachloroanisole (PCA) is not industrially produced. As a result, the available toxicology database is deficient and does not permit a complete characterization of potential mammalian toxicity. The available scientific data has been summarized in the following text.

**Metabolism**

1. Male Sprague-Dawley rats and New Zealand White Rabbits were administered 14C-labelled PCA in corn oil by gavages as single doses of 25 mg/kg. Peak blood level of radioactivity occurred 6 hr after administration of the dose to rats and between 3 and 4 hr in rabbits; the blood elimination half-life ranged from 8 to 15 hr in rats and averaged 6 hr in rabbits. Rats excreted an average of 54% of the administered radiolabel in the urine and 32% in the feces during the 96 hr. The rabbits excreted an average of 84% and 13% of the radiolabel in the urine and feces, respectively during the 96 hr. Examination of the metabolites in the rat showed that 60% of the urinary radioactivity was attributable to tetrachlorohydroquinone (TCH), 3% to free PCP and 29% to conjugated PCP. Fecal metabolites were PCP (86%), TCH (4%), and polar metabolite(s) (10%). In the rabbits, 58% of the urinary radioactivity was attributable to TCH, 8% to free PCP and 34% to conjugated PCP. Fecal metabolites consisted of PCP and conjugated metabolites (Ikeda et al. 1994).
2. Vodicnik et al. (1980) determined that following injection of PCA into female mice that elimination of [14C]PCA equivalents was rapid with half-lives ranging from 5-10 hours in all tissues except the liver. Excretion of 14C was primarily through the urine. However, there was no evidence of parent PCA in either urine or feces. PCP was detected in the urine and feces at approximately 2 and 32% of applied radioactivity, respectively. The majority of the 14C was associated with the PCP conjugate. The authors concluded that PCA must be demethylated prior to conjugation and/or excretion.
3. National Toxicology Program (NTP, 1993) conducted toxicokinetics studies of PCA in F344 rats and B6C3F1 mice by gavage at doses of 10, 20 and 40 mg/kg and by i.v. @ 10 mg/kg. PCA was rapidly demethylated to PCP in both rat and mouse and the resulting PCP plasma concentrations were much higher than that of the parent PCA due to the much smaller apparent volume of distribution of PCP. Peak plasma concentration of PCA and PCP increased with dose in both rats and mice. Bioavailability of PCA was low in both rats and mice, increased with dose, and was sex independent. The biologic half-life of pentachlorophenol is relatively short, and bioaccumulation is slight (NTP, 1989).
4. Based on experimental animal metabolic data, PCA is not expected to bioaccumulate in humans due to its rapid metabolism (demethylation) to PCP, which is subsequently rapidly metabolised and eliminated. PCA is not expected to be a greater toxicological concern than PCP in humans.

**Acute Toxicity**

1. The acute toxicity of PCA was evaluated in mice. PCA was administered orally and intraperitoneally in male and female mice. The oral LD50 values were 318 (m) and 331 (f) mg/kg. The intraperitoneal LD50 values were 281 (m) and 293 (f) mg/kg for PCA (Renner et al., 1986). Based on the oral LD50 values for male and female mice, PCA was highly acutely toxic.

**Genotoxicity**

1. PCA was evaluated for its mutagenic potential in the mouse lymphoma forward mutation assay (McGregor et al. 1987). The dose levels ranged from 0-500 µg/ml. In the absence of metabolic activation, PCA did not produce a mutagenic response. In the presence of metabolic activation, PCA produced positive mutagenic response. It was concluded that there was sufficient evidence to suggest PCA can induce increases in mutant fraction in the presence of the metabolic activation. National Toxicology Program (1989) data also showed that PCA produced a positive response in the SCE test, but produced negative response in chromosomal aberration test. Overall, PCA can be considered to have mutagenic properties.

**Chronic Toxicity**

1. National Toxicology Program (NTP, 1993) also conducted chronic toxicity and carcinogenicity studies with PCA in F344 Rats and B6C3F1 mice. Increased incidence of benign pheochromocytomas of the adrenal medulla was noted in male F344 rats. Marginally increased incidence of benign pheochromocytomas of the adrenal medulla was observed in females F344 rats. Increased incidences of benign pheochromocytomas of the adrenal medulla and hemangiosarcomas of the liver were noted in male B6C3F1 mice. No evidence of oncogenicity was noted in female B6C3F1 mice given doses of 20 or 40 mg/kg. PCA administration was associated with increased incidences of adrenal medulla hyperplasia in female rats, and increased incidences of pigmentation in the renal tubule epithelium, olfactory epithelium, and hepatocytes of male and female rats. In addition, PCA administration was associated with 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. Overall, PCA can be considered to have carcinogenic properties.

**Developmental and Reproductive Toxicity**

1. Male and female Sprague-Dawley rats were exposed to dietary levels of 60, 200 or 600 ppm PCA for 181 days, through mating and pregnancy. The daily intakes were 0, 4, 12 or 41 mg/kg bw/day. A decrease in the number of corpora lutea and increased embryolethality was noted at 41 mg/kg bw/day. Reductions in fetal body weight and crown rump length of males were noted at 4 and 41 mg/kg bw/day. Female fetuses were unaffected. The results of this study suggested that PCA could be toxic to reproduction.

**Conclusion**

1. PCA is not industrially produced; therefore the toxicology database is deficient. A well characterized northern traditional diet is generally not available. Residue data for PCA in human diets is not sufficiently characterized to enable a robust estimate of human health risk to be developed. PCA is not expected to be a greater toxicological concern than PCP in humans.

