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Chlorophenols and Their Impurities in the Canadian Environment: 1983 Supplement

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CHLOROPHENOLS AND THEIR IMPURITIES IN THE CANADIAN ENVIRONMENT: 1983 SUPPLEMENT

by

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ABSTRACT

Since the publication in 1981 of the Environment Canada report "Chlorophenols and Their Impurities in the Canadian Environment" many changes have occurred in the use patterns of chlorophenols, and many research studies have been undertaken. The current Canadian use patterns for chlorophenols and the associated regulations under the Pest Control Products Act, administered by Agriculture Canada, as well as results of relevant research on the chlorophenols as reported in the literature since 1980, have been documented in this supplement. An annotated historical review of chlorophenols in Canada has also been included.

The earlier Environment Canada review on chlorophenols contained considerable information on polychlorinated dibenzo-p-dioxins and other impurities usually present in chlorophenols. The information presented in this supplement has been restricted to that on chlorophenols since recent reviews have dealt specifically with the polychlorinated dibenzo-p-dioxins and other related impurities.

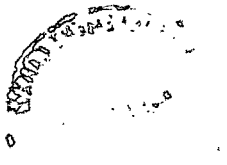
Major subject areas addressed in the chlorophenol supplement include those on: quantities in commerce, sources to the environment, levels and stability in the environment, ecotoxicology, and fate and mobility in the environment.

RÉSUMÉ

Depuis la publication en 1981 du rapport d'Environnement Canada "Les chlorophénols et leurs impuretés dans l'environnement canadien", beaucoup de changements ont eu lieu dans le mode d'utilisation des chlorophénols, et un grand nombre de travaux de recherches ont été entrepris. Le présent supplément examine les diverses applications en cours actuellement dans le secteur des chlorophénols au Canada, avec les règlements connexes dans le cadre de la Loi sur les produits antiparasitaires, administrée par Agriculture Canada, ainsi que les résultats des recherches pertinentes sur les chlorophénols publiés depuis 1980. Le présent document donne également une revue chronologique des événements impliquant les chlorophénols au Canada.

Le premier rapport d'Environnement Canada sur les chlorophénols renfermait un grand nombre de renseignements sur les dibenzo-p-dioxines polychlorées et d'autres impuretés apparentées. Le contenu du présent supplément se limite donc aux chlorophénols, vu que d'autres études récentes traitent plus spécialement des dibenzo-p-dioxines polychlorées et des impuretés apparentées.

Le supplément sur les chlorophénols aborde les principaux sujets suivants: quantités commerciales; voies de pénétration dans l'environnement; concentrations et stabilité dans l'environnement; écotoxicologie; destination finale et mobilité dans l'environnement.



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ABREVIATIONS

Chemicals

(Codes as used in the Pest Control Products Act regulations are listed in Appendix 3.2.3)

DCP	dichlorophenol
2,4-D	2,4-dichlorophenoxyacetic acid
HCB	hexachlorobenzene
m-CP (3-CP)	*m*-chlorophenol (3-chlorophenol)
NaPCP	sodium pentachlorophenate
NaTCP	sodium trichlorophenate
NaTTCP	sodium tetrachlorophenate
o-CP (2-CP)	*o*-chlorophenol (2-chlorophenol)
PCDD	polychlorodibenzo-*p*-dioxin
PCDF	polychlorodibenzofuran
p-CP (4-CP)	*p*-chlorophenol (4-chlorophenol)
PCP	pentachlorophenol
2,4,5-T	2,4,5-trichlorophenoxyacetic acid
TCP	trichlorophenol
TTCP	tetrachlorophenol

Miscellaneous

ai	active ingredient
EC_{50}	Toxic Effect Concentration - 50%
ec (ecd)	electron-capture (detection)
gc	gas chromatography
glc	gas-liquid chromatography
h	hour
hplc	high-pressure liqid chromatography
LC_{50}	lethal concentration - 50%
lc	liquid chromatography
ms	mass spectrometry

N.D.	not detected
ng	nanogram
pg	picogram
ppb	parts per billion (10^{-9})(µg/kg or µg/L)
ppm	parts per million (10^{-6})(mg/kg, mg/L, or µg/g)
ppt	parts per trillion (10^{-12})(ng/kg or ng/L)
t	metric tonne
tlc	thin layer chromatography
µg	microgram
wk	week
yr	year

CONCLUSIONS

The conclusions which follow are based on the information contained in this supplemental report. When they are at variance with the conclusions in Jones (1981), reprinted in Appendix 2, it is so stated.

Uses

1) Agriculture Canada's revised use standards for the chlorophenols (CPs) became effective January 1, 1981. The major uses which remain for tetra- and pentachlorophenol are for long-term wood preservation and for short-term wood protection, as has been the case since 1941 (Chapter 2 and Appendix 3).
2) The primary use for 2,4-dichlorophenol produced in Canada is as an intermediate in the production of phenoxy acid herbicides (Chapter 4).
3) There has been a substantial reduction in the number of CP-containing products registered under the Pest Control Products Act (Appendix 3).
4) The revised use standards still allow registration of products containing 2,4,5-trichlorophenol.

Composition of CPs

5) Free phenols identified in 2,4-DCP also included 2,6-DCP; 2,4,6-TCP; 2-CP; 4-CP; and 2,4-dichloro-6-ethylphenol (Chapter 3).
6) The free phenol contents for the majority of the higher chlorinated CPs used in Canada have not yet been determined.

Quantities in Commerce

7) The quantities of CPs in Canadian commerce for 1981 have been estimated (metric tonnes) as: production (4000 t), imports (>2600 t), exports (1500 t), and total consumption (5300 t) (Chapter 4). These values are higher than the estimates presented in Jones (1981).
8) The quantities of CPs in Canadian commerce are forecast to remain at the same level or to decline slowly for the next several years (Chapter 4).

Residue Analysis

9) The development of improved analytical techniques for CPs in environmental samples has resulted in further definition of fate of CPs in the environment (Chapters 5, 7, and 10).

Sources to the Environment

10) A major source of PCP entering the environment from wood preservation plant sites is wash-off and leaching of PCP from treated poles and timbers by rain water (Chapter 6).
11) The ratio of 2,3,4,6-TTCP to PCP in environmental samples may be used as a chemical marker for PCP derived from specific industrial sources (Chapter 6).
12) Some CPs are found in the environment as a result of the application of phenoxy herbicides but the quantities are minor (Chapter 6).
13) Municipal sewage treatment plant effluents are a continual but secondary source of entry for CPs to the environment (Chapter 6).

Levels and Stability in the Environment

14) TTCPs and PCP have been identified in sediments deposited annually since 1949 in the Bay of Quinte area, Lake Ontario (Chapter 7).
15) Concentrations of TTCPs and PCP in Bay of Quinte surface film and waters decreased with increasing distance from the point source, but PCP concentrations decreased at a faster rate than the TTCPs (Chapter 7).
16) 2,4,5-TCP has been found in biota from the mouth of the Niagara River but has not been detected in biota from Lake Erie (Chapter 7).

Metabolism

17) The uptake and depuration of (^{14}C) 2,4,5-TCP in fathead minnows parallels that for PCP. Rapid uptake was followed by rapid loss (up to 72% loss in 24 h), then a slower second phase in which the half-life of the remaining ^{14}C was 21-28 days (Chapter 8, Sect. 8.1.1).
18) Major post-metabolic residues in soils and plants following exposure to PCP are unextractable bound-residues (Chapter 8, Sect. 8.1.2 and 8.1.3).

Ecotoxicology

19) Although PCP can have a major effect on marine ecosystems, marine bacteria and phytoplankton can adapt to and recover from exposure to subacute concentrations of PCP (Chapter 9, Sect. 9.1.1).
20) Based on laboratory experiments, the "No Observable Effect Level" for PCP in estuarine benthic communities was calculated to fall between 13 and 14 µg PCP/L (Chapter 9, Sect. 9.1.2).

21) Concentrations of NaPCP below 100 µg/L are expected to alter marine copepod community structure (Chapter 9, Sect. 9.1.2).

22) Species balances in freshwater communities can serve as indicators of CP contamination (Chapter 9, Sect. 9.1.3).

23) Biochemical indicators in polychaetes under stress from PCP are depletion of tissue glycogen reserves and increases in tissue ascorbic acid, which are indicative of detoxification and elimination of PCP via the glucuronic acid pathway (Chapter 9, Sect. 9.2.1.1).

24) Biochemical indices in aquatic vertebrates exposed to PCP include those noted in 23), (above) and, additionally, an increase in plasma cortisol concentrations (Chapter 9, Sect. 9.2.1.2).

25) Reduction of photosynthesis has been observed in phytoplankton and aquatic macrophytes exposed to PCP (Chapter 9, Sect. 9.2.1.3 and 9.2.1.4).

26) Histopathological changes have been observed in liver of terrestrial vertebrates chronically exposed to PCP (Chapter 9, Sect. 9.2.2).

27) Based on LC_{50} and EC_{50} test data, marine and freshwater molluscs, worms, crustaceans, fish and algae are equally sensitive to PCP. The majority of the LC_{50} and EC_{50} values for these organisms fall in the range 0.1-1 mg PCP/L. Effects have been noted at lower PCP concentrations for the most sensitive species and life stages (Chapter 9, Sect. 9.3.1).

28) Toxicity of CPs to aquatic organisms can be modified by pH and temperature of the water and by the presence of sediments (Chapter 9, Sect. 9.3.1).

29) In aquatic biota resistance to infection is decreased by exposure to PCP (Chapter 9, Sect. 9.4.1).

30) No PCP-derived immune deficiency has been observed in cattle with blood levels of 12.5 ppm PCP (Chapter 9, Sect. 9.4.2).

31) CPs generated from the aqueous chlorination of unbleached kraft pulp are not mutagenic (Chapter 9, Sect. 9.5).

32) 2,4,6-TCP may be teratogenic in certain species of freshwater fish (Chapter 9, Sect. 9.6).

Chemodynamics - Fate and Mobility

33) In a natural environment, where some surface water might be close to neutral pH balance, there is little loss of PCP via evaporation (Chapter 10, Sect. 10.1.1). This

conclusion abrogates Conclusion 53 (Jones, 1981), which is reprinted in Appendix 2, page 70, of this report.

34) Bioconcentration factors for PCP will vary according to physical characteristics, including pH and ionic strength, of the media (Chapter 10, Sect. 10.2).

35) In a natural aquatic environment, such as the Bay of Quinte, the bioaccumulation of PCP and TTCP can vary from moderate in leeches (10^2) to high (approximately 10^4) in fish (Chapter 10, Sect. 10.2).

36) CPs are reactive and degrade in the environment at specific rates for given conditions and through complex mechanisms (Chapter 10, Sect. 10.3).

37) Biological degradation in an aquatic environment is enhanced by the presence of acclimated bacteria (Chapter 10, Sect. 10.3.1.1).

38) In either aquatic or terrestrial environments anaerobic conditions greatly retard degradation of CPs (Chapter 10, Sect. 10.3.1.1 and 10.3.1.2). Residues of PCP and TTCP have persisted for decades in anaerobic sediments (Chapter 10, Sect. 10.4).

39) A significant pathway for the removal of ionized TTCPs and PCP from an aquatic environment is loss through photolytic action in the surface film (Chapter 10, Sect. 10.3.2).

40) CPs become bound to humic material through chemical, physical, and biological mechanisms (Chapter 10, Sect. 10.4).

Waste Management

41) Granular activated carbon adsorption treatments have been used successfully in conjunction with biological treatments to reduce concentrations of CPs in effluents to insignificant levels (Chapter 11, Sect. 11.1).

1 INTRODUCTION

The Environment Canada technical review on "Chlorophenols and Their Impurities in the Canadian Environment" (Jones, 1981) was completed in February 1981, and released to the public on June 4, 1981. A list of corrections are found in Appendix 1. The literature from which the report was compiled was primarily that which was available through 1979. Since 1979 there have been a considerable number of reviews, scientific papers and government documents on chlorophenols in the environment (Ahlborg and Thunberg, 1980; Callahan et al., 1979; Crosby, 1980, 1982; National Research Council of Canada, 1982; United States Environmental Protection Agency, 1979, 1981a, 1981b). This "new" information on chlorophenols has been reviewed and, if relevant, extracted, summarized and incorporated in this 1983 supplement to the Environment Canada review on chlorophenols. The information was classed as relevant if it had an impact on any of the conclusions itemized in Jones (1981). For example, if a report provided evidence to strengthen or to disprove a conclusion, this new information has been incorporated and referenced in this supplement.

The order of presentation for the subject matter in this supplement follows in general that in the Jones (1981) report although only those subject areas with "new" information have been included. Since reference has been made to the Conclusions in Jones (1981), they have been reprinted as Appendix 2.

The most noticeable change in the subject matter covered in this supplement from that in the earlier Environment Canada review (Jones, 1981) has been the deletion of specific references to the polychlorinated dibenzo-ρ-dioxins (PCDDs), the polychlorinated dibenzofurans (PCDFs) and hexachlorobenzene (HCB). These are the major neutral impurities in the chlorophenols. The environmental and health problems associated with the PCDDs and the PCDFs are now matters of concern in their own right. They have been addressed in criteria documents by the Environmental Secretariat of the National Research Council of Canada (NRCC, 1981a; NRCC, 1981b). Furthermore, a Department of the Environment/National Health and Welfare Ministers' Advisory Committee on Dioxins, comprised of persons from outside government, has been formed and a Committee report will be forthcoming in 1983. Therefore it was concluded that it would be redundant to include information on dioxins in the Canadian context in this supplement.

The environmental and health concerns of HCB in Canada were documented in three unpublished reports prepared for the Department of the Environment/National Health and Welfare (DOE/NH&W) Environmental Contaminants Committee in 1979.

These reports have been collected under one cover as an unpublished manuscript report which was made available to interested parties in 1982 by the Contaminants Control Branch, Environmental Protection Service, Environment Canada. The unpublished report will be supplanted by means of a major revision and expansion of the subject areas in a joint report from Environment Canada and the Department of National Health and Welfare. The revision is under preparation.

The Environment Canada report on chlorophenols (Jones, 1981), plus this supplement and a major companion document from National Health and Welfare titled "Chlorophenols and Their Impurities: A Health Hazard Evaluation" (Gilman et al., 1983) along with the National Research Council of Canada (1982) publication have documented the chlorophenol situation in Canada through 1982.

Following the publication in 1981 of the Environment Canada report "Chlorophenols and Their Impurities in the Canadian Environment" many changes have occurred in the use patterns of chlorophenols, and many research studies have been undertaken. A large portion of the knowledge gaps and uncertainties noted in the conclusions of the earlier report, and the recommendations that were based on them, have been clarified. No need for further restrictions in the uses of chlorophenols is now seen, provided that the codes of good practice now under development are implemented to reduce needless losses of chlorophenols into the environment.

2 USES

Agriculture Canada announced on Nov. 28, 1980, in Memorandum T-1-229 (Jones, 1981), a revision in the use standards for chlorophenols. The following suspensions became effective January 1, 1981:

1) Suspension of chlorophenol (CP) products carrying label instructions for use as wood preservatives and/or wood stains for INTERIOR home use;
2) Suspension of products containing sodium pentachlorophenate (SPC) for use as fungicides in mushroom houses and on tools for mushroom culture;
3) Suspension of products containing pentachlorophenol (PCP) for use as wood preservatives on wooden food containers and on horticultural lumber, e.g. seed flats, stakes, greenhouse lumber, etc;
4) Suspension of products containing PCP and carrying label instructions for use as wood preservatives on above-ground interior woodwork of farm buildings, e.g. dry locations such as walls, floors, bins, feed troughs, silos, stalls, chicken roosts, etc. It is recommended that PCP-treated wood be used on the farm only where the wood is in ground contact, e.g. fence posts, support poles, foundation supports, and the bottom six inches of stall skirt boards;
5) Suspension of all CP products carrying label instructions for use as microbiocides in curing hides;
6) Suspension of CP products labelled for use as herbicides and soil sterilants, except those labelled for destruction of moss on roofs;
7) Suspension of products containing the chlorophenols and their sodium salts for use as slimicides in pulp and paper mill operations;
8) Suspension of all DOMESTIC class products for application by SPRAY methods.

The revised use standards are reflected in the reduced number of CP products available in the Canadian domestic and industrial market (Appendix 3). Major uses which remain for the CPs include long-term wood preservation and short-term wood protection.

3 COMPOSITION OF CHLOROPHENOLS

As noted in Jones (1981) the production of CPs is not an isomer specific process, so that when 2,4-DCP is produced there is usually 6-7% of 2,6-DCP formed at the same time. Cochrane et al. (1983) analyzed ten 2,4-DCP samples, obtained from manufacturers, for chlorinated phenolic impurities. The percentage phenol content (range and mean) of the samples were 2,4-DCP (83.90-98.30, 92.24), 2,6-DCP (1.40-9.18, 4.48), 2,4,6-TCP (0.53-3.23, 1.24), 2-CP (0.10-3.52, 1.09), 4-CP (0.05-1.19, 0.46), and 2,4-dichloro-6-methylphenol (N.D. - 0.34, 0.07). 3-CP was not detected in any sample of 2,4-DCP. The primary use for 2,4-DCP in Canada is for production of phenoxy herbicides. As a result, small amounts of the free phenols originally in the 2,4-DCP are likely to be present in the 2,4-D acids, together with the acids derived from these free phenols, and thus would be available for direct entry into the environment. Section 6, titled "Sources to the Environment" has relevant information developed by Cochrane et al. (1983) on the free phenol content of 2,4-D acids.