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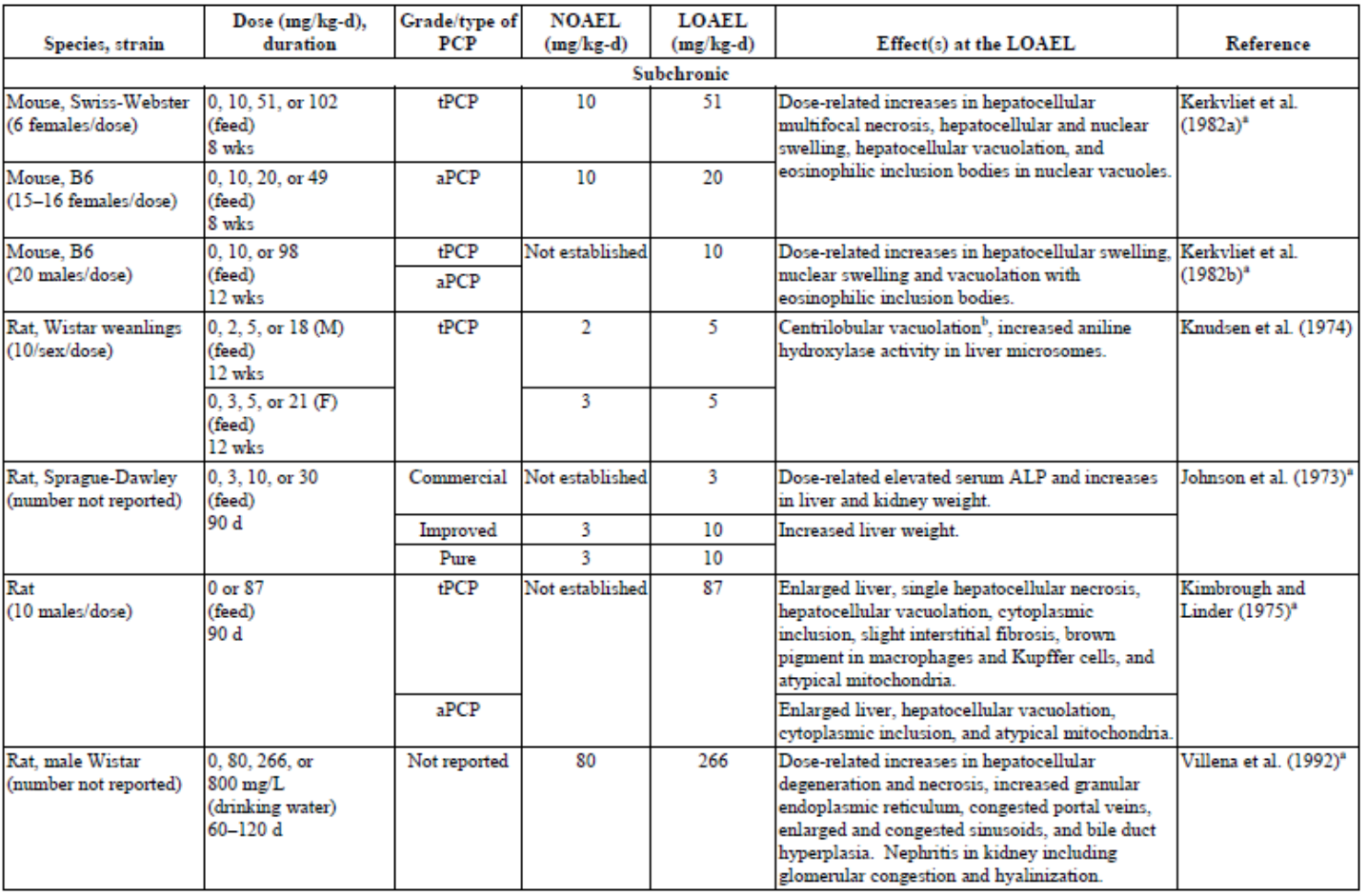


Table ‑. Subchronic chronic, developmental and reproductive oral toxicity studies for PCP





a NOAELs and LOAELs determined by the EPA for these studies; values are for both genders unless otherwise specified.

b Denotes statistical significance.

F = female; M = male



## Environmental Concentrations and Effects

1. Contaminated sites from historical misuse, improper handling/storage/disposal and illegal dumping are point sources of PCP in the environment. The highest environmental monitoring levels provided by parties were from contaminated sites and industrial rivers prior to the strict regulations reported by most parties in the 1990s. Monitoring studies have shown declining concentrations in countries where bans and regulations have been implemented.

**Effects to the environment**

1. The risk of PCP to the environment has been reviewed by several national regulatory authorities and is well established. Contaminated sites continue to be of concern and there is much effort worldwide to remediate these sites. Strict regulations have been implemented to minimise exposure in countries where PCP is still in use. The effects of PCP and PCA on remote areas as a result of long range transport has not been examined as thoroughly.
2. No concentrations of PCP or PCA in water were reported for remote areas. With the exception of one concentration from an industrial site in the U.K. in the early 1990’s, all other reported concentrations were below the lowest no observed effect concentration (larval survival and reproduction) of 6 µg/L for the fathead minnow (Brooks 2001).
3. The highest water concentration reported for PCA was 0.6 ng/L from an impacted area of the Yangtze River, China. This value is below the most sensitive sublethal endpoint reported for PCP or PCA (0.1 µg/L, Orton et al. 2009). It is also below the WHO provisional drinking water guideline of 9 µg/L for PCP (WHO, 2003). No concentrations for PCP or PCA in water were reported for remote areas, however, concentrations are expected to be lower than in more populated areas.
4. Reported sediment concentrations of <7.4 ng/g PCA cannot be compared to effects information as no information on sediment dwelling organisms was provided for either PCP or PCA. The concentration ranges in sediment appear to be similar for remote and populated areas.
5. 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 chemcials, McCarty and MacKay (1993) reference critical body burdens of 0.08 mmol/kg for chronic (effects not specified) and 0.3 mmol/kg for acute exposures specifically for PCP. The median internal threshold for narcosis is about 5 mmol/kg.
6. There is a 3-fold margin of exposure between the highest measured historical concentration in fish (1980-84), 100 ng/g (0.028 mmol PCA/kg) (Table 5‑20), Schmitt et al., 1990), to critical body burden estimates for PCA (0.08 mmol PCA/kg). Tissue residues reported for other sites and in particular, Arctic biota were much lower <1-10 ng/g (0.00028-0.0028 mmol/kg), which indicates there is a 30-fold margin of safety for PCA.
7. Reported environmental monitoring concentrations are generally lower than those levels expected to cause an environmental effect, particularly in remote areas.

Table 5‑21: Margin of Exposure for Fish compared to Critical Body Burdens

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Species** | **Maximum residue found in biota** | **Conversion to mmol PCA/kg** | **Critical Body residue estimate (PCP or PCA\*\*)** | **Margin of Exposure** | **remark** |
| Fish (whole body) | 100 ng/g from US river 1980-84 (Schmitt et al., 1990) | 0.028 PCA\* | 0.3 mmol/kg1 (acute)  0.08 mmol/kg (chronic) | 3 | Year is prior to regulations, residues are high |
| Fish (muscle) | 9 ng/g US EPA 2009 | 0.00252 | 0.08 mmol/kg | 32 |  |

\* ng/g x 10-6 g/ng x 280.37 g/mol / 1000 kg x 1000 mmol/mol

\*\*assume the same toxicity

**Effects on health**

1. PCP is detected in air, water, and soil throughout the world, as well as in the blood, urine, seminal fluid, breast milk and adipose tissue of humans (Zheng et al. 2011b and reported in Section 2.5.1). When biomonitoring information for PCP was compared between remote and more populated areas (Parks et al. 2008; Rylander et al 2012), levels were found to be similar. Sjödin et al. (2000) determined that in Sweden and Latvia, consumption of fish was not a major source of exposure to PCP; levels of PCP in blood plasma were related to the country where the person lived. Only one study, Fréry et al. (2013), compared biomonitoring information to an effect endpoint. In a study examining concentrations in a representative sample of the French population, the authors reported that none of the values exceeded the German toxicological value HBM-II for PCP (30 µg/L (40 µg/g of creatinine)) or HBM-I for PCP (25 µg/L). One person, however, did exceed the HBM-I limit when the value was expressed on a creatinine basis (20 µg/g).
2. All water concentrations reported in Section 5.8 are below the drinking water quality guideline of 9 µg/L reported in WHO (2003).
3. A suitable dietary human health risk assessment for PCP and PCA cannot be developed for people that are exposed to these compounds via their diet (traditional Inuit foods) because residues in their foods are not known, their diets are not well characterized and the toxicological database on PCA is lacking. However, the occupational exposures in wood treatment plants evaluated in U.S. EPA (2010) are expected to be much greater than incidental environmental or current dietary exposures to PCP based on general population exposure information available in ATSDR (2001). It should be noted that lifetime cancer risks were evaluated and included in the assessment.

# Synthesis

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

# Conclusion

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

# List of abbreviations

BAF Bioaccumulation factor

BCF Bioconcentration factor

BMF Biomagnification factor

bw body weight

CEC cation exchange capacity

d day(s)

dw dry weight

EC50 Effective concentration 50%

EPI Estimation Program Interface

f female

GIDMO Gravitational Induced Downward Migration of Oil

h hour

HCB hexachlorobenzene

hr hour

Kow octanol water partition coefficient

Koa octanol air partition coefficient

Kd adsorption quotient

Koc adsorption quotient normalized to organic carbon

LC50 lethal concentration 50%

LD50 lethal dose 50%

L/kg litres per kilogram

LOAEL lowest-observed-adverse-effect-level

<LOD below the level of detection

<LOQ below the level of quantification

lw lipid weight

m male

MDL method detection limit

g micrograms

μg/mL microgram per millilitre

mg milligrams

mg/kg milligrams per kilogram

mg/L milligrams per litre

mmol/Kg micromoles per kilogram

ng/g nanograms per gram

ng/L nanogram per litre

ng/mL nongram per millilitre

ng.m2/yr nanogram per square metre per year

ND not detected (below the level of detection)

NOAEL no-observed-adverse-effect-level

OC Organic carbon

OCDD octachlorodibenzo-p-dioxin

OSPAR Oslo Paris Convention

PCA pentachloroanisole

PCDD polychlorinated dibenzodioxins

PCNB quintozene

PCP pentachlorophenol

pg/L picogram per litre

pg/m3 picograms per cubicmetre

pKa acid dissociation constant

ppm parts per million

R.E.D. Reregistration Eligibility Decision

TBP tribromophenol

TCA tetrachloroanisole

TCH tetrachlorohydroquinone

TCHQ Tetrachlorohydroquinone

T1/2 half-life

TeCA Tetrachloroanisole

TeCP Tetrachlorophenol

TP transformation products

ww wet weight

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# APPENDIX I: *Summary of data submitted by Parties and observers for information specified in Annex E of the Convention*

|  |  |
| --- | --- |
| **Title** | **Reference** |
| Pentachlorophenol, Dossier prepared in support of a proposal of pentachlorophenol to be considered as a candidate for inclusion in the Annex I to the Protocol of the 1979 Convention on Long-Range Transboundary Air Pollution on Persistent Organic Pollutants | UNEP/POPS/POPRC.7/INF/5 |
| Addendum for the Risk Profile of Pentachlorophenol prepared for LRTAP | UNEP/POPS/POPRC.7/INF/5/Add.1 |
| Prior Informed Consent Procedure for Banned or Severely Restricted Chemicals in International Trade Decision Guidance Document, Pentachlorophenol and its salts and esters | UNEP/POPS/POPRC.7/INF/6 |
| Information on transformation of pentachlorophenol to pentachloroanisole and proposal by Japan to fill information gaps | UNEP/POPS/POPRC.7/19/Annex III |
| Information on PCP and its salts and esters   * Includes information received by Bulgaria, Canada, Japan, Kiribati, Latvia, Mexico, Monaco, Norway and Thailand. Information was also received from the Alaska Community Action on Toxics (ACAT) and international POPs Elimination Network (IPEN) and Wood Preservation Canada. | UNEP/POPS/POPRC.8/INF/7 |
| **Annex E information submitted by countries** |  |
| * Use information   **Documents:**   * Proposed Re-evaluation Decision Document * Re-evaluation Decision Document * Information on pentachloroanisole * Regulation of wood treatment facilities in Canada * Information on the management of dioxins/furans/HCB under the National Pollutant Release Inventory | Canada |
| * Letter indicating no information on production or use | Croatia |
| **Documents:**   * Monitoring information * Information also submitted for Lithuania and Latvia | Estonia |
| * Import and export information * Release information * References were supplied for hazard assessment, environmental fate and toxicology, however the references were not submitted | Mexico |
| * Information on sources, uses, releases, contaminants and exposure | Nigeria |
| * Letter indicating no information available | Romania |
| * Prohibited since 1982 * No production * Information on environmental monitoring will be sent at a later date * Information on releases not available | Slovak Republic |
| * No production/manufacturing * No additional information provided | Sri Lanka |
| * Banned in 1978 * Information on historical uses * Summary on PCP and PCA national monitoring data (IVL Report B1474, June 2002, summary in English) * Summary of PCP/PCA in human biomonitoring studies (Sjodin et al., 2000) * Information on dioxin releases in Sweden from chlorophenol treated wood   **Document:**   * Sjodin et al., 2000 | Sweden |
| * Production, use and release estimates * Toxic Release Inventory data   **Documents:**   * Reregistration Eligibility Decision for PCP (EPA-739-R-08-008) * Integrated Risk Information System (IRIS) Summary (EPA-635-R-09-004F) * U.S. EPA memo: Environmental Fate and Transport Assessment for PCP for Reregistration Eligibility Decision (RED) Process (EPA-HQ-OPP-2004-0402-0066) * U.S. EPA memo: A Qualitative Economic Impact Assessment of Alternatives to PCP as a Wood Preservative. EPA Docket: OPP2004-0402-0078 * 2010-March 2010-LRTAP Track A and B reviews and summaries * 2009-2011 Meeting Reports (Executive Body (EB), Working Group on Strategies and Reviews (WGSR), and the POPs Task Force (TF) | United States |
| * Additional information on sources, hazard, environmental fate and monitoring | Alaska Community Action on Toxics |
| * Production and use information for the United States/Canada/Mexico markets * Information on hazard, environmental fate, monitoring, national risk profiles for the U.S. and Canada   **Documents:**   * 2 letters with additional considerations and information * Other documentation has already been submitted | Wood Preservation Canada |

# APPENDIX II: Contaminant levels reported in Canadian PCP technical active ingredients.

Table A-1: Contaminant levels in PCP Technical Product, PCP#21785

| **Contaminant** | **Min Detected** | **Max Detected** | **Levl Unit** | **LOD LOQ** | **LOD LOQ Value** | **LOD LOQ Unit** | **Year** | **Remark** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| hexachlorobenzene (HCB) | 8.5 | 17.1 | PPM | NOT PROVIDED | - | - | 2006 | n=7, average=13 ppm analytical range 2-200 ppm batch production Jan-Nov 2006 |
| pentachlorobenzene (QCB) | - | - | - | - | - | - | 2008 | Suspected to be present due to synthesis used, but no analytical data received. |
| tetrachlorobenzene (non-specific) | - | - | - | - | - | - | 2008 | Suspected to be present due to synthesis used, but no analytical data received. |
| 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) | 28 | 175 | PPT | LOD | 22.8 | PPT | 2006 | n=7 |
| 1,2,3,7,8-pentachlorodibenzo-p-dioxin (1,2,3,7,8-PCDD) | 0.247 | 1.08 | PPB | LOD | 30.3 | PPT | 2006 | n=7 |
| 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin (1,2,3,4,7,8-HxCDD) | 1.1 | 86.8 | PPB | LOD | 78.9 | PPB | 2006 | n=7 |
| 1,2,3,6,7,8-hexachlorodibenzo-p-dioxin (1,2,3,6,7,8-HxCDD) | 232 | 344 | PPB | LOD | 81.5 | PPB | 2006 | n=7 |
| 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin (1,2,3,7,8,9-HxCDD) | 14.8 | 203 | PPB | LOD | 85.2 | PPB | 2006 | n=7 |
| 1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin (1,2,3,4,6,7,8-HpCDD) | 4.57 | 13.5 | PPM | LOD | 72.1 | PPB | 2006 | n=7 |
| octachlorodibenzo-p-dioxin (OCDD) | 34 | 130 | PPM | LOD | 435 | PPB | 2006 | n=7 |
| 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF ) | 0.022 | 0.068 | PPB | LOD | 15.4 | PPT | 2006 | n=7 |
| 1,2,3,7,8-pentachlorodibenzofuran (1,2,3,7,8-PCDF ) | 0.099 | 0.309 | PPB | LOD | 31.3 | PPT | 2006 | n=7 |
| 2,3,4,7,8-pentachlorodibenzofuran (2,3,4,7,8-PCDF ) | 0.431 | 2.74 | PPB | LOD | 2.5 | PPT | 2006 | n=7 |
| 1,2,3,4,7,8-hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF ) | 176 | 577 | PPB | LOD | 20.4 | PPB | 2006 | n=7 |
| 1,2,3,6,7,8-hexachlorodibenzofuran (1,2,3,6,7,8-HxCDF ) | 12 | 38.2 | PPB | LOD | 18.9 | PPB | 2006 | n=7 |
| 2,3,4,6,7,8-hexachlorodibenzofuran (2,3,4,6,7,8-HxCDF ) | 34.9 | 245 | PPB | LOD | 19.1 | PPB | 2006 | n=7 |
| 1,2,3,7,8,9-hexachlorodibenzofuran (1,2,3,7,8,9-HxCDF ) | 31.1 | 178 | PPB | LOD | 23.9 | PPB | 2006 | n=7 |
| 1,2,3,4,6,7,8-heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF ) | 3.14 | 17.7 | PPM | LOD | 37.4 | PPB | 2006 | n=7 |
| 1,2,3,4,7,8,9-heptachlorodibenzofuran (1,2,3,4,7,8,9-HpCDF ) | 0.681 | 3.15 | PPM | LOD | 53.4 | PPB | 2006 | n=7 |
| octachlorodibenzofuran (OCDF) | 54.4 | 283 | PPM | LOD | - | - | 2006 | n=7 |