Following the program to identify the chlorinated phenolic impurities in technical 2,4-DCP used in Canada, Agriculture Canada gave priority to a similar program for technical PCP and NaPCP. In 14 samples of technical PCP from five suppliers, Lanouette and Cochrane (1983) identified two isomers of DCP, three isomers of TCP, and three isomers of TTCP. Based on percent content, the major impurities were 2,4-DCP; 2,3,5,6-TTCP and 2,3,4,6-TTCP. In three samples of NaPCP from one supplier, the only major impurity was 2,3,4,6-TTCP.

4 QUANTITIES IN COMMERCE

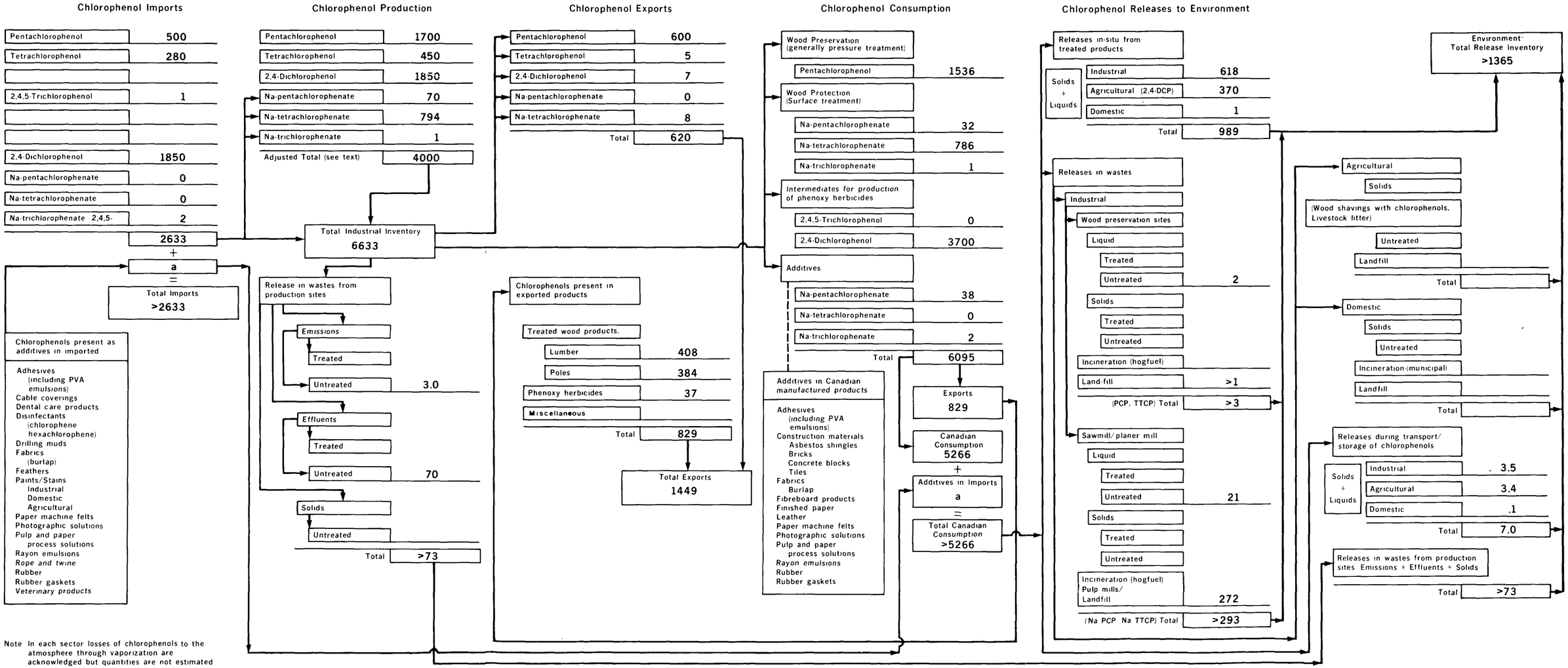
One of the major information gaps identified in the Jones (1981) report was the lack of information on quantities of chlorophenols in Canadian commerce. A materials balance flow chart for chlorophenols in Canada has been constructed (Figure 1). The information for this has been drawn, in part, from reports from Statistics Canada and Agriculture Canada, and consultations with the Council of Forest Industries of British Columbia. Another major source of information has been the CPI Product Profiles published by Corpus Information Services Ltd., Don Mills, Ontario. An additional guide for estimation of Canadian consumption of CPs by the various industrial sectors was the Lamb, Guay, Inc. (1979) report. Hand-in-hand with consumption are releases to the environment. Since there is still very little information available on quantities of CPs released to the environment from emissions and effluents and other losses during manufacturing, transport, storage and uses of CPs, estimates of such releases, for the purposes of this supplement, specifically in Figure 1, were based on information in a U.S. EPA report titled "Materials Balance for Chlorophenols: Level I - Preliminary" (Hall et al., 1980).

The quantities of CPs in Canadian commerce (Figure 1) were estimated for 1981, which was just prior to the decline in the Canadian forest economy and the probable concomitant decrease in consumption of CPs. In Figure 1 not all of the estimated quantities of CPs in any one sector of commerce are additive. For example, the estimated quantities of PCP, TTCP and 2,4-DCP produced in Canada total 4000 t, but from that total are subtracted the quantities of PCP and TTCP which are utilized for production of the 864 t of sodium salts of these CPs. Also, it is obvious that there are a considerable number of unkowns, such as the amount of CPs present as additives in both imported and Canadian manufactured products.

The estimated quantities of CPs in Canadian commerce (Figure 1) may well be maximum quantities since Lamb, Guay, Inc. (1979) had forecast that the consumption of CPs for treatment of lumber and timber will decline over the next few years in favour of treatments utilizing the water-borne salts, chromated copper arsenate (CCA) and ammoniacal copper arsenate (ACA). In addition to the forecast decline in use of CPs in wood preservation, there may be a parallel decline in use of CPs for wood protection, that is, protection of wood from sapstain organisms, as a result of the anticipated development of replacement products for the CPs.

In an attempt to obtain further information in this area the DOE/NH&W Environmental Contaminants Committee recommended on August 9, 1979, that commercial flow information for the CPs should be collected. In support of this recommendation, the Chemical Data Division, Contaminants Control Branch, Environmental Protection Service, issued five notices in 1980 under authority of paragraph 4(1)b of the Environmental Contaminants Act (ECA) to Canadian manufacturers, suppliers, and formulators of CPs, and to those companies with CP-containing products registered under the Pest Control Products Act. The information collected concerned the quantities of CPs and their sodium salts in Canadian commerce for the years 1977-79. Some of the information submitted by the respondents was identified by them as being given in confidence and, therefore, since that information cannot be disclosed except as may be necessary for purposes of the Act, it was not used in the compilation of this report.

FIGURE 1 MATERIALS BALANCE FOR CHLOROPHENOLS IN CANADA FOR 1981
(estimates in metric tonnes (t) of technical material)



Note In each sector losses of chlorophenols to the atmosphere through vaporization are acknowledged but quantities are not estimated

TOTAL IMPORTS· >2633 t

TOTAL PRODUCTION· 4000 t

TOTAL EXPORTS· 1449 t

TOTAL CONSUMPTION· >5266 t

TOTAL RELEASES· >1365 t

5 RESIDUE ANALYSES

CP analytical methodology for a variety of environmental media was reviewed in Jones (1981). In general, most of these methods involve pH adjustment to convert CPs to their non-ionized forms, extraction into an organic solvent, clean-up by back-extraction into aqueous base and derivatization, usually by acetylation, prior to analysis by gc-ecd (Fox, 1978; Rudling, 1970; Chau and Coburn, 1974). Some of the more recently-published analytical methods for CPs are briefly reviewed below and are summarized in Table 1.

Recent improvements in analytical methodology include the isolation and concentration of CPs in hydrolyzed urine by means of a macroreticular resin, followed by direct gc-ecd on polar support-bonded columns (Edgerton et al., 1980), and isolation and concentration from water samples by an adsorption/desorption method which employs a reversed phase octadecyl-modified silica gel cartridge, followed by aqueous acetylation and quartz capillary gc-ecd (Renberg and Lindstrom, 1981). Several papers have recently been published on detection systems other than gc-ecd. These include gc-ms (Ingram et al., 1979; Eiceman et al., 1979), hplc equipped with spectrophotometric detectors (Mundy and Machin, 1981; Ugland et al., 1981) and gc equipped with Coulson conductivity (Bristol et al., 1982) or flame ionization detectors (van Rossum and Webb, 1978). Since several of the above reports have described difficulties in total isomeric separation, it is of interest that Ugland et al. (1981) were able to separate all 18 CP isomers with hplc on a C_{18} column.

Lamour (1982) has published a method for the determination of PCP in fuel oil as used in the wood preservation industry, i.e., fuel oil containing 1.7 to 4% PCP by weight. This method involves clean-up on a silica gel absorption column, followed by gc analysis of the PCP fraction on an NPGS plus phosphoric acid column or of the methylated PCP fraction on a QF-1 column.

Schonhaber et al. (1982) compared two methods, hplc and glc, for the determination of PCP in mushrooms. Results of the analyses by both methods were in good agreement. Steam distillation of the acidified fraction was used to separate PCP from interfering plant compounds in the mushrooms. For the hplc system of Schonhaber et al. (1982) a detection limit of 0.5 µg PCP/kg (fresh weight basis) of mushrooms was obtained by use of an eluant with low UV absorption and a detection wavelength of 220 nm. A similar detection limit was found for the gc determination of PCP as its acetate.

TABLE 1 ANALYTICAL METHODOLOGY FOR CHLOROPHENOLS

Matrix	Compounds	Method	Reference
Water	PCP	Isotope dilution; gc-ms of methylated sample.	Ingram, L.L., Jr., et al. 1979.
Water	CPs	Solvent extraction; gc-ecd of acetates.	Wegman, R.C.C., and A.W.M. Hofstee. 1979.
Water	CPs	Ring-oven colorimetric technique.	Buckman, N.G., et al. 1983.
Water: Sea water & wastewater	CPs	adsorption/desorption; gc-ecd of acetates with quartz capillary column.	Renberg, L., and K. Lindstrom. 1981.
Water & wastewater	PCP	Solvent extraction; gc-ecd of acetate.	Environ. Canada, Water Quality Branch, Methods Manual. 1979.
Pulp mill effluent	CPs	*in situ* acetylation; solvent extraction; gc-ecd of acetates.	LaFleur, L., et al. 1981.
Suspended matter & sediment	CPs	solvent extraction; gc-ecd of acetates.	Eder, G., and K. Weber. 1980.
Fly ash	PCP TTCPs TCPs	solvent extraction; gc-ecd and gc-ms.	Eiceman, G.A., et al. 1979.
Urine	CPs	adsorption/desorption on XAD-4; direct gc-ecd analysis.	Edgerton, T.R., et al. 1980.
Tissue, eggs & serum	PCP TTCPs	extraction, adsorption and desorption; gc analysis with Coulson conductivity detector.	Mundy, D.E., and A.F. Machin. 1981.
Potatoes: tubers & vines	2,4-DCP & 2,4-D	extraction, adsorption and desorption; gc analysis with Coulson conductivity detector.	Bristol, D.W., et al. 1982.
Commercial PCP, biological tissues	PCP TTCPs	steam distillation; extraction; analysis by negative chemical ionization ms.	Kuehl, D.W., and R.C. Dougherty. 1980.

Recently, Butte et al. (1983) described a method for determination of PCP and TTCPs in samples of sediments and clams from marine mud flats which had received effluent containing PCP from a paper mill from 1965 to 1978. Previously freeze-dried samples of sediments and clams were extracted with toluene under acidic conditions and then the CPs were back extracted into a methanol/water solution of triethylsulfonium hydroxide. When the methanol/water phase is injected into a gc, a pyrolytic ethylation occurs with the formation of ethyl ethers of PCP and TTCPs which are then separated in quartz capillary columns and detected by an electron capture detector. 2,4,6-Tribromophenol was used as an internal standard. Recoveries were calculated on a 2,3,5,6-TTCP basis since 2,3,4,6- and 2,3,5,6-TTCPs were not separated by this system. Recoveries for PCP and TTCPs ranged from 70 to 90% or from 80 to 100% – the higher recoveries were obtained when acidic hydrolysis of the samples was performed prior to the toluene extraction. The method used by Butte et al. (1983) did not require any evaporation or chromatographic clean-up steps. The authors noted that 2,3,4,5- and 2,3,5,6-TTCPs may be formed under anaerobic conditions from PCP and that 2,3,4,6-TTCP is a technical impurity in PCP. Levels of 2,3,4,5-TTCP in sediments ranged from 23-32 µg/kg and in clams from 0.5-46 mg/kg (both on a dry weight basis) but 2,3,5,6/2,3,4,6-TTCPs did not exceed the detection limits (approximately 500 ng/kg and 25 µg/kg for the CPs in the sediments and clams, respectively).

Fox and Joshi (1983) analyzed with a gc-ecd procedure CPs in environmental samples taken in 1978 in the Bay of Quinte area in eastern Ontario. The PCP, used at a wood preserving plant which was a point source for CPs in their study area, contained TTCPs as impurities. The ratio of TTCP to PCP in the technical PCP used at the plant was nominally 0.12. With the gc-ecd system and the packed column routinely used, Fox and Joshi (1983) could not separate 2,3,4,6-TTCP from 2,3,5,6-TTCP. Analysis by high resolution capillary gc, however, showed that 2,3,4,6-TTCP usually comprised more than 80% of the total TTCP.

Warren et al. (1982) devised and validated a procedure for sampling of PCP and TTCPs in air by employing silica gel collection tubes, desorption with benzene and direct analysis by gc-ecd. With each of four different techniques the authors were able to demonstrate collection and desorption efficiencies of at least 90% for PCP.

An analytical procedure was developed by Fullerton et al. (1982) to monitor for PCP in hardwood chips and cardboard, since these wood products were used as litter for both laboratory animals and livestock. PCP was isolated via liquid-liquid partitioning steps. Pentachlorophenyl acetate was formed by acetylation using pyridine and acetic

anhydride and derivatized material was partitioned into hexane for gc-ecd analysis. Average recoveries from hardwood chips spiked at five levels with PCP were 83% with a standard deviation of ± 6%. Results of analyses for PCP in 86 hardwood chip samples obtained commercially between 1977 and 1981, and intended for bedding, had residue levels that ranged from < 5 to 240 ppb of PCP.

An analytical system developed by Lee et al. (1984) for PCP in tallow has overcome interference problems associated with this sample type. Utilization of this new system has resulted in essentially complete recoveries and has lowered the detection limit for PCP in this matrix (by a factor of 10) to 1 ppb. The method utilizes automated gel permeation chromatography (gpc). Appropriate gpc fractions are derivatized with diazomethane followed by a final Florisil clean-up prior to gc-ecd analysis.

Cochrane et al. (1983) analyzed 2,4-D and 2,4-DCP samples for the presence of chlorinated phenolic impurities by utilizing hplc with an electrochemical detector which was 5 to 15 times more sensitive, depending on the CP, than a UV detector. Similarly, hplc with an electrochemical (coulometric mode) detector was 10 to 50 times more sensitive than a UV detector for quantitation of chlorinated phenolic impurities in technical PCP (Lanouette and Cochrane, 1984).

6 SOURCES TO THE ENVIRONMENT

CPs can enter the environment as a result of their agricultural, industrial and domestic uses, or they can be generated in the environment through various routes. Some of the possible routes identified by Crosby (1982) were metabolism of common pesticides, such as hexachlorobenzene and pentachloronitrobenzene, and chlorination during water treatment.

As noted, a source of CPs to the environment is the application of agricultural pesticides which form CPs during their degradation or contain free CPs as impurities. The 2,4-D based phenoxy herbicides are an example of this.

Cochrane et al. (1983) analyzed 13 samples of technical 2,4-D acid, obtained from manufacturers, for free phenols. The free phenols (% range, mean) found were 2,4-DCP (0.004-1.45, 0.19), 2,6-DCP (0.001-0.048, 0.01), 2,4,6-TCP (0.001-0.14, 0.02), 2-CP (0.0004-0.004, 0.001), and 4-CP (0.0004-0.005, 0.001). Two free phenols looked for but not detected were 3-CP and 2,4-dichloro-6-methylphenol. The CPs identified in Swedish formulations of the phenoxy herbicides, 2,4-D, MCPA, dichlorprop, and mecoprop included 2-CP, 4-CP, 2,4-DCP, 2,6-DCP, 2,4,6-TCP and 2-methyl-4-CP and constituted approximately 1% of the herbicide formulations (Akerblom and Lindgren, 1983).