Table A-2: Contaminant levels in PCP Technical Product, PCP#22024

| **Contaminant** | **Min Detected** | **Max Detected** | **Level Unit** | **LOD or LOQ** | **LOD or LOQ Value** | **LOD or LOQ unit** | **year** | **Remark** |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| hexachlorobenzene (HCB) | - | - | - | - | - | - | 2008 | Suspected to have similar HCB content as PCP# 21785. No analytical data received. |
| pentachlorobenzene (QCB) | - | - | - | - | - | - | 2008 | Suspected to have similar QCB content as PCP# 21785. No analytical data received. |
| tetrachlorobenzene (non-specific) | - | - | - | - | - | - | 2008 | Suspected to have similar QCB content as PCP# 21785. No analytical data received. |
| hexachlorobenzene (HCB) | 8.5 | 17.1 | PPM | LOD | - | - | 2006 | n=7 |
| 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) | 0.028 | 0.175 | PPB | LOD | 22.8 | PPT | 2006 | n=7 |
| 1,2,3,7,8-pentachlorodibenzo-p-dioxin (1,2,3,7,8-PCDD) | 0.247 | 1.08 | PPB | LOD | 30.03 | PPT | 2006 | n=7 |
| 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin (1,2,3,4,7,8-HxCDD) | 1.1 | 86.8 | PPB | LOD | 78.9 | PPB | 2006 | n=7 |
| 1,2,3,6,7,8-hexachlorodibenzo-p-dioxin (1,2,3,6,7,8-HxCDD) | 232 | 344 | PPB | LOD | 81.5 | PPB | 2006 | n=7 |
| 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin (1,2,3,7,8,9-HxCDD) | 14.8 | 203 | PPB | LOD | 85.2 | PPB | 2006 | n=7 |
| 1,2,3,4,6,7,8-heptachlorodibenzo-p-dioxin (1,2,3,4,6,7,8-HpCDD) | 4.57 | 13.5 | PPM | LOD | 72.1 | PPB | 2006 | n=7 |
| octachlorodibenzo-p-dioxin (OCDD) | 34 | 130 | PPM | LOD | 435 | PPB | 2006 | n=7 |
| 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF ) | 0.022 | 0.068 | PPB | LOD | 15.4 | PPT | 2006 | n=7 |
| 1,2,3,7,8-pentachlorodibenzofuran (1,2,3,7,8-PCDF ) | 0.099 | 0.309 | PPB | LOD | 31.3 | PPT | 2006 | n=7 |
| 2,3,4,7,8-pentachlorodibenzofuran (2,3,4,7,8-PCDF ) | 0.431 | 2.74 | PPB | LOD | 0.025 | PPB | 2006 | n=7 |
| 1,2,3,4,7,8-hexachlorodibenzofuran (1,2,3,4,7,8-HxCDF ) | 176 | 577 | PPB | LOD | 20.4 | PPB | 2006 | n=7 |
| 1,2,3,6,7,8-hexachlorodibenzofuran (1,2,3,6,7,8-HxCDF ) | 12 | 38.2 | PPB | LOD | 18.9 | PPB | 2006 | n=7 |
| 2,3,4,6,7,8-hexachlorodibenzofuran (2,3,4,6,7,8-HxCDF ) | 34.9 | 245 | PPB | LOD | 19.1 | PPB | 2006 | n=7 |
| 1,2,3,7,8,9-hexachlorodibenzofuran (1,2,3,7,8,9-HxCDF ) | 31.1 | 178 | PPB | LOD | 23.9 | PPB | 2006 | n=7 |
| 1,2,3,4,6,7,8-heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF ) | 3.14 | 17.7 | PPM | LOD | 37.4 | PPB | 2006 | n=7 |
| 1,2,3,4,7,8,9-heptachlorodibenzofuran (1,2,3,4,7,8,9-HpCDF ) | 0.68 | 3.15 | PPM | LOD | 53.4 | PPB | 2006 | n=7 |
| octachlorodibenzofuran (OCDF) | 54.4 | 283 | PPM | LOD | - | - | 2006 | n=7 |

**Table A-3: Total HCDD concentration expressed as TEQ (toxic equivalency quotient) in Canadian PCP technical products PCP#21785 and PCP#22024**

|  |  |  |  |
| --- | --- | --- | --- |
| **Congener** | **Maximum Detected (PPT) in PCP#21785 and PCP#22024** | **WHO Toxic Equivalency Factor (TEF 2005)** | **Toxic Equivalency Quotient** |
| *Chlorinated dibenzo-p-dioxin* |  |  |  |
| 2,3,7,8-TCDD | 175 | 1 | 175 |
| 1,2,3,7,8-PeCDD | 1.08 | 1 | 1.08 |
| 1,2,3,4,7,8-HxCDD | 86.8 | 0.1 | 8.68 |
| 1,2,3,6,7,8-HxCDD | 344 | 0.1 | 34.4 |
| 1,2,3,7,8,9-HxCDD | 203 | 0.1 | 20.3 |
| 1,2,3,4,6,7,8-HpCDD | 13500 | 0.01 | 135 |
| OCDD | 130000 | 0.0003 | 39 |
|  |  |  |  |
|  |  | Totals | 413.46 |
|  |  | Dioxins | 413.46 |
|  |  | Furans | 0 |
|  |  | PCB | 0 |