A study was conducted in Wood Lake and Kalamalka Lake in the Okanagan Valley in British Columbia in 1979 to determine the persistence and transport of residues of 2,4-dichlorophenoxyacetic acid (2,4-D), 2,4-D butoxyethanol ester (2,4-D BEE) and 2,4-DCP (Bothwell and Daley, 1981). 2,4-D BEE on bentonite granules had been applied to the lakes, initially in 1976 and then on a large scale in 1977 and 1978, as a control measure for Eurasian water milfoil. The researchers demonstrated in 1979 in Wood Lake that the detectable presence of 2,4-DCP, a degradation product of 2,4-D BEE, in the lake sediments at a depth of 0-2 cm was limited to a period of <77 d with highest concentrations of 2,4-DCP (≈ 0.7 ug/g) occurring between 11 and 19 days after treatment. The 2,4-D BEE had been applied at the rate of 22-45 kg of 20% granules/ha, depending on water depth. The lake water was ≈ pH 8.0. Sediment analysis for 2,4-DCP consisted of steam extraction followed by direct injection into a gc without clean-up. Recoveries from spiked samples were 75-76% (coefficient of variation of 2.9%).

Further experimental evidence for the presence of limited amounts of 2,4-DCP in environmental samples following the application of 2,4-D phenoxy hebicides has been provided by researchers at the National Water Research Institute. Scott et al. (1982) treated outdoor ponds, with surface dimensions of 15 x 7.5 m and a depth of 2.5 m.

with ester and amine formulations of 2,4-D to provide an initial concentration of approximately 1.5 mg 2,4-D/L. Using a newly developed simultaneous extraction method, and analysis of water, sediment and particulate samples by gc-ecd, they demonstrated with nearly quantitative recoveries, the presence of 2,4-DCP at concentrations near detection limits, i.e., trace to low ppb, in the samples at up to 125 days after treatment.

It was concluded from these test results that the quantity of 2,4-DCP released to an aquatic environment as a result of the application of a 2,4-D phenoxy herbicide would be inconsequential and would not be an environmental concern.

To assist in the identification of possible industrial sources of CPs to the environment a survey for CPs at selected plant sites was undertaken in New Brunswick and Nova Scotia in 1980 (MacKnight and LeBlanc, 1981). Results of analyses for specific CPs in the samples of plant final effluents, lagoons, and sewers are presented in Table 2.

Wood preservation plants are users of large quantities of PCP (Figure 1). Levels of PCP in effluent from these plants are not a good indicator of quantities of PCP entering the environment from these point sources relative to other industries which use CPs or generate CPs (Table 2). Fox and Joshi (1983), following a survey for CPs in the aquatic environment downstream from a wood treatment plant, identified the primary source of CPs as fugitive releases from the initial weathering of PCP from treated poles and timbers in storage yards, and not that from the physical plant effluent.

Paasivirta et al. (1980) demonstrated that the CPs and related compounds identified in sediments and food chain biota from the lakes of central Finland sampled in 1978 were derived from residues from pulp chlorobleaching and from wood preservation wastes. The authors suggested that of the six compounds generally found, 2,4,6-TCP, 4,5,6,-trichloroguaiacol, and tetrachloroguaiacol originated from pulp mills while 2,3,4,6-TTCP, PCP, and tetrachlorocatechol were commonly derived from wood preservation operations, and that each of these compounds was both mobile and persistent in the environment. As an example of the levels of CPs in the 0-2 cm depth of sediment, at a sampling site 5 km downstream from a pulp mill, 2,4,6-TCP, 2,3,4,6-TTCP, and PCP were found in five samples at 27.7 ± 17.2 (SD), 50.1 ± 17.5 (SD), and 9.48 ± 3.02 (SD) ng/g (dry weight), respectively.

As a follow-up to the sampling program of 1978, Paasivirta et al. (1983) resampled sediment and biota from the same locations in 1980 and 1981. Residues of 2,4,6-TCP and chlorinated guaiacols, which had been identified in the 1978 samples of pike, *Esox lucius*, taken 5 km downstream from a pulp mill, were below the level of quantification (0.2 ng/g wet weight) in pike samples in 1981 from the same locations. The

TABLE 2 LEVELS (µg/L) OF CHLOROPHENOLS IN INDUSTRIAL EFFLUENTS AND LAGOONS IN NEW BRUNSWICK AND NOVA SCOTIA, 1980

Industrial Sector	s*	n**	Chlorophenols							
			2,4-DCP		2,4,6-TCP		2,3,4,6-TTCP		PCP	
			N***	ML (µg/L)	N***	ML (µg/L)	N***	ML (µg/L)	N***	ML (µg/L)
Oil refineries	4	5	4	100.3	2	1.09	1	0.89	2	0.51
Pulp and paper industries	5	8	4	5.59	2	147.8	2	37.2	7	5.92
Textile mill	1	1	1	1.72	0		0		0	
Wood preservation plant	1	1	0		0		1	0.11	1	0.28
Oil-powered thermal power generators	5	8	4	16.12	1	0.48	1	0.16	4	0.87
Coal washing facilities	3	3	3	3.53	0		0		2	0.79
Coal mines	2	2	1	1.40	0		0		1	1.03
Coking oven	1	1	1	3.44	1	3.28	1	1.0	1	1.70
Carpet manufacturer	1	1	1	5.30	1	4.65	1	1.25	1	2.88
Lead smelter	1	1	0		0		0		0	
Chemical plants	2	3	1	3.67	2	6.04	2	0.66	2	1.73
Tire manufacturer	1	1	1	90.98	1	4.32	1	0.54	1	1.13

*: no. of sites; **: no. of samples; ***: no. of samples with a CP level >2 X the minimum detection limit (MDL); ML = maximum level (µg/L) of CP detected.

Note: Percent recovery and MDL in µg/L for the equivalent derivatized chlorinated anisoles were as follows: 2,4-DCP, 70/0.10; 2,4,6-TCP, 79/0.08; 2,3,4,6-TTCP, 84/0.05; PCP, 90/0.07.

Data extracted from MacKnight and LeBlanc (1981).

decrease was attributed to: 1) more use of hypochlorite in the chlorobleaching process, and 2) an increased flow of water passing the pulp mill effluent discharge point. Quantifiable residues of 2,3,4,6-TTCP and PCP, both associated with wood preservation, were present in pike sampled in all 3 years.

The probablility of release of CPs to the environment through their use in the Canadian pulp and paper industry was substantially reduced with the suspension of products containing CPs for use as slimicides in pulp and paper mill operations (Agriculture Canada Trade Memorandum T-1-229. (Jones, 1981. App. 10, Annex 2)). Although its use is minor, NaPCP is still available to the pulp and paper industry as a preservative for pulp mill processing materials, in paper machine felts, and to protect finished paper and fiberboard products (Jones, 1981). Nitka et al. (1982) have pointed out that if CPs were present in the acidic process pulp stream they would be sorbed on the unbleached wood pulp fiber, but their studies showed that CPs would be promptly desorbed from the fibers when the pH of a process stream was raised to pH 8-9 upon entry into a waste treatment system. In a process stream that was neutralized to pH 7, the quantity of CPs released from the pulp fibers would probably be proportionately lower.

Municipal sewage treatment plants (STPs) have been and remain a secondary source for CP entry into the environment. In 1978, samples of final effluent from STPs located in the Toronto-Hamilton-Waterloo/Kitchener triangle were shown to contain low ppb levels of PCP (Jones, 1981). More recently, samples of primary effluent and sludge collected in 1979-81 from STPs located in British Columbia, Manitoba, the St. Clair River area of Ontario, and in Nova Scotia have all shown low (ppb) levels of 2-CP, 2,4-DCP, 2,4,6-TCP and PCP (Environment Canada, 1982).

A difficult question to resolve has been whether or not the PCP in environmental samples originated from commercial PCP or from degradation of other compounds such as hexachlorobenzene (HCB). Kuehl and Dougherty (1980) used negative chemical ionization (nci) mass spectrometry to analyze for CPs in environmental samples. They concluded from the results of their analyses that the presence of TTCP in a particular ratio with PCP in environmental samples may serve as a chemical marker for PCP derived from commercial sources, since TTCP is usually present as a CP impurity in commercial PCP.

7 LEVELS AND STABILITY IN THE ENVIRONMENT

A comprehensive review of levels of CPs in the Canadian environment as presented by Jones (1981) demonstrated that CPs and PCP in particular were ubiquitous in the environment. Additional and more recent data on levels of CPs in environmental samples are now presented.

Fox (1983) measured PCP in rain collected near the Wastewater Technology Centre at the Canada Centre for Inland Waters, Burlington, Ontario, in 1982 and found up to 10 ng PCP/L of precipitation.

In Ontario in 1980, 10 lake trout (872-2356 g) from the Eastern basin of Lake Ontario (Main Duck Is.) and 11 lake trout (1543-3226 g) from the Western basin (Port Credit) were analyzed for PCP. PCP was identified above a detection limit of 0.5 µg/kg in five trout, all from the Western basin, at levels of 2, 3, 5, 10 and 11 µg/kg (wet weight) (Niimi,1982).

Fox and Joshi (1983) analyzed for CPs in environmental samples, including surface film, whole water, surficial sediments, sediments, and biota, from the 87-km long Bay of Quinte which has a wood preserving plant site on the Trent River at the head of the Bay as a point source for CPs. Reported concentrations of PCP and TTCPs (2,3,4,6- and 2,3,5,6-) in the surface film and water were in the low ppb range (1-6 ppb) in samples adjacent to the point source with erratic decreasing concentrations with increasing distance from the point source. PCP concentrations decreased at a faster rate than the TTCPs as distance from the source increased thereby increasing the ratio of TTCPs to PCP. The authors noted that the ratio of TTCPs to PCP at the source, 0.2:1, was close to the ratio of these in the commercial product used at the wood preserving plant, nominally 0.12:1. Biota from one site in the study area, approximately 25 km from the point source, had concentrations of 1-260 ppb PCP, and N.D. to 40 ppb TTCPs (2,3,4,6- plus 2,3,5,6-). Sectioned cores of sediments indicated to the authors that PCP and TTCPs accumulated by settling of particles to which CPs are adsorbed. Fox and Joshi (1983) demonstrated that the ratio of PCP to TTCPs in the sediments, over a measured period of 29 years of sediment deposition, was remarkably constant, although there was a noticeable decreasing concentration of CPs with increasing depth of sediment. They suggested that the CP decline with depth may indicate slow degradation under anaerobic conditions and that the relatively constant PCP/TTCP ratio suggested increased loading over the years of operation of the wood treatment plant.

A 1981 Canada-Ontario Review Board Report on the Niagara River included information on the levels of CPs in 20 young-of-the-year Spottail shiners collected in 1979 at Niagara-on-the-Lake, Lake Ontario, and at Centre Creek, Lake Erie. PCP and 2,4,6-TCP were identified in the samples from both locations at maximum levels of 28 and 33 ng/g (wet weight, whole fish), respectively, but 2,4,5-TCP, quantified at a maximum level of 22 ng/g, was identified only in the Spottail shiners from Niagara-on-the-Lake.

At Niagara Falls, New York, DCP and TCP were identified in surface waters and sediment which had originated at chemical dump sites adjacent to the Niagara River near Grand Island. Maximum levels for total DCP and total TCP in sediments were 2 ppm and 5 ppm, respectively (Elder et al., 1981).

In the southern U.S., Murray et al. (1981) analyzed for PCP in samples of water, sediment and biota from San Luis Pass, an estuary in the Galveston Bay area of Texas. Levels of PCP in the water samples ranged up to 11 ng/L; in sediments, levels up to 0.26 ng PCP/g were found on a dry weight basis. Levels of PCP, up to 17 ng/g (wet weight), were quantified in marine biota including flounders, longnose killifish, brown shrimp, blue crab, and squid. Previously, Murray et al. (1980) had reported levels of PCP up to 8.3 ng/g (wet weight) in oysters from Galveston Bay.

Since 1976, CPs have been monitored in Dutch surface waters and sediments of the major industrialized rivers flowing through the Netherlands, including the Rhine, Boven Merwede, Ijssel, and the Meuse, as well as Lake Ketelmeer which is a deposition area for Rhine river sediments. The CPs which had the highest frequency of occurrence in waters of the Rhine included 2,6-DCP, 2,4,6,-TCP, 2,3,4,6-TTCP, and PCP (Wegman and Hofstee, 1979). The CPs with a 100% frequency of occurrence in sediments from Lake Ketelmeer included 2,5-DCP, 2,3,5- and 2,4,5,-TCP, 2,3,4,5,- and 2,3,4,6,-TTCP and PCP (Wegman and van den Broek, 1983). The CP monitoring program identified the Nieuwe Maas River near Rotterdam, a heavily industrialized area, as the locale with the highest concentration of CPs in the sediments.

Eder and Weber (1980) quantified CPs in samples of Weser estuary moist sediments, suspended matter and in water. Incidentally, Eder (1980) demonstrated that a source for the 2,4,5-TCP was the phenoxy herbicide, 2,4,5-T, also identified in the sediments, at a level of 10.6 ng/g. Eder and Weber (1980) suggested that a positive correlation exists between the CP content and the water holding capacity of the sediments. They also suggested that even in highly turbulent estuarine waters, suspended sediments play only a minor role in lateral transport of CPs. Pierce and Victor (1978) as noted in Jones (1981) indicated that although suspended sediments may contain less PCP

than in the water column, i.e., particulate PCP vs. dissolved PCP, the suspended sediments are important for the vertical transport of PCP to the sediments.

The lack of information on levels of CPs in air was noted in Jones (1981. Sect. 5.3). One paper overlooked was by Cautreels et al. (1977) who reported on analyses of the organic fraction of airborne particulate matter from two diverse locations: in the mountains above La Paz, Bolivia, at an elevation of 5200 m and in the residential area of Antwerp, Belgium. Two samples from the Bolivia background station contained 0.93 and 0.25 µg PCP/1000 m^3, while levels from the four Antwerp samples varied from 5.7 to 7.8 µg PCP/1000 m^3.

8 METABOLISM

8.1 Aquatic

8.1.1 Biota. The metabolism of PCP in fish and shellfish was reviewed and summarized by Kobayashi (1978, 1979). He and his coworkers, who used the goldfish, *Carassius auratus*, as their test species have made major contributions of knowledge in this area (Jones, 1981. App. 5, Sect. 5.2.1.1). Pertinent points brought out in these reviews may be summarized as follows:

1) PCP was rapidly absorbed by goldfish and accumulated in various organs, especially the gall bladder, where a concentration factor of over 5000 was calculated from observed levels in the media and the gall bladder following 24-h exposure to (^{14}C) PCP and a further 24-h culture in running water.
2) Excretion of PCP from fish was rapid and mostly as the conjugate, pentachlorophenylsulfate.
3) The enzyme activity responsible for conjugation and hence detoxification of phenols decreased with increasing Cl substitution on the phenol ring, i.e., PCP was associated with the lowest enzyme activity. As a corollary, PCP had the highest bioconcentration factor, 475, whereas 2-CP had one of 6.4. These bioconcentration factors were obtained in fish which had died when exposed to 0.2 ppm PCP and 20 ppm of 2-CP during a 24-h LC_{50} test with each compound. The most toxic of the CPs to goldfish was PCP with a 24-h LC_{50} of 0.27 ppm compared to 16 ppm for 2-CP.
4) Most of the PCP identified in the gall bladder was the conjugate pentachlorophenyl-β-glucuronide.
5) Direct excretion of PCP from fish was mostly via the gills and kidneys. More than 95% of the PCP excretion via the kidneys was as pentachlorophenylsulfate.
6) Pre-exposing fish to sublethal PCP-levels increased both the PCP tolerance and the sulfate conjugation activity in the fish.

Rao et al. (1981) demonstrated that elimination of (^{14}C) PCP in a crustacean, the grass shrimp (*Palaemonetes pugio*), occurred along metabolic pathways similar to those in the goldfish, and included methylation (anisole formation), glucuronide conjugation, and dechlorination.

The uptake and depuration rate for 2,4,5-TCP in fathead minnow (*Pimephales promelas*) parallels that for PCP. Rapid uptake of (^{14}C) 2,4,5-TCP was observed by Call

et al. (1980) in minnows exposed to 4.8 µg and 49.3 µg 2,4,5-TCP/L. Uptake rates were approximately 0.2 and 3.4 µg/(g·L·h), respectively, at the two exposure levels. Exposure of the minnows to 2,4,5-TCP for 1-28 days led to a mean plateau concentration factor of approximately 1800. As with PCP (Jones, 1981: App. 8, Sect. 8.1) depuration of 2,4,5-TCP was initially rapid, i.e., up to 92% of the ^{14}C in 24 hours, which was then followed by an extended period of depuration in which the half-life of the remaining ^{14}C was 21-28 days. Similarly, Bahig et al. (1981) in an excretion-metabolism study observed that male Wistar, Sprague-Dawley rats excreted approximately 92.5% of the daily applied dose of (^{14}C) 2,4,6-TCP in feces. The 2,4,6-TCP was not significantly degraded by rats. Excretion products included conjugates and isomers including 2,3,6- and 2,4,5-TCP.

8.1.2 Vegetation. There has been little information available on the uptake and metabolism of PCP by plants grown in PCP-treated soil. Herbicides and soil sterilant products containing PCP have been suspended from sale in Canada, as of January 1, 1981 (Jones, 1981).

Weiss, Moza et al. (1982) investigated the fate of (^{14}C) PCP in rice plants grown in (^{14}C) PCP-treated soil (23 kg/ha) over two vegetation periods under flooded conditions. The authors noted that the uptake of radioactivity by plants was 12.9% in the first and 2.5% in the second year. The percent distribution of the applied radioactivity in the roots, after the first year of growth, was as follows: free PCP (0.14%), conjugated PCP (0.06%), total free and conjugated lower CPs (0.43%), anisoles (0.07%), dimethoxy-tetrachlorobenzenes (0.01%), 1,2-dihydroxy- and/or monohydroxymonomethoxytetrachlo-robenzene (0.03%), polar nonhydrolyzable substances (0.48%), and unextractable residues (3.95%). In the straw, no CPs or anisoles were detected. The authors stated that the main metabolite in the straw was probably tetrachlorobenzoquinone (0.63%). In the grain, low radioactive residues (0.12%) could not be identified as to compound. At the end of the first year the total unextractable radioactivity in the plants was 8.45% of that applied. The authors noted that in the second year the portion of unextractable residues in the plants increased, and lower chlorinated conjugated phenols were identified.

8.1.3 Flooded Soils. At the same time that Weiss, Moza et al. (1982) investigated the fate of (^{14}C) PCP in rice plants, the fate of (^{14}C) PCP in the treated soil was also examined (Weiss, Scheunert et al., 1982). Following the first growing period, the residues from (^{14}C) PCP in the soil, and the percent of applied radioactivity attributed to each, were as follows: unidentified unextractable substances (28.61%), free PCP (0.51%), conjugated PCP (0.61%), free TTCPs and TCPs (1.67%), conjugated TCPs (0.01%), anisoles

(0.33%), and highly polar, mostly nonhydrolyzable compounds (4.74%), for a total of 36.5%. Approximately 55% of the applied radioactivity was lost by volatilization. The authors noted that in the second growing period, the level of unextractable residues in the soil increased, as was the case for rice plants, although the composition of the extractable radioactivity was similar to that of the first vegetation period. As a result of their studies the authors concluded that PCP is not persistent in either flooded soils or rice plants. In the case of soil, the PCP is partly volatilized or mineralized, partly degraded, and incorporated into soil constituents as unextractable residues. In the rice plants, PCP is degraded and incorporated into natural plant constituents within one growing period. Under the specific conditions of this study of PCP in flooded agricultural soil, the unchanged free and conjugated PCP present in soil and plants after one growing season was less than 2 % of the applied amount and the major residues of PCP in soil as well as in plants were bound residues. The authors acknowledged that the toxicological importance of these bound residues could not be assessed at that time.

8.2 Terrestrial

Leighty and Fentiman (1982) demonstrated that PCP can be conjugated to palmitic acid in an *in vitro* rat liver coenzyme A fortified microsomal system. Leighty had voiced the concern that, if PCP is retained in trace amounts in human liver as in rats, then there is the possibility it could interfere with metabolism of other toxic substances (Anonymous, 1982).

9 ECOTOXICOLOGY

9.1 Ecosystem Effects

9.1.1 Marine. As noted in Jones (1981. Sect. 7) the impact of PCP on a marine ecosystem was investigated in Saanich Inlet, British Columbia, by the Institute of Ocean Sciences (Yunker, 1981). Nominal levels of PCP in the ecosystem enclosures were 10 and 100 µg/L which were reduced by approximately 67% after 25 days, primarily by photolysis. Exposure of the marine ecosystem to PCP shifted the centric diatom balance, reduced the numbers of phytoplankton, reduced sedimentation and, at the higher concentration, decimated the population of a major diatom. Marine bacteria populations were initially reduced by exposure to the PCP, but adapted and recovered as did phytoplankton populations with a concomitant shift in species.

9.1.2 Estuarine. Structures of field- and laboratory-developed estuarine benthic communities were significantly altered when exposed to ≃ 141 µg PCP/L for one week at a nominal temperature of 18°C (14 to 23°C) but not at a concentration of ≃ 13 µg PCP/L (Tagatz et al., 1981). In the aquaria with PCP at ≃ 141 µg PCP/L, observed effects on communities included a reduction in average number of species in both field- and laboratory-developed communities and a significant reduction in numbers of individuals in the laboratory-developed communities. The results also indicated that the "No Observable Effect Level" for PCP in estuarine benthic communities would fall between 13 and 141 µg PCP/L if similar test conditions prevailed.

Hauch et al. (1980) investigated acute and chronic toxicity of NaPCP to adults of the marine planktonic copepod, *Pseudodiaptomus coronatus*. A 96-h LC_{50} acute toxicity value of 68 µg NaPCP/L, with a 95% confidence interval of 32.6-141.8 µg NaPCP/L, was based on nominal initial concentrations. Chronic toxicity was determined on the basis of food ingestion rate as measured by fecal pellet production. The authors concluded that concentrations of NaPCP below 100 µg/L could alter community structure in a two-step sequence. Initially there would be overexploitation of primary producers as a result of the copepods increased feeding rate followed by a copepod population decline coincidental with its reduced food supply.

9.1.3 Freshwater. Somewhat similar effects to those noted by Hauch et al. (1980) were observed by Schauerte et al. (1982) in the population dynamics of biota in an outdoor experimental pond subjected to application of single doses of 2,4,6-TCP at 5 mg/L and PCP at 1 mg/L, in duplicate. The pond, 2-m diam. by 50-cm deep, was compartment-

alized by six PVC-tubes of 50-cm diameter with an average volume of 100 L each. Daphnia populations declined to zero after three days exposure to PCP and eight days for TCP. At the same time, there was a decrease in autotrophic phytoplankton, i.e., blue-algae and diatoms which are species indicative of clean water associations, coupled with an increase in populations of flagellates and microorganisms, biota indicative of contamination. As a result of this shift in balance between autotrophic and heterotrophic populations, a secondary effect was a significant decrease in dissolved oxygen concentration.

9.2 Biochemical Effects

9.2.1 Aquatic Biota.

9.2.1.1 Polychaetes. The response to PCP in the sandworm, *Neanthes virens* (Sars.), was investigated through monitoring of the biochemical indices: 1) coelomic fluid glucose; 2) coelomic fluid osmolality; and 3) tissue ascorbic acid and glycogen (Carr and Neff, 1981; Thomas et al, 1981). Carr and Neff (1981) stated that of the three indices, tissue glycogen concentration was the only parameter showing significant depletion during chronic exposure to 100 µg PCP/L. This decrease in glycogen reserves was accompanied by a significant increase in ascorbic acid in both parapodial tissue and posterior segments.

9.2.1.2 Vertebrates. In juvenile striped mullet (*Mugil cephalus*), stress responses to PCP were monitored via plasma cholesterol concentrations, plasma osmolality, plasma glucose concentrations, and hepatic glycogen concentration (Thomas et al, 1981). Exposure of mullet to 100 and 200 µg PCP/L affected these metabolic parameters as follows: 1) primarily, the plasma cortisol concentration increased accompanied by a secondary response of marked hyperglycemia; 2) additionally there was depletion of hepatic glycogen reserves; and 3) hepatic ascorbic acid concentrations and serum osmolality rose slowly. Thomas et al. (1981) noted that the magnitude and the time course of the biochemical responses of mullet were directly related to exposure concentrations and accumulation of PCP and, further, that "profound changes in ascorbic acid metabolism do occur during lethal and sublethal exposure to pollutants." It was speculated that there was a requirement for ascorbic acid for induction of the glucuronic acid pathway for the detoxification and elimination of PCP.

9.2.1.3 Phytoplankton. Jayaweera et al. (1982) correlated the toxic effect of PCP, in the green alga, *Selenastrum capricornutum*, as measured by the degree of reduction of photosynthetic ^{14}C assimilation over a 2-h period at a given pH, with a PCP-induced

decrease in electrical resistance of artificial lipid membranes and development of a negative membrane surface charge. The experimental results of the authors suggested to them that "the toxic effect of PCP is associated with the adsorption of PCP on cell and subcellular membranes and that PCP toxicity can be due to the resulting induced hydrogen ion transfer, which is the process responsible for the decrease of membrane electrical resistance." Jayaweera et al. (1982) noted that, under illumination, the toxic effect of PCP to *S. capricornutum* started to occur at (2-5) x 10^{-7} M PCP and at 10^{-5} M PCP the carbon fixation was essentially completely abolished.

Erickson and Hawkins (1980) determined through use of ^{14}C uptake in 48-h tests, the effects of 4-CP, 2,4,6-TCP, and PCP on photosynthesis in native estuarine phytoplankton in flowing sea water. Tests were conducted under existing environmental conditions; for example, pH was approximately 7.8 and temperatures ranged from 12.5-13.5°C for 4-CP, 11-13°C for 2,4,6-TCP, and 3.0-5.5°C for PCP. The concentrations used in the tests were 0.5, 1.0 and 2.0 mg/L for all three CPs, plus additional concentrations of 0.125 and 0.25 mg/L which provided a broader range for PCP. These concentrations are those which could occur near spills or outfalls. No effects were shown by the phytoplankton exposed to the 2,4,6-TCP and only slight inhibition was exhibited from exposure to the 4-CP. The greatest effect was from exposure to 0.5, 1.0 and 2.0 mg PCP/L which inhibited photosynthesis in the phytoplankton by 61, 84 and 98%, respectively.

In an 8-11 day study by Gotham and Rhee (1982), PCP appeared to inhibit photosynthesis as measured both per cell and per unit of chlorophyll in three species of algae, *Ankistrodesmus falcatus, Microcystis* sp., and *Melosira* sp., at PCP concentrations of (4-8) x 10^{-7}, (2-11) x 10^{-8}, and (2-6) x 10^{-7} ng PCP/cell, respectively. Although photosynthesis was inhibited in the three species of algae at these PCP concentrations, only the *Melosira* sp. showed a discernible decrease in growth rate.

9.2.1.4 Macrophytes. There have been few published studies on effects of PCP on aquatic macrophytes (Jones, 1981). Recently, Huber et al. (1982) reported on the effects of PCP on the metabolism of the aquatic macrophyte, *Lemna minor* L. The authors stated that exposure of *L. minor* to PCP inhibited photosynthetic O_2 production. The reduction of photosynthetic activity was accompanied by structural changes in the chloroplast membranes. PCP did not have a pronounced effect on O_2 consumption of *L. minor* during dark respiration. The chlorophyll content of *L. minor* decreased rapidly with increasing concentration of PCP in the growth medium. In regard to enzyme activity, PCP in low

concentrations inhibited glutamate dehydrogenase activity in *L. minor*, but had no distinct effect on the activity of alanine aminotransferase.

9.2.2 Terrestrial Biota. Histopathological effects of 2,3,4,6-TTCP applied intragastrically in two-month-old Wistar rats were almost completely confined to the liver. The effect level was between 50 and 100 mg 2,3,4,6-TTCP/kg (Hattula et al., 1981a).

A study of chronic toxicity of technical and analytical grade PCP in female yearling Holstein cattle clearly demonstrated that the toxicity of PCP was primarily attributable to its contamination with impurities including polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans, and hexachlorobenzene (McConnell et al., 1980). Major findings in this clinicopathology study were that the animals exposed to analytical grade PCP were, in general, undifferentiated from the controls. In the animals exposed to technical grade PCP, there was dose-related decrease in body weight, decreased feed efficiency, progressive anaemia, a dose-related increase in liver and lung weight, and a decrease in thymus weight. Levels of dioxins and furans in liver and adipose tissue correlated with the concentration of technical PCP in the diet (Parker et al. 1980). Levels of dioxins were higher in the liver than in the adipose tissue. Levels of HCB in the blood increased as the dose of technical PCP increased, whereas blood PCP levels were lowest in the cows fed the highest technical PCP dose.

The rationale for curtailment of use of PCP treated wood where livestock could be exposed can be highlighted by a study by Kinzell et al. (1981) in which four Holstein cattle in early lactation were fed technical grade PCP at subchronic effect levels, 0.2 mg PCP/kg body weight per day for 75-84 days followed by 2 mg PCP/kg body weight per day for 56-60 days. Each treated cow was paired with a control cow at an equivalent stage of lactation. Kinzell et al. (1981) observed that milk production, feed intake and body weight were unaffected by the treatment, except that the treated cows were more efficient converters of feed to milk during the early stage of the 2 mg PCP/kg treatment period. However, postmortem examination showed enlargement of liver, lungs, kidneys and adrenals and thickening of the urinary bladder wall. *In vitro* testing of kidney slices confirmed significant loss of renal function.

The authors had cautioned that due care should be exercised if the experimental results were to be extrapolated "to dairy farms, since environmental exposure may involve substantial cutaneous and respiratory exposure to one or more penta components."

9.3 Toxicity to Aquatic Biota

9.3.1 Acute Toxicity. An exhaustive series of aquatic toxicity tests with PCP were carried out by Adema and Vink (1981) who utilized various life stages of 14 species of marine and freshwater animals including molluscs (4), a worm (1), crustaceans (6), and fish (3), together with seven species of algae. The criteria used to assess the acute, subchronic, and chronic tests and reproductive studies, as well as identifying the "No Observable Effect Level," included hatching, morphology, mortality, reproduction, and growth. The comprehensive test results were summarized and presented as averages based on duplicate tests which were repeated two to five times. Confidence limits were not calculated due to varying numbers of observations per combination of test compound/test species. The authors concluded that there were no significant differences in sensitivity to PCP between the groups as measured by average LC_{50} values, of which 80% fell in the range 0.1-1 mg PCP/L. As to specifics, they stated that the LC_{50} values for the usual exposure times lay between 1 and 10 mg PCP/L for three Crustacea, *Artemia salina*, *Crangon crangon*, and *Palaemonetes varians*; between 0.01 and 0.1 mg PCP/L for a mollusc, *Dreissena polymorpha*, and between 0.1 and 1 mg PCP/L for all the other test animals. The EC_{50} values for unicellular algae were between 0.1 and 10 mg PCP/L. The lowest concentration of PCP at which sublethal effects were noted in the most sensitive species and life stages was 0.01 mg PCP/L. As stated by Adema and Vink (1981), their results were in agreement with those of others presented in the literature through 1976, which were largely summarized in Jones (1981. Table A4-3).

When growth of the bacteria, *Pseudomonas fluorescens*, which does not degrade PCP (Trevors, 1982b), was used as a measureable response for assaying biological toxicity of PCP, a high correlation was observed between PCP concentration and inhibition of actively growing cells (Trevors et al., 1981). Concentrations of 25 to 50 µg PCP/mL of nutrient broth produced a delay in the log phase of growth of *P. fluorescens*, and, after 1-h exposure to 75 µg PCP/mL, growth was completely inhibited. Oxygen consumption by *P. fluorescens* incubated in nutrient broth at 20°C was inhibited from 21 to 43% by PCP at concentrations of 25 to 200 µg PCP/mL after 12 h of incubation, while CO_2 evolution was not inhibited by 25 µg PCP/mL (Trevors, 1982b). Trevors (1982b) has noted the implication of these test results to the proposed use of residual oxygen tests as a rapid test for estimating the toxicity of aquatic contaminants, in that higher concentrations of PCP were required to produce a toxic response in the respiratory tests than in the growth tests.

In an additional study, Trevors et al. (1982) demonstrated that sequence of exposure to toxicants, such as PCP and 2,3,4,5-TTCP, as well as toxicant concentration would affect bioassay results when using *Pseudomonas fluorescens*. The LC_{50} values and their standard deviation for PCP and 2,3,4,5-TTCP were 29.2 ± 0.6 and 23.2 ± 1.2 µg/mL, respectively, when test data were analyzed by probit analysis. Little is known of the mechanisms for "sensitizing" and "desensitizing" an organism to a toxicant. For example, Trevors et al. (1982) observed that when a standardized cell suspension of *P. fluorescens* was exposed to PCP for 1-h, followed by a 6-h or 16-h recovery period, and then re-exposed to PCP, the cells became less susceptible to the second treatment; whereas, if the second exposure was to 2,3,4,5-TTCP, then the organism was sensitive to the second exposure. It was determined that the most toxic sequence was an initial exposure to 2,3,4,5-TTCP followed by a second exposure to PCP.

Chapman et al. (1982a, 1982b) studied the relative tolerance of aquatic oligochaetes to selected chemical pollutants and environmental factors by utilizing 96-h acute lethal bioassays. Tests were conducted both with and without sediments. The 12 oligochaete species selected for the study included nine freshwater species and three salt-water species. Each of these species is usually associated with a particular level of trophism in an environmental system, i.e., eutrophic, mesotrophic, or oligotrophic. In the bioassay tests with no sediment present, the 96-h LC_{50} values for the oligochaetes exposed to NaCP at pH 7.0 and 10°C ranged from 0.105 to 0.98 mg PCP/L. These values rank the oligochaete species tested as somewhat more tolerant to NaPCP than fresh-water fishes (Jones, 1981. Table A4-3). When sediment was included in the tests the LC_{50} values ranged from 0.56 to 3.6 mg PCP/L. In most cases this meant the presence of sediments increased the 96-h LC_{50} value by an order of magnitude, which the authors pointed out demonstrated the role of sediments as important modifiers of toxic effects on oligochaetes. They also noted that an unexpected result of the study showed that freshwater oligotrophic species were most tolerant to NaPCP, and a eutrophic species was least tolerant, which was not anticipated from known field distributions of oligochaetes. When test conditions included variations in pH, temperature, and salinity, the relative tolerance of particular oligochaete species to NaPCP was maintained although particular factors may have enhanced or depressed tolerances, e.g., the tolerances of all species to NaPCP were significantly enhanced at 1 and 20°C as compared to 10°C, and three freshwater species were significantly more tolerant to NaPCP under saline conditions than in fresh water.