**Table A-4: Total chlorinated dioxins and furan expressed as TEQ (toxic equivalency quotient) in Canadian products PCP#21785 and PCP#22024**

|  |  |  |  |
| --- | --- | --- | --- |
| **Congener** | **Maximum Detected (PPT)**  **In PCP# 21785 and PCP# 22024** | **WHO Toxic Equivalency Factor (TEF 2005)** | **Toxic Equivalency Quotient** |
| *Chlorinated dibenzo-p-dioxin* |  |  |  |
| 2,3,7,8-TCDD | 175 | 1 | 175 |
| 1,2,3,7,8-PeCDD | 1.08 | 1 | 1.08 |
| 1,2,3,4,7,8-HxCDD | 86.8 | 0.1 | 8.68 |
| 1,2,3,6,7,8-HxCDD | 344 | 0.1 | 34.4 |
| 1,2,3,7,8,9-HxCDD | 203 | 0.1 | 20.3 |
| 1,2,3,4,6,7,8-HpCDD | 13500 | 0.01 | 135 |
| OCDD | 130000 | 0.0003 | 39 |
|  |  |  |  |
| *Chlorinated dibenzofurans* |  |  |  |
| 2,3,7,8-TCDF | 0.068 | 0.1 | 0.0068 |
| 1,2,3,7,8-PeCDF | 0.309 | 0.03 | 0.00927 |
| 2,3,4,7,8-PeCDF | 2.74 | 0.3 | 0.822 |
| 1,2,3,4,7,8-HxCDF | 577 | 0.1 | 57.7 |
| 1,2,3,6,7,8-HxCDF | 38.2 | 0.1 | 3.82 |
| 1,2,3,7,8,9-HxCDF | 245 | 0.1 | 24.5 |
| 2,3,4,6,7,8-HxCDF | 178 | 0.1 | 17.8 |
| 1,2,3,4,6,7,8-HpCDF | 17700 | 0.01 | 177 |
| 1,2,3,4,7,8,9-HpCDF | 3150 | 0.01 | 31.5 |
| OCDF | 283000 | 0.0003 | 84.9 |
|  |  |  |  |
| *Non-ortho substituted PCBs* |  |  |  |
| 3,3',4,4'-tetraCB (PCB 77) |  | 0.0001 | 0 |
| 3,4,4',5-tetraCB (PCB 81) |  | 0.0003 | 0 |
| 3,3',4,4',5-pentaCB (PCB 126) |  | 0.1 | 0 |
| 3,3',4,4',5,5'-hexaCB (PCB 169) |  | 0.03 | 0 |
|  |  |  |  |
| *mono-ortho substituted PCBs* |  |  |  |
| 2,3,3',4,4'-pentaCB (PCB 105) |  | 0.00003 | 0 |
| 2,3,4,4',5-pentaCB (PCB 114) |  | 0.00003 | 0 |
| 2,3',4,4',5-pentaCB (PCB 118) |  | 0.00003 | 0 |
| 2',3,4,4',5-pentaCB (PCB 123) |  | 0.00003 | 0 |
| 2,3,3',4,4',5-hexaCB (PCB 156) |  | 0.00003 | 0 |
| 2,3,3',4,4',5'-hexaCB (PCB 157) |  | 0.00003 | 0 |
| 2,3',4,4',5,5'-hexaCB (PCB 167) |  | 0.00003 | 0 |
| 2,3,3',4,4',5,5'-heptaCB (PCB 189) |  | 0.00003 | 0 |
|  |  |  |  |
| *Other PCB* |  |  |  |
| PCB 28 |  |  | 0 |
| PCB 52 |  |  | 0 |
| PCB 101 |  |  | 0 |
| PCB 138 |  |  | 0 |
| PCB 153 |  |  | 0 |
| PCB 180 |  |  | 0 |
|  |  |  |  |
|  |  | Totals | 811.5181 |
|  |  | Dioxins | 413.46 |
|  |  | Furans | 398.0581 |
|  |  | PCB | 0 |

# APPENDIX III: Modelled Summary Output for PCP and PCA

|  |
| --- |
| EPI Suite Results For CAS 000087-86-5 |

smile

SMILES : Oc(c(c(c(c1CL)CL)CL)CL)c1CL

CHEM : Phenol, pentachloro-

MOL FOR: C6 H1 CL5 O1

MOL WT : 266.34

------------------------------ EPI SUMMARY (v4.10) --------------------------

Physical Property Inputs:

Log Kow (octanol-water): ------

Boiling Point (deg C) : ------

Melting Point (deg C) : ------

Vapor Pressure (mm Hg) : ------

Water Solubility (mg/L): ------

Henry LC (atm-m3/mole) : ------

Log Octanol-Water Partition Coef (SRC):

Log Kow (KOWWIN v1.68 estimate) = 4.74

Log Kow (Exper. database match) = 5.12

Exper. Ref: HANSCH,C ET AL. (1995)

Boiling Pt, Melting Pt, Vapor Pressure Estimations (MPBPVP v1.43):

Boiling Pt (deg C): 311.71 (Adapted Stein & Brown method)

Melting Pt (deg C): 106.77 (Mean or Weighted MP)

VP(mm Hg,25 deg C): 1.08E-005 (Modified Grain method)

VP (Pa, 25 deg C) : 0.00144 (Modified Grain method)

MP (exp database): 174 deg C

BP (exp database): 309.5 deg C

VP (exp database): 1.10E-04 mm Hg (1.47E-002 Pa) at 25 deg C

Subcooled liquid VP: 0.00327 mm Hg (25 deg C, exp database VP )

: 0.436 Pa (25 deg C, exp database VP )

Water Solubility Estimate from Log Kow (WSKOW v1.42):

Water Solubility at 25 deg C (mg/L): 3.09

log Kow used: 5.12 (expkow database)

no-melting pt equation used

Water Sol (Exper. database match) = 14 mg/L (25 deg C)

Exper. Ref: YALKOWSKY,SH & DANNENFELSER,RM (1992)

Water Sol Estimate from Fragments:

Wat Sol (v1.01 est) = 45.051 mg/L

ECOSAR Class Program (ECOSAR v1.00):

Class(es) found:

Phenols

Henrys Law Constant (25 deg C) [HENRYWIN v3.20]:

Bond Method : 1.25E-007 atm-m3/mole (1.27E-002 Pa-m3/mole)

Group Method: 2.94E-007 atm-m3/mole (2.98E-002 Pa-m3/mole)

Exper Database: 2.45E-08 atm-m3/mole (2.48E-003 Pa-m3/mole)

For Henry LC Comparison Purposes:

User-Entered Henry LC: not entered

Henrys LC [via VP/WSol estimate using User-Entered or Estimated values]:

HLC: 1.225E-006 atm-m3/mole (1.241E-001 Pa-m3/mole)

VP: 1.08E-005 mm Hg (source: MPBPVP)

WS: 3.09 mg/L (source: WSKOWWIN)

Log Octanol-Air Partition Coefficient (25 deg C) [KOAWIN v1.10]:

Log Kow used: 5.12 (exp database)

Log Kaw used: -5.999 (exp database)

Log Koa (KOAWIN v1.10 estimate): 11.119

Log Koa (experimental database): None

Probability of Rapid Biodegradation (BIOWIN v4.10):

Biowin1 (Linear Model) : -0.1755

Biowin2 (Non-Linear Model) : 0.0000

Expert Survey Biodegradation Results:

Biowin3 (Ultimate Survey Model): 1.6340 (recalcitrant)

Biowin4 (Primary Survey Model) : 2.6765 (weeks-months)

MITI Biodegradation Probability:

Biowin5 (MITI Linear Model) : 0.0149

Biowin6 (MITI Non-Linear Model): 0.0031

Anaerobic Biodegradation Probability:

Biowin7 (Anaerobic Linear Model): -1.0946

Ready Biodegradability Prediction: NO

Hydrocarbon Biodegradation (BioHCwin v1.01):

Structure incompatible with current estimation method!