Gupta and Rao (1982) developed 96-h static LC_{50} test data for PCP and NaPCP for the freshwater pulmonate snail, *Lymnaea acuminata* (Lamarck). At a nominal temperature of 18°C and at pH 7.9, the 96-h LC_{50} values and their 95% confidence limits were 0.16 (0.138-0.186) mg PCP/L and 0.19 (0.161-0.224) mg NaPCP/L. These values are within the range of the LC_{50} values for PCP for crustaceans as noted by Adema and Vink (1981).

There has been a lack of comparative toxicity data for CPs in crustaceans (Jones, 1981. Table A4-3). Rao et al. (1981) carried out a series of 96-h toxicity tests with the grass shrimp, *Palaemonetes pugio.* Shrimp were exposed to CPs in filtered sea water held at 20 ± 1°C, and with salinity at 10% and pH 7.6-7.7. Rao et al. (1981) demonstrated that molting shrimp were more sensitive to most of the CPs, with the exception of 2,4-DCP, than shrimp in the intermolt cycle (Table 3).

TABLE 3 ACUTE TOXICITY (96-h LC_{50}) OF CHLOROPHENOLS TO A GRASS SHRIMP, *PALAEMONETES PUGIO* (from Rao et al., 1981)

CP	96-h LC_{50} (95% fiducial limits) mg/L	
	Intermolt shrimp	Molting shrimp
2,4-DCP	2.55 (2.28-2.86)	2.16 (1.49-2.73)
2,4,5-TCP	1.12 (0.92-1.43)	0.64 (0.36-0.80)
2,4,6-TCP	3.95 (3.28-4.95)	1.21 (1.11-1.31)
2,3,4,5-TTCP	0.86 (0.73-0.98)	0.37 (0.35-0.39)
2,3,4,6-TTCP	3.70 (2.98-5.25)	0.81 (0.64-0.89)
2,3,5,6-TTCP	4.10 (3.30-5.31)	1.17 (1.08-1.27)
PCP	2.50 (1.91-3.29)	0.44 (0.18-0.67)

Although *Daphnia magna* are commonly used for aquatic toxicity tests, there were no CP toxicity data for this producer in Table A4-3 in Jones (1981), therefore data from LeBlanc (1980) are now presented (Table 4). Test conditions and procedures followed those recommended by the U.S. EPA and included use of reconstituted well water with a mean hardness of 173 mg/L as $CaCO_3$, pH 8.0 ± 0.2 and a dissolved oxygen concentration of greater than 60% of saturation. The temperature was controlled at 22 ± 1°C. Mortality data were used to calculate an LC_{50} and its 95% confidence limits using various statistical techniques.

Heitmuller et al. (1981), using a similar test protocol obtained LC_{50} data for sheepshead minnow (*Cyprinodon variegatus*) for three of the CPs tested by LeBlanc (1980). Although tests were conducted at a slightly higher temperature range of 25-31°C, than were the *Daphnia magna* tests, the sheepshead minnow were as sensitive to CPs as the *Daphnia magna* (Table 4).

TABLE 4 ACUTE TOXICITY OF CHLOROPHENOLS TO *DAPHNIA MAGNA* AND *CYPRINODON VARIEGATUS**

Compound (Confidence Limits)	Organism tested	24-h LC_{50} (mg/L)	48-h LC_{50} (mg/L)	No discernible effect conc. (mg/L)
2-CP	*Daphnia magna*	>22	2.6 (2.1-3.2)**	1.0
4-CP	"	8.8 (6.9-12)	4.1 (3.2-5.0)	1.1
4-CP	*C. variegatus*	5.7 (4.9-6.4)	5.4 (4.3-7.1)	3.2
2,4-DCP	*Daphnia magna*	>10	2.6 (1.7-3.7)	0.46
2,4,5-TCP	"	3.8 (3.2-4.7)	2.7 (2.3-3.0)	0.78
2,4,5-TCP	*C. variegatus*	2.4 (1.9-3.0)	1.7 (0.9-3.2)	1.0
2,4,6-TCP	*Daphnia magna*	15 (12-19)	6.0 (3.8-8.5)	0.41
2,3,4,6-TTCP	"	>1.0	0.29 (0.070-1.2)	0.010
2,3,5,6-TTCP	"	2.5 (1.1-5.1)	0.57 (0.28-1.3)	0.010
2,3,5,6-TTCP	*C. variegatus*	2.0 (1.5-2.5)	2.0 (1.5-2.5)	1.0
PCP	*Daphnia magna*	1.5 (1.1-2.0)	0.68 (0.60-0.79)	0.32

*Data extracted from LeBlanc (1980).

**95% Confidence interval.

The toxicity of a CP varies with pH and degree of ionization of the CP. For example, changes in pH did not appreciably affect the toxicity of 4-CP to guppy, *Poecilia reticulata* Peters, as measured by its 96-h LC_{50} of 49 µmol/L at pH 5 to 70 µmol/L at pH 8. 4-CP is primarily non-ionized over this pH range. For 2,4,5-TCP, 2,4,6-TCP, and PCP, which are more acidic phenols, the toxicity decreased as the pH increased; for example, the 96-h LC_{50} for PCP at pH 5 was 0.16 µmol/L and at pH 8 was 3.42 µmol/L (Saarikoski and Viluksela, 1981).

Phipps et al. (1981) had determined the 96-h LC_{50} values for PCP, 2,4-DCP, 2,4,6-TCP, and 2-CP as 0.22, 8.25, 9.1, and 12.0 mg/L, respectively, for fathead minnow (*Pimephales promelas*) in Lake Superior water in flow-through tests at 25 ± 2°C and at a pH of 7.2-7.9 Holcombe et al. (1980) demonstrated the effect of pH increases on the acute toxicity of 2,4-DCP to fathead minnow. These authors observed that schooling behaviour was completely disrupted and the equilibrium of most of the fish was affected after a 24-h exposure to 7.43 mg 2,4-DCP/L at pH 7.57, but neither effects were observed at pH 8.68 and 9.08 even after 192 h. When the 2,4-DCP concentration was increased to 12.33 mg/L both schooling and swimming behaviour were affected at all pH levels; furthermore, the authors noted that survival of these fish after 24 h ranged from 0% at pH 7.84 to 46% at pH 8.81.

Liu et al. (1982) who developed a modified resazurin reduction procedure to measure chemical toxicity, utilized the improved test method to evaluate the toxicity/structure relationship of 2,4- and 2,6-DCPs, and 2,3,5-, 2,4,5- and 2,4,6-TCPs. They found that the 2,6-chlorine substitution on the phenol nucleus decreased the CPs toxicity in bacterial culture, as had been noted by Hattula et al. (1981b) in CP toxicity tests with trout.

9.3.2 Chronic Toxicity. There exists considerable information on acute toxicity of PCP to fish (Jones, 1981), but chronic toxicity test data remain scarce. Cleveland et al. (1982) evaluated via 90-d partial life-cycle toxicity studies with fathead minnow (*Pimephales promelas*) three preparations of PCP at exposure concentrations as follows: Dowicide EC-7 (8-139 µg/L), a purified PCP (10-142 µg/L), and a commercial composite PCP (6-121 µg/L). The Dowicide EC-7 contained a broad spectrum of impurities including hexachlorobenzene, chlorophenoxyphenols, chlorodiphenyloxides, chlorodibenzo-*p*-dioxins and chlorodibenzofurans, but all were at relatively low concentrations. The purified PCP only contained chlorophenoxyphenols, but at high levels, while the commercial composite PCP, by comparison, was grossly contaminated with chlorophenoxyphenols,

chlorodibenzo-*p*-dioxins and chlorodibenzofurans. Cleveland et al. (1982) stated that Dowicide EC-7 was the least toxic of the PCPs tested and that Dowicide EC-7 at the maximum level tested, 139 µg/L, did not adversely affect growth or survival of fathead minnow. The purified PCP at concentrations $\geq$ 85 µg/L reduced growth of fathead minnow but did not affect survival. The commercial composite PCP reduced growth at concentrations $\geq$ 13 µg/L and reduced survival at concentrations of $\geq$ 27 µg/L. In addition, fathead minnow exposed to commercial composite PCP showed degredation of fins and opercles and malformation of the anterior regions of the skull. Cleveland et al. (1982) ably demonstrated that impurities present in PCP can contribute to the toxic effects of PCP in fathead minnow when they are chronically exposed to PCP.

The effects of 2,4-DCP and PCP on fathead minnow, *Pimephales promelas*, were investigated in 32-day flow-through tests with the embryo-larval and early juvenile life stages (Holcombe et al., 1982). The most sensitive indicator of stress from exposure to PCP was growth, whereas survival of the minnow was the most sensitive indicator of toxic effects from 2,4-DCP exposure. The maximum acceptable toxicant concentration for fathead minnow in Lake Superior water at pH 7.2-7.9 was within the range of 290-460 µg 2,4-DCP/L and 44.9-73.0 µg PCP/L.

Hodson and Blunt (1981) exposed the three early life stages of rainbow trout (*Salmo gairdneri*) - embryo, alevin, and fry - to NaPCP at four nominal concentrations of 0, 10, 32, and 100 µg NaPCP/L and at two temperature regimes, cold (6, 6 and 12°C for each life stage, respectively) and warm (10, 15 and 20°C for each life stage, respectively), and at a pH of $\approx$ 8.0. The diluent for the NaPCP was dechlorinated Burlington municipal water from Lake Ontario. When the results were examined and the interactions tested statistically, the following general but significant conclusions were noted by Hodson and Blunt (1981), which may apply to field populations of trout which are chronically exposed to NaPCP.

1) Exposure to NaPCP during springtime egg development coupled with low temperatures would have adverse effects on eggs and alevins.
2) As temperatures increase through the season, the PCP effect on growth is enhanced.
3) NaPCP toxicity and temperature effects on toxicity were not as great to alevin and fry if eggs were not exposed to NaPCP.

Based on these conclusions the authors recommended that waterborne NaPCP concentrations be kept to a minimum during springtime egg development.

9.4 Immunosuppression

9.4.1 Aquatic Biota. Anderson et al. (1981) examined the role of PCP as an immunosuppressant in a bivalve, *Mercenaria mercenaria*. They used the clearance rate from the clam haemolymph of an injected marine *Flavobacterium* sp. as an indicator. Clams, injected with the bacteria, were exposed to PCP in both flow-through and recirculation systems for up to 18 weeks, at PCP levels which produced no increased mortality. In short, when PCP reaches tissue levels of approximately 500 ppb then significant inhibition of clearance can be expected. In some cases treated clams showed complete failure to clear, whereas untreated clams routinely cleared 90% of the bacteria. The authors interpreted these findings as implying that resistance to bacterial infection is decreased by exposure to PCP.

9.4.2 Terrestrial Biota. Adult male C57B1/6 mice which were fed diets containing 50 or 500 ppm pure (99%) or technical grade (86%) PCP for up to 12 weeks were assessed for immunocompetence by *in vivo* and *in vitro* assays (Kerkvliet et al., 1982). In essence, the study showed that the immune system in mice was sensitive to PCP exposure and, additionally, the impurities present in the technical grade PCP also adversely affected immunocompetence.

Early lactating Holstein-Friesian cattle fed technical PCP at 0.2 and 2.0 mg/kg/day for 75-84 and 56-62 days, respectively, had total PCP concentrations in the blood at steady state of 2.9 and 12.5 ppm, respectively, for the low and high levels of exposure (Forsell et al., 1981). The levels chosen were to approximate those found in a dairy farm environment. Forsell et al. (1981) using both *in vitro* and *in vivo* immunoassays concluded that, in cattle with blood levels of 12.5 ppm PCP, there was no PCP-induced immune deficiency.

9.5 Mutagenicity

There has been some concern that the CPs generated from aqueous chlorination of unbleached kraft pulp might be mutagenic. Rapson et al. (1980) confirmed, in a series of Ames TA 100 tests, the results of Rasanen et al. (1977) (Jones, 1981. Sect. 3.1.1.4) that no mutagenicity was shown by 2,3-, 2,4-, 2,5-, 2,6-, 3,4-, and 3,5-DCP, or 2,3,4-, 2,3,5-, and 2,4,6-TCP. Rapson et al. (1980) did show that, following the chlorination of groundwood pulp, lignin was the main source of mutagenicity.

9.6 Teratogenicity

In a study by Virtanen and Hattula (1982) on the fate of 2,4,6-TCP in a flow-through aquatic system, in addition to the observations on bioconcentration of 2,4,6-TCP in the organisms studied, there was some evidence that 2,4,6-TCP may be a "potent teratogen." This conclusion was based on the observations that adult guppy, *Poecilia reticulata*, which had been exposed to ≈0.8 ppb of 2,4,6-TCP in the aquaria water at pH ≈7.0 and temp. ≈20°C, gave birth to fewer offspring than the control fish, 90 compared to 180. As well, of the young that were born the mortality rate was higher, 24.4% compared to 4.4%. In addition, several young fish born from the 2,4,6-TCP exposed parents had an unusual curvature of the spine. In four exposed female fish sampled at day 36, the mean whole body concentration of 2,4,6-TCP was 0.609 ± (SD) 0.516 ppm (Virtanen and Hattula, 1982).

10 CHEMODYNAMICS - FATE AND MOBILITY

10.1 Transport

10.1.1 Volatilization. One factor which controls the mobility of PCP in the environment is its rate of evaporation from an aqueous solution. Volatilization of PCP as measured by its residence time in an aqueous solution (in the absence of other removal pathways) is dependent on temperature and pH of the solution (Klopffer et al., 1982). The pH of the aqueous solution controls the degree of dissociation of PCP, a moderately strong acid. Only the unionized form is volatile. Klopffer et al. (1982) found that, at pH 5.1 with 13.2% free phenol, the half-residence time was 328 h at 30°C, while at pH 6.0, with a calculated undissociated fraction of 1.9%, the half-residence time had increased to 3120 h at 30°C. At pH 8.0 no volatilization could be measured (calculated undissociated fraction 0.03%). Therefore, in a natural environment where the surface waters might be close to neutrality, there is little loss of PCP via evaporation.

10.1.2 Adsorption. Concerns in the Federal Republic of Germany with the effect of groundwater pollution on aquifers led Zullei (1981) to investigate infiltration processes of CPs including 2-CP and 2,4-DCP. In a bench-scale activated carbon-sand filter system, both adsorption and biodegradation processes were observed. Zullei (1981) hypothesized that, in soils with high filter velocities (such as 1 m/h) no adsorption takes place because of the high water solubility of the 2-CP and 2,4-DCP, and therefore these phenols can be expected to reach the groundwater.

10.2 Bioaccumulation

Normally, bioaccumulation is studied with molecules in neutral form. Many authors have shown a direct relationship between K_{ow} and bioaccumulation (Jones, 1981). This is difficult for molecules like the CPs where the percent of un-ionized material is pH dependent. This question has been studied recently by Canadian workers, Kaiser and Valdmanis (1982), who drew the following conclusions:

> "Our results demonstrate the strong dependence of the apparent partition coefficient of PCP on the pH and, in basic medium, on the ionic strength. As pH and ionic strengths of effluents containing PCP as well as those of the receiving waters vary widely, the environmental effects and pathways of ionizing compounds such as PCP may vary accordingly. In particular, it would appear that the bioconcentration factors, which generally correlate well with partition coefficients, could vary between different aquatic systems by several orders of magnitude."

PCP and 2,4,6-TCP were included in a series of organic chemical compounds screened for their environmental behaviour in an ecotoxicological profile analysis (Freitag et al., 1982). The test system used ^{14}C-labelled compounds in five accelerated test procedures which provided information on their bioaccumulation in algae; their bioaccumulation in fish; their retention, dispersion, and excretion in rats; their degradation, transformation, and accumulation in activated sludge; and their photomineralization. Although the environmentally well-known chemicals initially tested and reported on were used to calibrate the test system and to provide a basis for screening less well known chemicals, the placement of 2,4,6-TCP and PCP relative to the other compounds in each bioaccumulation test and the photomineralization test is of interest.

The following results were reported by Freitag et al. (1982). The bioaccumulation factors (BFs) for 2,4,6-TCP and PCP in the 24-h algal bioaccumulation test were 51 and 1250, respectively, compared to a BF of 11,500 for 2,2',4,4',6-pentachlorobiphenyl and a BF of 24,800 for hexachlorobenzene (HCB) at the top end of the scale, and a BF of 6 for 2,4-dichlorophenoxyacetic acid (2,4-D) near the low end of the scale. In the fish (Golden orfe) 3-d bioaccumulation test, the BFs for 2,4,6-TCP and PCP were 310 and 1050, respectively, compared to HCB at 1200, 2,2',4,4',6-pentachlorobiphenyl at 2320, and aldrin at 3890. In this test 2,4-D had a BF of <10. In the activated sludge 5-d test the BFs for 2,4,6-TCP and PCP were 40 and 1100, respectively, compared to a BF of 27,800 for the pentachlorobiphenyl, and 35,000 for HCB. The photomineralization 17-h tests produced the following comparative ratings (%CO_2): HCB (1.5), pentachlorobiphenyl (5.2), 2,4-D (26.2), PCP (62.0), and 2,4,6-TCP (65.8).

The experimental findings of Freitag et al. (1982) are in agreement with the environmental evidence for persistence, bioaccumulation, and biodegradation of PCP and 2,4,6-TCP compiled and summarized in Jones (1981).