Sorption to aerosols (25 Dec C)[AEROWIN v1.00]:

Vapor pressure (liquid/subcooled): 0.436 Pa (0.00327 mm Hg)

Log Koa (Koawin est ): 11.119

Kp (particle/gas partition coef. (m3/ug)):

Mackay model : 6.88E-006

Octanol/air (Koa) model: 0.0323

Fraction sorbed to airborne particulates (phi):

Junge-Pankow model : 0.000248

Mackay model : 0.00055

Octanol/air (Koa) model: 0.721

Atmospheric Oxidation (25 deg C) [AopWin v1.92]:

Hydroxyl Radicals Reaction:

OVERALL OH Rate Constant = 0.5505 E-12 cm3/molecule-sec

Half-Life = 19.430 Days (12-hr day; 1.5E6 OH/cm3)

Ozone Reaction:

No Ozone Reaction Estimation

Reaction With Nitrate Radicals May Be Important!

Fraction sorbed to airborne particulates (phi):

0.000399 (Junge-Pankow, Mackay avg)

0.721 (Koa method)

Note: the sorbed fraction may be resistant to atmospheric oxidation

Soil Adsorption Coefficient (KOCWIN v2.00):

Koc : 4959 L/kg (MCI method)

Log Koc: 3.695 (MCI method)

Koc : 1.17E+004 L/kg (Kow method)

Log Koc: 4.068 (Kow method)

Experimental Log Koc: 3.7 (database)

Aqueous Base/Acid-Catalyzed Hydrolysis (25 deg C) [HYDROWIN v2.00]:

Rate constants can NOT be estimated for this structure!

Bioaccumulation Estimates (BCFBAF v3.01):

Log BCF from regression-based method = 3.045 (BCF = 1110 L/kg wet-wt)

Log Biotransformation Half-life (HL) = 0.3423 days (HL = 2.199 days)

Log BCF Arnot-Gobas method (upper trophic) = 2.405 (BCF = 254)

Log BAF Arnot-Gobas method (upper trophic) = 2.406 (BAF = 254.7)

log Kow used: 5.12 (expkow database)

Volatilization from Water:

Henry LC: 2.45E-008 atm-m3/mole (Henry experimental database)

Half-Life from Model River: 3.9E+004 hours (1625 days)

Half-Life from Model Lake : 4.256E+005 hours (1.773E+004 days)

Removal In Wastewater Treatment:

Total removal: 81.16 percent

Total biodegradation: 0.70 percent

Total sludge adsorption: 80.46 percent

Total to Air: 0.00 percent

(using 10000 hr Bio P,A,S)

Level III Fugacity Model:

Mass Amount Half-Life Emissions

(percent) (hr) (kg/hr)

Air 0.0377 466 1000

Water 4.12 4.32e+003 1000

Soil 93.7 8.64e+003 1000

Sediment 2.18 3.89e+004 0

Persistence Time: 7.81e+003 hr

....

**BIOWIN-PCP**

SMILES : Oc(c(c(c(c1CL)CL)CL)CL)c1CL

CHEM :

MOL FOR: C6 H1 CL5 O1

MOL WT : 266.34

--------------------------- BIOWIN v4.10 Results ----------------------

Biowin1 (Linear Model Prediction) : Does Not Biodegrade Fast

Biowin2 (Non-Linear Model Prediction): Does Not Biodegrade Fast

Biowin3 (Ultimate Biodegradation Timeframe): Recalcitrant

Biowin4 (Primary Biodegradation Timeframe): Weeks-Months

Biowin5 (MITI Linear Model Prediction) : Not Readily Degradable

Biowin6 (MITI Non-Linear Model Prediction): Not Readily Degradable

Biowin7 (Anaerobic Model Prediction): Does Not Biodegrade Fast

Ready Biodegradability Prediction: NO

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin1 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.1158 | 0.1158

Frag | 5 | Aromatic chloride [-CL] | -0.1824 | -0.9121

MolWt| \* | Molecular Weight Parameter | | -0.1268

Const| \* | Equation Constant | | 0.7475

============+======================================+=========+=========

RESULT | Biowin1 (Linear Biodeg Probability)| | -0.1755

============+======================================+=========+=========

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin2 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.9086 | 0.9086

Frag | 5 | Aromatic chloride [-CL] | -2.0155 |-10.0775

MolWt| \* | Molecular Weight Parameter | | -3.7820

============+======================================+=========+=========

RESULT |Biowin2 (Non-Linear Biodeg Probability) | 0.0000

============+======================================+=========+=========

A Probability Greater Than or Equal to 0.5 indicates --> Biodegrades Fast

A Probability Less Than 0.5 indicates --> Does NOT Biodegrade Fast

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin3 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.0564 | 0.0564

Frag | 5 | Aromatic chloride [-CL] | -0.2066 | -1.0330

MolWt| \* | Molecular Weight Parameter | | -0.5886

Const| \* | Equation Constant | | 3.1992

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RESULT |Biowin3 (Survey Model - Ultimate Biodeg)| | 1.6340

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TYPE | NUM | Biowin4 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.0397 | 0.0397

Frag | 5 | Aromatic chloride [-CL] | -0.1653 | -0.8267

MolWt| \* | Molecular Weight Parameter | | -0.3843

Const| \* | Equation Constant | | 3.8477

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RESULT |Biowin4 (Survey Model - Primary Biodeg)| | 2.6765

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Result Classification: 5.00 -> hours 4.00 -> days 3.00 -> weeks

(Primary & Ultimate) 2.00 -> months 1.00 -> longer

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TYPE | NUM | Biowin5 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.0642 | 0.0642

Frag | 5 | Aromatic chloride [-CL] | 0.0062 | 0.0309

MolWt| \* | Molecular Weight Parameter | | -0.7924

Const| \* | Equation Constant | | 0.7121

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RESULT |Biowin5 (MITI Linear Biodeg Probability) | 0.0149

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TYPE | NUM | Biowin6 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.4884 | 0.4884

Frag | 5 | Aromatic chloride [-CL] | -0.2191 | -1.0957

MolWt| \* | Molecular Weight Parameter | | -7.6889

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RESULT |Biowin6 MITI Non-Linear Biodeg Probability | 0.0031

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A Probability Greater Than or Equal to 0.5 indicates --> Readily Degradable

A Probability Less Than 0.5 indicates --> NOT Readily Degradable

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TYPE | NUM | Biowin7 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 1 | Aromatic alcohol [-OH] | 0.0807 | 0.0807

Frag | 5 | Aromatic chloride [-CL] | -0.4023 | -2.0114

Const| \* | Equation Constant | | 0.8361

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RESULT |Biowin7 (Anaerobic Linear Biodeg Prob)| | -1.0946

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A Probability Greater Than or Equal to 0.5 indicates --> Biodegrades Fast

A Probability Less Than 0.5 indicates --> Does NOT Biodegrade Fast

Ready Biodegradability Prediction: (YES or NO)

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Criteria for the YES or NO prediction: If the Biowin3 (ultimate survey model) result is "weeks" or faster (i.e. "days", "days to weeks", or "weeks" AND the Biowin5 (MITI linear model) probability is >= 0.5, then the prediction is YES (readily biodegradable). If this condition is not satisfied, the prediction is NO (not readily biodegradable). This method is based on application of Bayesian analysis to ready biodegradation data (see Help). Biowin5 and 6 also predict ready biodegradability, but for degradation in the OECD301C test only; using data from the Chemicals Evaluation and Research Institute Japan (CERIJ) database.