Based on results of a 14-d non-feeding exposure study of the polychaete *Neanthes virens* (Sars) to [^{14}C] PCP at a concentration of 100 µg/L in Instant Ocean sea water at 10°C and 32-35% salinity, a bioconcentration factor of ≈280 on a wet weight basis was determined (Carr and Neff, 1981). The uptake rate was linear and based on the assumption that if the rate was to remain linear then an extrapolated body burden for non-labelled PCP for *N. virens* for 8-weeks of exposure was calculated as 112 µg PCP/g wet weight (Carr and Neff, 1981). Converting this to a dry weight basis, the bioaccumulation factor was calculated as ≈5600. This compares to the bioconcentration factor of 3830 for PCP in another polychaete, *Lanice conchilega*, exposed to 2-5 µg PCP/L of sea

water at 27% salinity (Ernst, 1979) (Jones, 1981. App. 8, Sect. 8.1). For these studies the concentration of PCP in the water was determined through use of a simple and rapid spectrophotometric technique (Carr et al., 1982).

Thomas et al. (1981) demonstrated that *N. virens* could tolerate a chronic exposure level of 100 µg PCP/L whereas this same concentration was the 48-h LC_{33} for PCP to juvenile striped mullet (*Mugil cephalus*).

An average bioaccumulation factor for PCP of 53 in a marine fish, killifish (*Fundulus similis*), was reached after 168 hours of exposure to PCP at a concentration of 57-610 µg PCP/L of Instant Ocean at pH 7.5 (Trujillo et al., 1982). Although PCP is readily accumulated by marine fish, it is to a lesser extent than in freshwater fish, such as trout, *Salmo trutta*, in which a PCP concentration factor of 100 has been measured in an aquarium study at 5°C (Hattula et al., 1981b). The following information in Table 5 on 24-h LC_{50} values for CPs to trout (Hattula et al., 1981b), is additional to or expands on that which was provided in Jones (1981, Table A4-3). Five trout per test were exposed to nominal concentrations of 0.1 ppm of each CP.

TABLE 5 LC_{50} (24 h) AND CONCENTRATION FACTORS FOR CHLOROPHENOLS IN TROUT (*SALMO TRUTTA*) (Hattula, 1981b)

Compound	LC_{50} (ppm)	Conc. in tissue mg/kg (± SD) (wet weight)	Conc. Factor
2,4-DCP	1.7	18 ± 7	10
2,6-DCP	4.0	no analysis	
2,3,5-TCP	0.8	5.7 ± 4.6	12
2,4,5-TCP	0.9	no analysis	
2,4,6-TCP	1.1	no analysis	
2,3,4,6-TTCP	0.5	210 ± 120	450
PCP	0.2	200 ± 110	100

When rainbow trout (*Salmo gairdneri*) were exposed for 115 days at 15 ± 1°C to low levels of NaPCP, 35 ± 6 ng NaPCP/L and 660 ± 220 ng NaPCP/L, they bioaccumulated the PCP, particularly in the organs, to levels which were related to the concentration and duration of exposure. The trout exposed to 35 ng NaPCP/L contained slightly higher levels of PCP than in the water after 115 days of exposure, while the trout exposed

to 660 ng NaPCP/L had the highest concentrations of PCP, i.e., an average concentration of 2200 µg PCP/kg in the liver and gall bladder (Niimi and McFadden, 1982). As pointed out by the authors, the study did demonstrate that rainbow trout will accumulate PCP when exposed to waterborne concentrations as low as 35 ng NaPCP/L over prolonged periods. They also suggested that in view of their results and those of others that chronic exposure of trout to PCP, at levels up to 660 ng NaPCP/L, would probably not prove lethal but could have adverse effects, particularly in young fish. Controlling factors which influence the toxic effect of PCP to trout, other than the concentration of the PCP, include water temperature and the life-stage of the trout at the time of their exposure to PCP (Hodson and Blunt, 1981) (Sect. 9.3.2). Owen and Rosso (1981) noted that the types of lesions which were observed in the liver of bluegill sunfish (*Lepomis macrochirus*) following exposure to PCP were a non-specific reaction, and would occur following exposure to various pesticides.

Analysis for PCP and TTCP (2,3,4,6- plus 2,3,5,6-) in a limited collection of biota from the Bay of Quinte study (see Sect. 7) showed that PCP and TTCPs bioaccumulated in fish, brown bullhead and yellow perch, to approximately 10^4, with only moderate bioaccumulation of these CPs in leeches (Fox and Joshi, 1983). The low levels, 1-7 ppb of the CPs, quantified in the limited number of samples of chironomids and cladophora, from the bottom of the Bay, indicated little, if any, bioaccumulation of PCP and TTCPs in these biota.

10.3 Degradation

CPs are reactive and do degrade in the environment (particularly in aerobic, illuminated, or bacteria containing media) at specific rates for given conditions and through complex mechanisms, but many problems remain to be solved to adequately account for the continued presence of low, endemic levels of CPs in environmental samples (Jones, 1981; and Sect. 7).

10.3.1 Biological Degradation.

10.3.1.1 Aquatic. de Kreuk and Hanstveit (1981) compared degradation rates of 4-CP; 2,6-DCP; 2,4,5-TCP; 2,4,6-TCP; and PCP in freshwater media and salt water, but because of variability of their data they could not develop a general rule to predict the behaviour of a CP in marine water based on freshwater data. They did note that use of data derived from degradation tests for CPs in freshwater media may overestimate degradation of CPs in the marine environment. Lee and Ryan (1979) used ^{14}C-labeled 4-CP and 2,4,5-TCP to

determine the rate of microbial degradation of these compounds in estuarine waters and sediments and to calculate the half-lives at two temperature regimes of 9°C and 21°C. Although these compounds are no longer of commercial importance in Canada, the research did demonstrate that the rate of microbial degradation of these lower chlorinated CPs is much reduced in water, compared to sediment, and much slower at the lower temperature of 9°C than at 21°C. The results of the microbial degradation tests of some relevance to Canada were the calculated half-life of 490 days, for 4-CP in estuarine water at 9°C compared to 20 days at 21°C. In sediment at 22°C the half-life of 4-CP was calculated as 3 days.

Liu, D., et al. (1981), in a laboratory degradation study of NaPCP in a cyclonic fermentor operated at 22°C with the NaPCP solution at pH 7.0, determined that under aerobic conditions and with an acclimated bacterial culture the half-life of PCP was 0.36 days and under anaerobic conditions the half-life was 192 days. Liu, D., et al. (1981) noted that these results were in qualitative agreement with those of Boyle et al. (1980) who had determined half-lives for PCP in a simulated lentic environment. The half-life for PCP was 19 days, where the pH ranged from 7.5 to 8.5 and the solution was exposed to light, whereas under anaerobic and dark conditions and at a pH of 3.0-5.5, the half-life for PCP was 80 days.

Blades-Fillmore et al. (1982) examined the role of sediments in the biodegradation of 2,4,6-TCP in Delaware River water. They demonstrated that, under their test conditions, 1) the rate of biodegradation of 2,4,6-TCP was directly influenced by the amount of available surface area at the sediment/water interface, and 2) the aerobic bacteria capable of degrading the 2,4,6-TCP were available in the river water. Circumstantial evidence indicated to the authors that biodegradation occurred primarily from attached rather than planktonic microorganisms. Furthermore, Blades-Fillmore et al. (1982) suggested that solid surfaces may act as collection sites for nutrients as well as for the microorganisms that utilize these nutrients. The authors speculated that, under static conditions and following the build-up of a surface colony of microorganisms, a limiting factor in the rate of biodegradation might be the slow diffusion of nutrients or oxygen through the colony, which would affect metabolic efficiency. The presence of oxidizable organic matter also slows the rate of biodegradation. Under optimum aerobic test conditions the 2,4,6-TCP disappeared with a half-life of 3 d.

10.3.1.2 Terrestrial. Baker and Mayfield (1980) attributed loss of CPs and phenols in sterile and non-sterile clay-loam soils of pH 7.1, incubated at 23°C under aerobic and

anaerobic conditions, to two main mechanisms, microbial and non-biological. A common feature of the CP compounds, which showed substantial decreases in levels, was a chlorine that was positioned ortho to the phenolic hydroxyl; conversely, a chlorine in the meta-position to the phenolic hydroxyl renders the compound resistant to microbial degradation. From the data of Baker and Mayfield (1980), the CPs studied can be classified according to the number of days required to reach a certain percent loss of the phenolic compound in soils under aerobic conditions (Table 6). Under anaerobic conditions, decreases in levels of the phenolic compounds were minimal.

TABLE 6 CLASSIFICATION OF CHLOROPHENOLS BY RATE OF LOSS FROM SOIL UNDER AEROBIC CONDITIONS (Baker and Mayfield, 1980)

Rate of Loss Classification	Minimum time in days for (%) decrease in phenolic compound in soils under aerobic conditions at pH 7.1		CPs
1) Rapid	0.25-2	($\geq$70)	2-CP, 4-CP, 2,6-DCP, 2,4,6-TCP
2) Moderate	7-20	($\geq$70)	2,4-DCP
3) Slow	80-160	($\geq$70)	3-CP, 3,4-DCP, 2,4,5-TCP, PCP
4) Persistent	160	(<31)	3,4,5-TCP, 2,3,4,5-TTCP

Baker and Mayfield (1980) stated that volatilization and photodecomposition of CPs did not occur in the test system, but some non-biological loss through adsorption to soil organic matter and polymerization into such organic matter was not ruled out. In addition, the rate of loss of CPs in sterile silica sand progressively increased with temperature and decreased with concentration. Suggested mechanisms for the non-biological loss of the CPs from the silica included catalysis at the surface of the silica or by oxidative mechanisms.

Baker et al. (1980) demonstrated that degradation of 2-CP, 4-CP, and 2,4-DCP occurs in sediments and clay-loam soils at low temperatures, i.e., 0°C and 4°C, but to a lesser extent than at 20°C. In non-sterile stream-water held at 0°C for 40 days there was no loss of CPs directly attributable to microbial action; in fact, 2,4-DCP was the only CP which showed a significant loss in stream-water and that was at 20°C. In a related study Trevors (1982a) identified three *Pseudomonas* spp., isolated from an agricultural sandy loam soil and the freshwater stream, which degraded PCP at a starting concentration of

50 µg/mL at both 4°C and 20°C, but not at 0°C even after 100 days incubation. After 12 days incubation at 20°C the three isolates degraded the PCP 50-56% while at 4°C, degradation of the PCP by the isolates was much slower with more variation in rates between isolates. After 100 days incubation at 4°C, loss of PCP was calculated at 11.9, 23.1 and 51.7% for the three isolates. These results indicated that PCP can be degraded, although slowly, by *Pseudomonas* spp. at low suboptimal growth temperatures which are often found in the environment. The results of the study also demonstrated that pseudomonads can adapt to and degrade toxic compounds such as PCP in the environment. Tam and Trevors (1981) reported that NaPCP at a concentration of 50 µg/g of soil had an inhibitory effect on nitrogen fixation in nonsterile sandy loam soil when incubated aerobically in the dark at 20°C for 12 days; while strong inhibition of nitrogen fixation in nonsterile soil occurred in the presence of 100 µg NaPCP/g of soil. The authors also noted that their results indicated that NaPCP was less inhibitory to soil nitrogenase activity under anaerobic than aerobic conditions.

Edgehill and Finn (1983) conducted a feasibility study on the use of microbial treatment of PCP-contaminated soil to reduce the environmental hazards usually associated with PCP spills or when PCP-treated poles are put into service near fish-bearing waters. When a PCP degrading strain of bacteria, *Arthrobacter* (ATCC 33790) was inoculated at a concentration of 10^6 cells per gram of dry soil, to enhance the disappearance rate of PCP, the half-life of PCP in the soil was reduced from 2 weeks to <1 day. The PCP half-lives were calculated on recoveries of 90%, and for incubations carried out in the laboratory at 30°C. In an associated outdoor trial where daily air temperatures ranged from 8 to 16°C, the growth rate of the inoculated culture was reduced compared to those determined in the laboratory trials. However, the outdoor trial did demonstrate the need for thorough mixing of the soil at time of inoculation. This protocol led to a reduction of 85% of the extractable PCP at 12 days compared to approximately 15-30% reduction of PCP without this treatment.

10.3.2 Photochemical Degradation. Wong and Crosby (1981), in a further refinement of their studies on photolysis of PCP (Jones, 1981. App. 6, Sect. 6.1.2), noted that with laboratory irradiation (equivalent to summer sunlight) of buffered dilute aqueous solutions of PCP (0.38 mM, 100 mg/L) the half-life of PCP at pH 3.3 was ≈100 h, while at pH 7.3 it was only 3.5 h. This latter number compares to a half-life of 48 h (total elapsed) and a total disappearance of the PCP within 10 days. The photodecomposition products from PCP identified by Wong and Crosby (1981) included tetrachlorocatechol, tetrachloro-

resorcinol, and tetrachlorohydroquinone. Subsequent air oxidization products included chloranil, hydroxyquinones, and 2,3-dichloromaleic acid. The acid decomposed more slowly to carbon dioxide, chloranil, and unidentified organic fragments. Other products identified from the photoreduction of PCP included TTCPs, TCPs, and a cyclic diketone.

In 1979-80, the Institute of Ocean Sciences, Sidney, British Columbia, investigated the effect and fate of PCP in a pelagic marine ecosystem with the aid of Controlled Experimental Ecosystem enclosures (Yunker, 1981). The researchers concluded that photolytic breakdown of the dissolved pentachlorophenate was the only significant pathway for the removal of PCP from the enclosures. Results of sampling and analysis indicated that PCP was not adsorbed in significant amounts onto the enclosure walls or onto water particulates which would have been removed from the system as sedimented material. It was also concluded that the removal mechanism was independent of concentrations in the ranges studied, nominally 10 and 100 µg/L PCP respectively, since there was 32.8 and 33.1% respectively, of PCP remaining in the enclosures at day 25.

Boule et al. (1982) provided further insight into the photochemical behaviour of mono-CPs in dilute aqueous solution. For each mono-CP, the initial observation following irradiation is scission of a C-Cl bond and formation of HCl, with little or no kinetic influence from oxygen. The authors noted that the position of the chlorine on the ring strongly influences the transformation. In the molecular form, 2-CP is converted during irradiation into pyrocatechol, while in the anionic form photochemical dechlorination is followed by ring contraction to cyclopentadiene carboxylic acids which undergo subsequent Diels-Alder cycloaddition reactions.

Circumstantial evidence for photoreduction of PCP in the surface film of water has been provided by Fox and Joshi (1983). Results of their analyses for PCP and TTCP (2,3,4,6- plus 2,3,5,6-) in surface film and whole water samples from the CP-contaminated Bay of Quinte showed that TTCP:PCP ratios in surface film were consistently higher than those from whole water, i.e., 1.4 vs. 1.0. In addition, use of the TTCP:PCP ratios indicated to Fox and Joshi (1983) that some photoreduction of PCP occurred prior to the entry of the fugitive releases into the aquatic system or on the treated wood in storage at the wood preservation plant site adjacent to the Bay of Quinte. An initial TTCP:PCP ratio of approximately 0.1 in the technical PCP becomes 0.2 as the fugitive releases reach the Trent River a short distance from the Bay. At the mouth of the Bay, approximately 82 km from the point source, the ratio was approximately 1.0:1. Fox and Joshi (1983) did not find 2,3,4,5-TTCP in samples from any environmental compartment in their study. However, 2,3,4,6- and 2,3,5,6-TTCP, usually associated with

photodegradation of PCP, were found in environmental samples in enhanced amounts relative to PCP, which suggested to these researchers that the photolytic process dominated the reductive degradation of PCP in the Bay of Quinte.

10.4 Persistence

Bollag et al. (1980) demonstrated that cross-coupling between phenolic constituents of humus and 2,4-DCP can easily occur in the presence of an extracellular fungal laccase. The same research team investigated the lower molecular weight reactive intermediates, that is, the phenolic oligomers and quinones. These were formed initially by oxidation and oxidative coupling of the 2,4-DCP with phenolic humus constituents (Minard et al., 1981). They also went on to demonstrate the formation of hybrid oligomers when the fungal oxidase was incubated with 2,4-DCP and various halogenated anilines. They concluded from this aspect of their investigation that both enzymatic and nonenzymatic causes were responsible for the formation of cross-coupling products (Liu, S.-Y., et al., 1981).

Khan (1982) reviewed the current knowledge on bound pesticide residues in soils and plants as revealed by research which had used ^{14}C-labelled pesticides. He pointed out that the methodology for analysis of bound pesticides residues is still in the developmental stage. Khan (1982) suggested that, in addition to chemical binding, the pesticides may be physically bound to humic materials through adsorption on external surfaces and "entrapment in the internal voids of a molecular sieve-type structural arrangement." Additionally, the bound residues which may be released from organic soil through microbiological action would be available to plants and could then be bound up in the lignin of plants. Khan (1982) also concluded that bound residues may not present a potential environmental problem as long as such residues do not accumulate in toxicologically significant amounts.

Further to the above comments on the detoxification of chlorophenols and their related intermediate breakdown products, such as the chlorocatechols, Stott et al. (1983), who studied the fate of ^{14}C-labelled carbon atoms in 2,4-D and chlorocatechol in organic soils, stated the chlorocatechols were detoxified through polymerization within the soil environment through microbial enzyme action. They went on to state that many natural phenolic compounds which are produced in soil are toxic until they become bound into the humic acid. Stott et al. (1983) further stated that the chlorocatechols which had been linked into soil humic acid polymers were not likely to provide a future source of contamination.

The persistence of PCP and TTCP (2,3,4,6- plus 2,3,5,6-) in sediments has been illustrated in the results of analyses of sediment core samples from the Bay of Quinte. Fox and Joshi (1983) quantified these CPs in sediments, dated as to year of deposition in the Bay of Quinte via ^{210}Pb and ^{137}Cs. Sedimentation rates established via ^{210}Pb varied from a high of 1.8 ± 0.6 cm/yr, just offshore from the confluence of the Trent River and the bay to a more moderate rate of 0.8 ± 0.2 cm/yr at a site approximately 60 km down-bay, which had 23 m of overlying water. The upper half of the bay is much shallower with a depth of 5 m. Sediments deposited in the lower bay 29 years prior to sampling still contained 6 ppb PCP and 7 ppb TTCP (2,3,4,6- plus 2,3,5,6-). Analysis of TTCP:PCP ratios in Bay of Quinte samples by Fox and Joshi (1983) indicated that TTCPs degrade more slowly and thus are more persistent than PCP in water and sediments.

11 WASTE MANAGEMENT

11.1 Industrial Effluents

The management of liquid effluents from wood preservation operations, particularly those containing PCP, have been a concern of both private industry and governments. The results of a study carried out by Guo et al. (1979), of the Wastewater Technology Institute of Environment Canada, were briefly noted by Jones (1981). Based on the draft report by Guo et al. (1979) recommendations for management and treatment of effluent from wood preservation plants using PCP and oil were formulated by Guo et al. (1980) and Jank and Fowlie (1980) and included the following:

1) Wood preservation effluent containing PCP should be treated in an extended aeration activated sludge process.
2) The activated sludge system should be preceded by oil removal and flow equalization facilities.
3) The biological treatment should be followed by granular media filtration and granular activated carbon treatment since the activated sludge treatment by itself is incapable of removing all toxic substances, such as PCP and creosote.

To illustrate these recommendations Guo et al. (1980) noted that the effluent from the activated sludge treatment system during two monitoring periods contained an average concentration of 5.5 and 3.6 mg PCP/L of effluent while the corresponding numbers for phenol were 0.16 and 0.16 mg/L of effluent. When the effluent from the activated sludge process was further treated through granular activated carbon adsorption, then the combined treatment system reduced the level of PCP to 0.03 mg PCP/L of effluent and the phenol to 0.08 mg phenol/L of effluent.

In the United States, the EPA funded a project to investigate economically feasible processes for the treatment of wastewaters from wood preserving facilities. In a summary of the contractors report, Wallin et al. (1981) noted that two technologies yielded consistently high levels of treatment, i.e., removal of PCP to < 1.0 mg/L of wastewater. In one system, pH adjustment of the wastewater was followed by adsorption with bentonite clay and final polishing by the polymeric adsorbant XAD-4. In the second system, pH adjustment of the wastewater was followed by extraction with a mixture of No.2 fuel oil and a co-solvent such as still bottoms from amyl alcohol production. Both systems had to be preceded by fuel oil separation and flow equalization. Total annual operating costs for the treatment of 38 m^3/d (10 000 gpd) of wastewater were calculated

in 1980 to be $40 000 and $23 600 (U.S.) respectively for the two technologies, although operating parameters for the systems were not validated on a continuously flowing pilot-plant scale. The report noted that "The advantage of fuel oil extraction process is that the PCP can be removed from the wastewater without creating an additional waste and without bringing large capital and operating expense to bear on the wood preserver."

In the United States, the Clean Water Act of 1977 obliges industry to achieve by July 1, 1984, effluent limitation through the application of the "best available technology economically achievable" for control of toxic and nonconventional pollutants, such as PCP (U.S. EPA, 1981b). For the timber products industry the U.S. EPA issued final regulations in the Federal Register, January 26, 1981, which limit the discharge of pollutants into navigable water and publicly owned treatment works from existing and potential new sources. With respect to PCP, the regulations were directed at new sources rather than existing sources since the standards, already in effect, ensured significant reduction in the concentration of PCP in wood-preserving wastewaters. The new regulations provide for no discharge of process wastewaters from new plants in the following categories: Wood Preserving-Water Borne or Non-pressure, as well as Wood Preserving Steam and Boulton.

11.2 Water Quality

In November 1980, the Aquatic Ecosystem Objectives Committee (AEOC) of the Great Lakes Science Advisory Board of the International Joint Commission recommended the adoption of a water quality objective for pentachlorophenol into the 1978 Great Lakes Water Quality Agreement. The wording of the AEOC recommendation was as follows: "Pentachlorophenol in water should not exceed a concentration of 0.4 µg/L for the protection of aquatic life" (Fox, 1980). This recommendation was based on experiments that indicated growth inhibition of sockeye salmon under-yearlings at 1.74 µg PCP/L. Application of a safety factor of 0.2, which has been used on non-lethal but observable-effect concentrations for aquatic organisms, reduced the 1.74 µg PCP/L to the water quality objective number of 0.4 µg PCP/L (Fox, 1980).

In the United States, Ambient Water Quality Criteria have been developed by the Environmental Protection Agency for CPs and specifically for 2-CP, 2,4-DCP, and PCP, which are those which have been included in the list of 65 toxic pollutants under the U.S. Clean Water Act. The criteria, which are the basis for protection of aquatic life, are the best estimates by scientists of the maximum concentration of CPs that can be tolerated by aquatic life and will, when not exceeded, reasonably protect most, but not

all, aquatic life (United States Environmental Protection Agency 1980a, 1980b, 1980c, 1980d, 1980e). The criteria, as summarized in Table 7, were primarily for freshwater aquatic life since, with the exception of PCP, acute and chronic toxicity data for saltwater aquatic life were lacking (Jones, 1981. Table A4-6). In some cases where criteria were not provided, apparent threshold levels for other effects, such as flavour impairment, were included. The criteria, as issued under the U.S. Clean Water Act, section 304(a)(1), are non-regulatory, scientific assessments of ecological effects and only when they are adopted as State water quality standards do they have regulatory significance.

TABLE 7 SUMMARY OF UNITED STATES AMBIENT WATER QUALITY CRITERIA FOR CHLOROPHENOLS FOR PROTECTION OF AQUATIC LIFE

Compound	Reference	Aquatic environ.[1]	Acute toxicity[2] (µg/L)	Chronic toxicity[2] (µg/L)
2-CP	U.S. EPA 1980a	F	4 380	2 000[3]
4-CP	U.S. EPA 1980d	S	29 700	
2,4-DCP	U.S. EPA 1980b	F	2 020	365
2,4,6-TCP	U.S. EPA 1980d	F	970	
2,3,5,6-TTCP	U.S. EPA 1980d	S	440	
PCP	U.S. EPA 1980c	F	55	3.2
PCP	U.S. EPA 1980c	S	53	34

1 Aquatic environment: F - fresh water, S - salt water.

2 Acute and chronic toxicity occurs at concentrations as low as those presented, but can occur at lower concentrations among species or life-stages that are more sensitive than those tested.

3 No definitive data are available for chronic toxicity of 2-CP to sensitive freshwater aquatic life, but flavour impairment occurred in one species of fish at a concentration as low as 2 000 µg/L.

Most managers of water quality recognize that although concentrations of specific CPs in water may be below acute toxic levels, the total CP loading present, when added to other stress factors on the biota, may cause effects at chronic levels in the biota (Eder and Weber, 1980).

12 CURRENT CANADIAN RESEARCH

Research projects which are directly or indirectly related to CPs and which are currently underway or expected to receive funds in the 1983-84 fiscal year from the federal government are listed below. The list is not exhaustive since there may be related research on CPs underway or planned in the industrial and academic sectors which are supported in part by federal funds and of which we are not aware.

1) The development of thermal elution techniques for the recovery, analysis, and destruction of toxic organics from sludges, residues, and spent activated carbon. More specifically the project will develop a laboratory system capable of generating thermal destruction profiles for pure compounds and these compounds in environmental samples. The thermal profiles will be within the temperature range from ambient to 1200°C with residence times from 0.5-4.0 s. The compounds investigated will include PCP, 2,4,5-TCP, 2,4,6-TCP, and 2,3,5,6-TTCP, as well as two wood protection compounds containing CPs. Environmental samples to be included in the test are sludge samples and wood chip wastes from a wood preservation plant located in British Columbia.
2) A project on the development of removal techniques for specific contaminants from industrial effluents to examine the application of activated carbon for removal of CPs from wood protection/wood preservation wastewaters.
3) In British Columbia, the development of a Code of Good Practice for operation of wood protection facilities at sawmills and planer mills. This code will probably be used as a model for adoption in other parts of Canada.
4) In British Columbia, the development of a Code of Good Practice for operation of wood preservation plants.
5) In British Columbia, a study to determine the fixation of CPs in treated lumber exposed to simulated rain.
6) In British Columbia, a comprehensive study on leaching of contaminants from wood that has been pressure treated for wood preservation. Aspects to be investigated will include a mass balance of chemicals released, analysis of the preservatives and their impurities remaining in the treated wood, and the toxicity of the leachate.
7) Design and operational characteristics of a major wood waste burner in British Columbia to determine its adequacy for destruction of CPs.

8) In conjunction with (7), to monitor two full scale sawmill waste burners in B.C. which are fueled with wood wastes containing PCP residues.

9) An in-depth study and characterization of total effluents from a wood preservation plant.

10) An inventory of industrial and municipal effluents, which will include those from wood preservation plants.

11) Monitoring of technical CPs currently marketed in Canada to identify levels of active ingredients and their impurities.

12) A fish toxicity study of CPs.

13) In Ontario, a current program to monitor for CPs in wood shavings, poultry fat, and milk.

14) A project in environmental toxicology to study the effects of PCP at low concentrations on predator-prey behaviour using young large-mouth bass and the guppy as the test species.

15) An examination of the role of suspended solids in benthos uptake of organics, including CPs. The study area is the mouth of the Niagara River.

16) A laboratory model ecosystem study on the bioavailability of organic contaminants, including CPs, in sediments. The system will use Lake Ontario sediments and water plus the addition of oligochaetes from an uncontaminated source. Substrates analyzed for organics will include sediment, water, oligochaetes, and oligochaete feces.

17) An 85-day study on acute and sublethal effects at 100-1000 ppb of 2,4-DCP on fish eggs - larvae (eggs to 28 d after hatch).

18) Levels of CPs in leeches (Hirudinea) and waters from five New Brunswick rivers and streams. Sample sites are downstream from pulp and paper mills.

19) Levels of CPs and neutral chlorinated organics in leeches (Hirudinea) and water collected on a temporal basis from seven sites in the Grand River, Ontario.

20) Levels of CPs in water, sediment, and benthos collected from 12 sites in the Qu'Appelle River watershed in 1982.

21) A two-part study on the accumulation and effect of contaminants including CPs in aquatic biota:

    a) Studies to establish the potential of leeches (Hirudinea) as bio-indicators of contaminants in freshwater systems. Leeches will be compared with other sentinel species, especially molluscs, in terms of bioconcentration potential.

Samples will be obtained from several previously identified polluted sites on the Canagagigue Creek, Grand River, and Detroit River.

b) Pathways (food vs. water mechanisms) and accumulation rates of organic contaminants, including CPs and neutral chlorinated organics, in fish.

22) An investigation of the effects upon the Eastern oyster (*Crassostrea virginica*) of chronic exposure to sub-acute levels of pentachlorophenol and its by-products.

23) Further development of analytical methods for CPs in chicken liver and tallow.

24) Investigation of chlorinated compounds in chickens raised on PCP contaminated litter.

25) A federal interdepartmental "Risk/Benefit Analysis Committee" with participation by Agriculture Canada, Environment Canada and Health and Welfare Canada will examine chlorophenols as an initial project.

26) Water Quality Objectives for chlorinated phenols are under development by the Ontario Ministry of the Environment.

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APPENDIX 1 CORRIGENDA - Jones (1981)

Chlorophenols and Their Impurities in the Canadian Environment

Report EPS 3-EC-81-2 **March 1981**

Page	Correction
ii	Line 4. Delete "current (1979) and"
iii	Line 6. Change "dioxins" to "dioxines", "polychlorés" to "polychlorées", "dibenzofurans" to "dibenzofuranes"
xix	In List of Tables. Table 6: Change 1974 to 1977
xxxiii	Item 39. Should read: "PCP has been implicated in a few cases of industrial poisoning in Canada as the result of mishandling of this toxic material (App. 3, Sect. 3.2.3)."
xxxiii	In (43) change "organims" to "organisms"
5	Line 2. Add "acute" in "the . . . toxicity"
5	Line 3. Add "the acute" in "and . . . toxicity may also vary. . . ."
6	Line 12. Change "in the rat at 1.0 and 500 µg/kg/day," to "in the rat at 0.1 µg and 500 mg/kg/day,"
16	Line 4 of text. Change "sales figures for the U.S. " to "sales figures for PCP in the U.S."
16	Table 6. Heading: Change 1974 to 1977
17	Second last line from bottom. Change 19% to 9%
22	Line 15. Change "flourine" to "fluorine"
39	Line 4. Add) to Shields (1976)
61	Line 4. Change "coumpounds" to "compounds"
65	Line 17. Change "statment" to "statement"
68	Under Gaspereau and Shad change "alosa" to "Alosa"
77	Line 5. Change 2.4.6- to 2,4,6-
82	Line 22. Add the "x" in "To ic"
86	Line 14. Add) to "Swackhammer (1965)."

86 Line 22. Add) to "1978, 1979)."

106 Line 15. Add (to "resolved (Smith, ..."

106 Line 14. Add "x" to "complex"

108 Line 2. Add "x" in "he a"

108 Line 3. Add "z" in "chlorodiben o"

117 Reference Cluett, J. Pentic n should be Penticton

118 Line 13. Add "z" in "ionization"

119 Line 4. "Brerh." should be "Bremerh."

134 Table A1-1. Interchange the vapor pressure entry for 2,4-DCP and 2,4,6-TCP, so that the vapor pressure for 2,4-DCP=1 mm @ 53.0 and 2,4,6-TCP=1 mm @ 76.5

134 Table A1-1. Under Vapor Pressure delete C or °C in each entry

134 Table A1-1. Under Water Solubility move 2.1×10^{-1} from 3-CP to 4-CP

134 Table A1-1. Water Solubility for PCP should be 3.6×10^{-5}

134 Table A1-1. Under $pK^{c,e}$ add 5.3 for 2,3,4,6-TTCP

134 Table A1-1. Under pK^{d} add 5.46 for 2,3,4,6-TTCP

135 Table A1-3. Change "Dioxan" to "Dioxane"

136 Line 1. Add "α" to "with α-halo"

141 Item 2 b) underline "o"; item 2 c) underline "o,o'"

151 Line 27. Change "florosil" to "Florisil"

152 Line 9. Change "Florosil" to "Florisil"

188 Line 7. Change "HCDD (1.0 µg/kg/day)" to "HCDD (0.1 µg/kg/day)"

212 Table A4-3. Name change: "Lebistes reticulatus" to "Poecilia reticulata"

222 Table A4-3. Name change: "Lebistes reticulatus" to "Poecilia reticulata"

223 Table A4-3. Name change: "Lebistes reticulatus" to "Poecilia reticulata"

226 Table A4-3. Change "Fundulus similis" to "Fundulus similus"

231 Line 19. Name change: "Lebistes reticulatus" to "Poecilia reticulata"

234 Line 10. Change "similis" to "similus"

299 Line 11. Change "acutally" to "actually"

303 Fig. A7-7. In OCDD structural formula delete lines between inner ring and hexagon

317 Line 17. Change "conjugales" to "conjugates"

427 Line 27. Change "A plastic" to "Aplastic"

APPENDIX 2 CONCLUSIONS (reprinted from Jones, 1981)

Quantities in Commerce

1) Although approximately 3.4 million kg of chlorophenols (CPs) are used annually in Canada (Sect. 2), there has been a lack of specific commercial flow information for each of the CPs, including such data as:

   i) quantities of each CP produced in Canada, both for captive use and resale (Sect. 2.1.1),
   ii) quantities of each CP imported into Canada (Sect. 2.1.3),
   iii) quantities of each CP exported from Canada, and
   iv) quantities of each CP and their sodium salts used by the various industrial and agricultural sectors (Sect. 2.2).

Uses

2) Since uses for the CPs manufactured, imported, and marketed in Canada are based on the biological activitiy of CPs as bactericides, slimicides, fungicides, herbicides, and insecticides, they are regulated, in part, by Agriculture Canada under the Pest Control Products Act. Under the Food and Drug Act, Health and Welfare Canada regulates those products which contain CPs and which have health care and veterinary uses (Sect. 2.2.3).

3) The primary commercial use of the higher chlorinated CPs is for the prevention of wood decay. The relative importance to Canadian industry of all use claims, as approved by Agriculture Canada, other than those for wood treatment and wood preservation, are not well documented (Sect. 2.2.2, 2.2.3).

Composition of CPs

4) The chlorination of phenol to produce DCP, TTCP, and PCP, which is the currently used process in Canada and the U.S., is not a quantitative process. For example, a commercial grade PCP will contain some TTCP (Sect. 2.1).

Contaminants in Products

5) As a result of process chemistry, all higher chlorinated phenols, including trichlorophenol (TCP), tetrachlorophenol (TTCP), and pentachlorophenol (PCP), contain biologically active polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). The identities and quantities of these impurities in

Canadian produced CPs and in CPs imported from Europe are not well documented in contrast to the information on the impurities in CPs produced in the U.S. (Sect. 3.1).