-----------------------------------------------------------------------

|  |
| --- |
| EPI Suite Results For CAS 001825-21-4 |

smile

SMILES : COc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM : Pentachloroanisole

MOL FOR: C7 H3 CL5 O1

MOL WT : 280.37

------------------------------ EPI SUMMARY (v4.10) --------------------------

Physical Property Inputs:

Log Kow (octanol-water): ------

Boiling Point (deg C) : ------

Melting Point (deg C) : ------

Vapor Pressure (mm Hg) : ------

Water Solubility (mg/L): ------

Henry LC (atm-m3/mole) : ------

Log Octanol-Water Partition Coef (SRC):

Log Kow (KOWWIN v1.68 estimate) = 5.30

Log Kow (Exper. database match) = 5.45

Exper. Ref: OPPERHUIZEN,A & VOORS,PI (1987)

Boiling Pt, Melting Pt, Vapor Pressure Estimations (MPBPVP v1.43):

Boiling Pt (deg C): 297.85 (Adapted Stein & Brown method)

Melting Pt (deg C): 84.27 (Mean or Weighted MP)

VP(mm Hg,25 deg C): 0.000344 (Modified Grain method)

VP (Pa, 25 deg C) : 0.0458 (Modified Grain method)

MP (exp database): 107-109 deg C

Subcooled liquid VP: 0.00219 mm Hg (25 deg C, Mod-Grain method)

: 0.292 Pa (25 deg C, Mod-Grain method)

Water Solubility Estimate from Log Kow (WSKOW v1.42):

Water Solubility at 25 deg C (mg/L): 0.3535

log Kow used: 5.45 (expkow database)

no-melting pt equation used

Water Sol Estimate from Fragments:

Wat Sol (v1.01 est) = 0.78504 mg/L

ECOSAR Class Program (ECOSAR v1.00):

Class(es) found:

Neutral Organics

Henrys Law Constant (25 deg C) [HENRYWIN v3.20]:

Bond Method : 7.12E-005 atm-m3/mole (7.21E+000 Pa-m3/mole)

Group Method: 1.94E-003 atm-m3/mole (1.97E+002 Pa-m3/mole)

For Henry LC Comparison Purposes:

User-Entered Henry LC: not entered

Henrys LC [via VP/WSol estimate using User-Entered or Estimated values]:

HLC: 3.590E-004 atm-m3/mole (3.638E+001 Pa-m3/mole)

VP: 0.000344 mm Hg (source: MPBPVP)

WS: 0.353 mg/L (source: WSKOWWIN)

Log Octanol-Air Partition Coefficient (25 deg C) [KOAWIN v1.10]:

Log Kow used: 5.45 (exp database)

Log Kaw used: -2.536 (HenryWin est)

Log Koa (KOAWIN v1.10 estimate): 7.986

Log Koa (experimental database): None

Probability of Rapid Biodegradation (BIOWIN v4.10):

Biowin1 (Linear Model) : -0.1661

Biowin2 (Non-Linear Model) : 0.0002

Expert Survey Biodegradation Results:

Biowin3 (Ultimate Survey Model): 1.4885 (recalcitrant)

Biowin4 (Primary Survey Model) : 2.6937 (weeks-months)

MITI Biodegradation Probability:

Biowin5 (MITI Linear Model) : 0.1046

Biowin6 (MITI Non-Linear Model): 0.0049

Anaerobic Biodegradation Probability:

Biowin7 (Anaerobic Linear Model): -1.0768

Ready Biodegradability Prediction: NO

Hydrocarbon Biodegradation (BioHCwin v1.01):

Structure incompatible with current estimation method!

Sorption to aerosols (25 Dec C)[AEROWIN v1.00]:

Vapor pressure (liquid/subcooled): 0.292 Pa (0.00219 mm Hg)

Log Koa (Koawin est ): 7.986

Kp (particle/gas partition coef. (m3/ug)):

Mackay model : 1.03E-005

Octanol/air (Koa) model: 2.38E-005

Fraction sorbed to airborne particulates (phi):

Junge-Pankow model : 0.000371

Mackay model : 0.000821

Octanol/air (Koa) model: 0.0019

Atmospheric Oxidation (25 deg C) [AopWin v1.92]:

Hydroxyl Radicals Reaction:

OVERALL OH Rate Constant = 1.0945 E-12 cm3/molecule-sec

Half-Life = 9.773 Days (12-hr day; 1.5E6 OH/cm3)

Half-Life = 117.274 Hrs

Ozone Reaction:

No Ozone Reaction Estimation

Fraction sorbed to airborne particulates (phi):

0.000596 (Junge-Pankow, Mackay avg)

0.0019 (Koa method)

Note: the sorbed fraction may be resistant to atmospheric oxidation

Soil Adsorption Coefficient (KOCWIN v2.00):

Koc : 2474 L/kg (MCI method)

Log Koc: 3.393 (MCI method)

Koc : 1.38E+004 L/kg (Kow method)

Log Koc: 4.140 (Kow method)

Aqueous Base/Acid-Catalyzed Hydrolysis (25 deg C) [HYDROWIN v2.00]:

Rate constants can NOT be estimated for this structure!

Bioaccumulation Estimates (BCFBAF v3.01):

Log BCF from regression-based method = 3.263 (BCF = 1832 L/kg wet-wt)

Log Biotransformation Half-life (HL) = 1.8390 days (HL = 69.03 days)

Log BCF Arnot-Gobas method (upper trophic) = 4.137 (BCF = 1.371e+004)

Log BAF Arnot-Gobas method (upper trophic) = 5.619 (BAF = 4.155e+005)

log Kow used: 5.45 (expkow database)

Volatilization from Water:

Henry LC: 0.00194 atm-m3/mole (estimated by Group SAR Method)

Half-Life from Model River: 2.214 hours

Half-Life from Model Lake : 164.6 hours (6.856 days)

Removal In Wastewater Treatment:

Total removal: 88.89 percent

Total biodegradation: 0.68 percent

Total sludge adsorption: 83.56 percent

Total to Air: 4.65 percent

(using 10000 hr Bio P,A,S)

Level III Fugacity Model:

Mass Amount Half-Life Emissions

(percent) (hr) (kg/hr)

Air 3.39 235 1000

Water 5.94 4.32e+003 1000

Soil 89.3 8.64e+003 1000

Sediment 1.39 3.89e+004 0

Persistence Time: 1.73e+003 hr

....

SMILES : COc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM :

MOL FOR: C7 H3 CL5 O1

MOL WT : 280.37

--------------------------- BIOWIN v4.10 Results ----------------------

Biowin1 (Linear Model Prediction) : Does Not Biodegrade Fast

Biowin2 (Non-Linear Model Prediction): Does Not Biodegrade Fast

Biowin3 (Ultimate Biodegradation Timeframe): Recalcitrant

Biowin4 (Primary Biodegradation Timeframe): Weeks-Months

Biowin5 (MITI Linear Model Prediction) : Not Readily Degradable

Biowin6 (MITI Non-Linear Model Prediction): Not Readily Degradable

Biowin7 (Anaerobic Model Prediction): Does Not Biodegrade Fast

Ready Biodegradability Prediction: NO

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin1 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -0.1824 | -0.9121

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 0.1319 | 0.1319

MolWt| \* | Molecular Weight Parameter | | -0.1335

Const| \* | Equation Constant | | 0.7475

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RESULT | Biowin1 (Linear Biodeg Probability) | -0.1661

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TYPE | NUM | Biowin2 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -2.0155 |-10.0775

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 2.2483 | 2.2483

MolWt| \* | Molecular Weight Parameter | | -3.9812

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RESULT | Biowin2 (Non-Linear Biodeg Probability) | 0.0002

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A Probability Greater Than or Equal to 0.5 indicates --> Biodegrades Fast

A Probability Less Than 0.5 indicates --> Does NOT Biodegrade Fast

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin3 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -0.2066 | -1.0330

Frag | 1 | Aromatic ether [-O-aromatic carbon]| -0.0581 | -0.0581

MolWt| \* | Molecular Weight Parameter | | -0.6196

Const| \* | Equation Constant | | 3.1992

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RESULT | Biowin3 (Survey Model - Ultimate Biodeg) | 1.4885

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TYPE | NUM | Biowin4 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -0.1653 | -0.8267

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 0.0771 | 0.0771

MolWt| \* | Molecular Weight Parameter | | -0.4045

Const| \* | Equation Constant | | 3.8477

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RESULT |Biowin4 (Survey Model - Primary Biodeg)| | 2.6937

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Result Classification: 5.00 -> hours 4.00 -> days 3.00 -> weeks

(Primary & Ultimate) 2.00 -> months 1.00 -> longer

------+-----+--------------------------------------+---------+---------

TYPE | NUM | Biowin5 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | 0.0062 | 0.0309

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 0.1952 | 0.1952

Frag | 1 | Methyl [-CH3] | 0.0004 | 0.0004

MolWt| \* | Molecular Weight Parameter | | -0.8341

Const| \* | Equation Constant | | 0.7121

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RESULT |Biowin5 (MITI Linear Biodeg Probability)| | 0.1046

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TYPE | NUM | Biowin6 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -0.2191 | -1.0957

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 1.3227 | 1.3227

Frag | 1 | Methyl [-CH3] | 0.0194 | 0.0194

MolWt| \* | Molecular Weight Parameter | | -8.0938

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RESULT |Biowin6 (MITI Non-Linear Biodeg Probability)| | 0.0049

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A Probability Greater Than or Equal to 0.5 indicates --> Readily Degradable

A Probability Less Than 0.5 indicates --> NOT Readily Degradable

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TYPE | NUM | Biowin7 FRAGMENT DESCRIPTION | COEFF | VALUE

------+-----+--------------------------------------+---------+---------

Frag | 5 | Aromatic chloride [-CL] | -0.4023 | -2.0114

Frag | 1 | Aromatic ether [-O-aromatic carbon]| 0.1780 | 0.1780

Frag | 1 | Methyl [-CH3] | -0.0796 | -0.0796

Const| \* | Equation Constant | | 0.8361

============+======================================+=========+=========

RESULT | Biowin7 (Anaerobic Linear Biodeg Prob) | -1.0768

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A Probability Greater Than or Equal to 0.5 indicates --> Biodegrades Fast

A Probability Less Than 0.5 indicates --> Does NOT Biodegrade Fast

Ready Biodegradability Prediction: (YES or NO)

----------------------------------------------

Criteria for the YES or NO prediction: If the Biowin3 (ultimate survey model) result is "weeks" or faster (i.e. "days", "days to weeks", or "weeks" AND the Biowin5 (MITI linear model) probability is >= 0.5, then the prediction is YES (readily biodegradable). If this condition is not satisfied, the prediction is NO (not readily biodegradable). This method is based on application of Bayesian analysis to ready biodegradation data (see Help). Biowin5 and 6 also predict ready biodegradability, but for degradation in the OECD301C test only; using data from the Chemicals Evaluation and Research Institute Japan (CERIJ) database.

Water Sol (v1.01 est): 0.24396 mg/L

Compound Being Estimated:

SMILES : COc(c(c(c(c1CL)CL)CL)CL)c1CL

CHEM : pentachloroanisole

MOL FOR: C7 H3 CL5 O1

MOL WT : 280.37

Compound Being Used To Make Estimate:

SMILES : Oc(c(c(c(c1CL)CL)CL)CL)c1CL

CHEM : pentachlorophenol

MOL FOR: C6 H1 CL5 O1

MOL WT : 266.34

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TYPE | NUM | WATER SOLUBILITY FRAGMENT DESCRIPTION | COEFF | VALUE

-------+-----+--------------------------------------------+----------+---------

Frag | 1 | -CH3 [aliphatic carbon] |-0.3213 | -0.3213

Frag | -1 | -OH [hydroxy, aromatic attach] | 1.6578 | -1.6578

Frag | 1 | -O- [oxygen, one aromatic attach] | 0.1980 | 0.1980

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Known Wat Sol of Base Compd: 14 (mg/L)

Known Wat Sol of Base Compd: -4.2793 (moles/L)

Adjustment Difference: -1.781

Experimental Value Adjusted Log Wat Sol: -6.0604 (moles/L)

Experimental Value Adjusted Wat Sol: 0.24396 (mg/L)

EVA Method: 1.54E-007 atm-m3/mole (1.56E-002 Pa-m3/mole)

(HenryWin v3.20 estimate)

====> Compound Being Estimated:

SMILES : COc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM : pentaclhoroanisole

MOL FOR: C7 H3 CL5 O1

MOL WT : 280.37

====> Base Compound Used to Make Estimate:

SMILES : Cc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM : pentachlorophenol

HENRY : 2.870E-006 atm-m3/mole (LWAPC = 3.931)

MOL FOR: C7 H3 CL5

MOL WT : 264.37

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BOND DIFFERENCE DESCRIPTIONS | NUMBER | LWAPC VALUE

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C-Car | -1 | -0.1619

C-O | 1 | 1.0855

Car-O | 1 | 0.3473

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TOTAL LWAPC DIFFERENCE (from Base Compound) ---> 1.2708

Estimated HENRY at 25 deg C = 1.54E-007 atm-m3/mole (LWAPC = 5.201)

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EVA Method: 1.64E-003 atm-m3/mole (1.66E+002 Pa-m3/mole)

(HenryWin v3.20 estimate)

====> Compound Being Estimated:

SMILES : COc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM : pentachloroanisole

MOL FOR: C7 H3 CL5 O1

MOL WT : 280.37

====> Base Compound Used to Make Estimate:

SMILES : Oc1c(CL)c(CL)c(CL)c(CL)c1CL

CHEM : pentachlorophenol

HENRY : 2.880E-006 atm-m3/mole (LWAPC = 3.929)

MOL FOR: C6 H1 CL5 O1

MOL WT : 266.34

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BOND DIFFERENCE DESCRIPTIONS | NUMBER | LWAPC VALUE

----------------------------------------------------+--------+-----------------

Hydrogen to Carbon (aliphatic) Bonds | 3 | -0.3590

Hydrogen to Oxygen Bonds | -1 | -3.2318

C-O | 1 | 1.0855

Car-OH | -1 | -0.5967

Car-O | 1 | 0.3473

----------------------------------------------------+--------+-----------------

TOTAL LWAPC DIFFERENCE (from Base Compound) ---> -2.7547

Estimated HENRY at 25 deg C = 1.64E-003 atm-m3/mole (LWAPC = 1.174)

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