6) The highly toxic 2,3,7,8,-TCDD has been reported only in 2,4,5-TCP but not in TTCP or PCP. (Sect. 3.1).

7) Some PCP imported from Europe had been produced by the alkaline hydrolysis of hexachlorobenzene (HCB) and, therefore, contained HCB as an impurity (Sect. 2.1).

Sources to the Environment

8) Wood treatment plants employing CPs are a major point source for CPs to enter the environment (Sect. 4.1). Although the number and the locations of pressure treatment plants in Canada are well documented, there exists minimal information on the location, size, and effluent treatment of dip-tank operations (Sect. 2.2.1, and App. 9).

9) Preservation of wood with excess amounts of oil-PCP can lead to PCP losses from the wood to the environment, particularly when the wood surface is in contact with large quantities of water (App. 6, Sect. 6.2.4).

10) Information on the release of CPs to the environment from production and processing point sources has been inadequate and, generally, has not examined the volume of fugitive releases during the processing and storage of treated material, nor the release of CPs to the air and water from wastes and effluents (Sect. 4.1, 4.2, and App. 9).

11) An unknown quantity of CPs enters the environment following their use in personal health care and veterinary products, and in sanitation products used in homes, hospitals, and on farms (Sect. 2.2.3).

12) CPs can form in the environment as a result of the interaction of aqueous chlorine with the appropriate organic molecule (Sect. 4.2.3).

Levels in the Environment - CPs

13) From the CP residue information that is available, there is every indication that CPs are ubiquitous in the Canadian environment. They have been detected in snow pack, water, landfill leachates, sewage effluent, sediment, and in aquatic and terrestrial organisms (Sect. 5.0).

14) There is a general lack of information on levels and sources of CPs in the surface waters of Canada (Sect. 5.1.1).

15) Recent studies on PCP residue levels in sediment and fish in the Great Lakes indicate that there has been widespread contamination of the Great Lakes Basin with PCP. The PCP levels can be related to the extent of urban and industrial development of individual watersheds (Sect. 5.1.2).

16) Limited information is available on residues of CPs in Canadian aquatic invertebrates and vertebrates (Sect. 5.1.4.1, and 5.1.4.2, respectively).

17) Studies on PCP metabolism in fish have identified the gall bladder as a highly useful organ as a qualitative monitoring aid for certain types of xenobiotics in water (App. 5, Sect. 5.2).

Levels in the Environment - PCDDs and PCDFs

18) Refined analytical techniques and equipment, developed during the 1970's, allow the detection of PCDDs and PCDFs in environmental samples at the ppt level (Sect. 6.1, 6.2, 6.3, 6.4, 6.5.2, and 6.6, and App. 2, Sect. 2.2). No data have been published to indicate the presence of PCDDs or PCDFs in the Canadian environment (Sect. 6.3, 6.4, 6.5, and 6.6), although fly ash from municipal incinerators in Ontario has been identified as a source of PCDFs and PCDDs (Sect. 6.2).

Levels in Human Environments - CPs

19) In the U.S., detectable levels of PCP have been identified in dairy herds exposed to PCP treated wood in total-confinement barns (Sect. 5.2.4). Information on possible similar situations in Canada is lacking.

20) In the U.S., PCP has been detected in human urine and seminal fluid in non-occupationally exposed persons (Sect. 5.2.5).

21) Food and feed have been contaminated with CPs during storage or transport (Sect. 5.2.6, 5.2.7).

22) Residues of CPs and chloroanisoles have been detected in livestock products, particularly poultry and eggs, where CP contaminated wood shavings were used for litter. The shavings have been a waste product from the wood processing industry (Sect. 5.2.2., 5.2.4).

23) At the present time in Canada there is no atmospheric data available for CPs (App. 6, Sect. 6.2.6).

Levels in Human Environment - PCDDs and PCDFs

24) PCDDs and PCDFs have been identified in fly ash, flue gas, air-borne particulates (Sect. 6.2, 6.3), and in food (Sect. 6.6).

Taste and Odour

25) CP compounds, when present in minute amounts in water, can cause taste and odour problems in the water and can ruin the flavor of fish (App. 4, Sect. 4.2.1.2).

Residue Analysis - CPs

26) Most published methods for the determination of CPs in trace quantities from environmental samples rely on the electron capture gc analysis of derivatization CPs coupled with appropriate extraction and clean-up techniques (App. 2, Sect. 2.1).

Residue Analysis - PCDDs and PCDFs

27) Few laboratories in either Canada or the U.S. have the capabilities for analysis for low ppt levels of PCDDs or PCDFs in environmental samples; therefore, this will be a limiting factor in the detection, quantification, and confirmation of PCDDs and PCDFs in environmental samples (App. 2, Sect. 2.2).

Mode of Action

28) The mode of action of PCP is not clearly understood, but PCP may be acting as an uncoupler of oxidative phosphorylation. The mammalian toxicity of CPs may, in part, be due to the perturbation of membranes (App. 5, Sect. 5.1).

29) The mode of action of PCDDs and PCDFs is unknown but they do affect several enzyme systems (App. 5, Sect. 5.1).

Metabolism

30) Studies on the metabolism of PCP in rats have shown that rapid dechlorination occurs (App. 5, Sect. 5.2).

31) Pentachlorophenyl-β-glucuronide is a metabolite of PCP in fish and mammals. Tetrachloro-*p*-hydroquinone, 2,3,4,5-TTCP, and tetrachloropyrocatechol have also been identified as metabolites of PCP in mammals, including man (App. 5, Sect. 5.2).

32) Some PCDDs of low chlorine content are metabolized in rats to mono- and dihydroxy derivatives (App. 5,Sect. 5.2).

33) No metabolites of TCDD have been identified. PCDD metabolism occurs exclusively via 2,3-epoxides; in TCDD these positions are blocked (App. 5, Sect. 5.2).

34) The half-lives of 2,3,7,8-TCDD in rats have been determined as 12 and 15 days for males and females, respectively (App. 3, Sect. 3.1.2.2).

35) The biological half-life of PCDFs in mice has been estimated to be two weeks (App. 5, Sect. 5.2).

Toxicology

36) The CPs are of toxicological significance to organisms in the environment. They exhibit increasing toxicity as the number of chlorine atoms substituted in the phenol ring increases (App. 3, Sect. 3.1.1.1).

37) The toxic effects of CPs on aquatic organisms are much greater than for terrestrial organisms (App. 4, Sect. 4.1.1, 4.1.2, 4.1.3).

38) The statistics that are maintained by various government agencies on the number of fish kills as a result of exposure to CPs may be unreliable, and the number of occurrences may be underestimated (App. 4, Sect. 4.2.1).

39) PCP has been implicated in a few cases of industrial poisonings in Canada as the result of mishandling of this toxic material (App. 3, Sect. 3.2.3).

40) Experimental evidence from test animals indicates that 2,3,4,6-TTCP caused only a minimal degree of fetotoxicity or embryotoxicity and was not teratogenic or embryolethal. PCP was not teratogenic but was highly embryolethal and embryotoxic. Experimental results also indicated a direct relationship between the presence of tumorigenic lesions in test animals and the isomeric structure of the CP (App. 3, Sect. 3.1.1.4).

41) Although not entirely conclusive, negative Ames test results indicated that there was little likelihood of DCP, TCP, TTCP, or PCP, being mutagenic (App. 3, Sect. 3.1.1.4).

42) Although the PCDDs and PCDFs include isomers which are highly toxic to organisms, for example 2,3,7,8-TCDD, others are much less toxic. The toxicity may be correlated in part with the degree of chlorination at the 2,3,7 or 8 positions (App. 3, Sect. 3.1.2.1).

43) There is little information available on the toxicity of PCDDs to aquatic organisms. In research on TCDD toxicity to fish there was an apparent delayed response, with duration of exposure less important than level of exposure (App. 4, Sect. 4.1.4).

44) The dioxin 2,3,7,8-TCDD is a known teratogen in mice and rats (App. 3, Sect 3.1.2.6).

45) Lifetime ingestion by Sprague-Dawley rats of 0.001 µg of 2,3,7,8-TCDD/kg body weight/day caused no effects which were of any toxicological significance; however, a higher rate of ingestion of TCDD, i.e., 0.1 µg 2,3,7,8-TCDD/kg/day, led to multiple toxicologic effects (App.3, Sect. 3.1.2.2).

46) A two-year feeding study with Sprague-Dawley rats being fed the equivalent of a weekly dose of 0.001 µg TCDD/kg body weight, led to an increase in incidence of certain types of tumors when compared with control rats (App. 3, Sect. 3.1.2.6).

47) The following CDDs have been tested for cytogenicity in rats: DD, 2,7-DCDD, and 2,3,7,8-TCDD. The evidence indicates that they are not cytogenic (App. 3, Sect. 3.1.2.6).

48) There has been positive evidence that at least one of the PCDDs, 2,3,7,8-TCDD, has immuno-suppression activity (App. 3, Sect. 3.1.2.5).

49) Research has shown that PCDDs are not alike in their toxicological properties. For instance, the symetrical 2,3,7,8-TCDD was highly embryotoxic in rats, whereas 1,2,3,4-TCDD was not embryotoxic to rats at doses as high as 800 µg/kg/day (App. 3, Sect. 3.1.2.6).

50) PCDDs have been identified as the compounds involved in cases of chick edema disease, and chloracne (App. 3, Sect.3.3).

Physicochemical Properties

51) Although the chemical and physical properties of the CPs have been adequately described (App. 1, Sect. 1.1.2), those of PCDDs and PCDFs are less well known (App.1, Sect. 1.2.2).

Chemodynamics

52) Mechanisms affecting environmental transport of CPs include adsorption (App.6, Sect. 6.2.1), diffusion and volatilization (App.6, Sect. 6.2.2), leaching (App. 6, Sect. 6.2.3), surface movement (App. 6, Sect. 6.2.5), and atmospheric movement (App. 6, Sect. 6.2.6).

53) Although CPs are water and soil contaminants, the moderate volatility (PCP 0.00011 mm Hg) of these compounds would suggest that atmospheric transport may be a significant route (App. 6, Sect. 6.2.6).

54) Both CPs and PCDDs are bioconcentrated in aquatic organisms (App. 8, Sect. 8.1, and 8.2, respectively).

55) In fish, the highest concentrations of CPs have been quantified in the following organs: gall bladder, liver, and gills (App. 5, Sect. 5.2).

56) CPs can be degraded in the environment through chemical (App. 6, Sect. 6.1.1), photochemical (App. 6, Sect. 6.1.2), and microbiological action (App. 6, Sect. 6.1.3). The relative rates at which the various actions may occur are influenced by such

factors as: 1) physical parameters of the media (e.g. in an aqueous solution of NaPCP, the photochemical reaction rate decreases as the pH is lowered (App. 6, Sect. 6.1.2)); 2) the energy available for the reaction (e.g. as the light intensity increases there is a concommittant increase in the velocity of the reaction (App. 6, Sect. 6.1.2)); 3) interdependence of one action on another (e.g. in PCP degradation in soil, chemical degradation is presumed to be caused and promoted by microbial action (App. 6, Sect. 6.1.1.2)).

57) Photodecomposition of PCP both in solution and in solid film, was shown to be a relatively unimportant mechanism for loss of PCP from the environment, in contrast to NaPCP which is unstable when exposed to UV irradiation (App. 6, Sect. 6.1.2).

58) Environmental generation of PCDDs and PCDFs can occur through a) photolysis of impurities in CPs (App. 7, Sect. 7.1.1), b) pyrolysis of wood products containing CPs (App.7, Sect. 7.1.2), and c) thermal generation by heating gases containing CP impurities (App. 7, Sect. 7.1.2).

59) Environmental degradation of PCDDs and PCDFs can occur through photolytic action (App. 7, Sect. 7.2.1), thermal degradation (App. Sect. 7.2.2), and rarely by microbial action (App. 7, Sect. 7.2.3).

60) PCDs are relatively immobile in soil. The main mechanism for movement of PCDD contaminated soil would be via surface erosion, or as sediment in water (App. 7, Sect. 7.3.1, 7.3.2).

61) PCDDs and PCDFs may be transported in air in plumes from incinerators, although there has been no monitoring activity (App. 7, Sect. 7.3.3).

62) Chemodynamic models have been a useful tool in the study of the fate of CPs and their impurities, including PCDDs, in aquatic, terrestrial-aquatic, and terrestrial ecosystems. Unfortunately, the models have provided only a limited amount of information (App. 8, Sect. 8.3).

Persistence

63) There is a lack of information on depletion and/or persistance of CPs in treated wood used in both freshwater and marine environments (App. 6, Sect. 6.2.3). There is also very limited information on the movement of CPs from treated wood, in-service, into the aquatic environment (App. 6, Sect. 6.2.4).

Waste Management

64) Technology is currently available for management of CP contaminated liquid and solid industrial wastes to reduce the CP content to environmentally safe levels (App. 9).

65) There has been little research on the use of deep-well disposal of liquid wastes containing CPs and their long-term effect on aquifers (App. 9).

APPENDIX 3 CHLOROPHENOL REGULATIONS IN CANADA

3.1 Products Containing Chlorophenols Registered under the Pest Control Products Act

Table A10-2 in JONES (1981) was a list of the products which contained CPs and which were registered as of April 1, 1980, under the Pest Control Products Act administered by the Canada Department of Agriculture. Since then there has been a substantial reduction in the number of products listed. Fifty-nine products have been discontinued. The reasons are twofold: 1) On January 1, 1981, Agriculture Canada suspended the uses of a number of products (Memorandum T-1-229. Jones, 1981); and 2) January 1, 1981, was a renewal date for all pesticide registrations. Thus some products no longer marketed had their registrations lapse.

As of February 1, 1983, there are 110 products registered to December 31, 1985, for the seven chlorophenol active ingredients listed. Of these, 73 are classified as COMMERCIAL and 37 as DOMESTIC (Table A3-1).

February 7, 1983
F. Cedar
P.A. Jones

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
TCH - 2,4,5-trichlorophenol							
11 990	C	DOW		Dowicide 2 Antimicrobial	SO	TECH 95	
STD - sodium trichlorophenate							
14 035	C	DRC		Biocide 207	SN	STD 17.30 NAB 7.45	SPC 14.10 IAL 10.00
14 036	C	DRC		Biocide 209	SN	STD 27.8 IAL 10.0	SPC 10.0
11 976	C	DOW		Dowicide 8 Antimicrobial	SO	STD 85	
TCP - tetrachlorophenol plus related chlorinated phenols							
12 801	C	RHC		49-167 Tetrachlorophenol	SO	TCP 94	
KTC - potassium tetrachlorophenate							
16 308	C	CHD		Permatox 180	SN		KTC 28.3
STC - sodium tetrachlorophenate plus sodium salts of other chlorophenols							
13 778	C	ALC		Alchem 4135 Fungicide Sap Stain Inhibitor	SN	STC 24	BTO 1.3
16 589	C	CAV		ML-21 Liquid for Control of Bacteria and Fungi	SN	NAB 7.45 SPC 14.10 STC 17.30	
9 933	C	CHD		Permatox 100 Liquid Fungicide Concentrate	SN	STC 22.82 PML 0.4	SMM 6.33
11 039	C	CHD		Chapco SSC Concentrate Liquid Fungicide for Lumber and Timber	SN	STC 22.82	SMM 13.23
13 585	C	DIM		Diatox	SN	STC 24.2	

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
STC - sodium tetrachlorophenate plus sodium salts of other chlorophenols (cont'd)							
10 924	C	VAR		VW and R Guardsman Stain Control - Woodbrite 24	SN	SPC 7.68 BNS 2	STC 16.32
14 874	C	WAB		18-600 Woodsheath Cherry Brown 10.0 IG	SU	STC 6.9	
15 976	C	WAB		18-528 Woodsheath Seabrite - 10.0 IG	SU	STC 14.2	
16 916	C	WAB		18-706 Tetra Concentrate 18.0 IG	SN	STC 21.93	
16 935	C	WAB		18-708 Woodsheath Clear 10.1 IG	SU	STC 13.59	
PCP - pentachlorophenol plus related active chlorophenols							
15 407	D	BEG	BPR	Behr Wood Preservative No. 91	SN	PCP 5.0	
10 792	D	BEN		Moorewood Penta Wood Preservative Clear 456-00	SN	PCP 4.8	
8 103	D	CAO		Bulldog Grip Wood Preservative Clear	SN	PCP 4.8	
12 392	C	CAO		Bulldog Grip Wood Preservative Clear	SN	PCP 4.8	
10 889	D	CBE		Mastercraft Clear Wood Preservative and Sealer	SN	PCP 2.85	
13 665	C	CEP		Penta-Mix Wood Preservative	SN	PCP 5	
3 267	C	CHD		Penta Preservative Concentrate 1 to 10 Wood Preservative Soil Poison	SN	PCP 36.3	TCP 5
8 150	C	CHD		Chapman Penta WR Concentrate 1-5	SN	PCP 22.2	TCP 1.2
8 168	C	CHD		Pol-Nu Pak Ground-Line Pole Treatment Bandage	PA	PCP 8.8	TCP 1.2
8 170	C	CHD		Pol-Nu Penta Preservative Grease for Ground-Line Treatment	PA	PCP 8.8	TCP 1.2
8 654	C	CHD		Timpreg Pak Pol-Nu Type Preservative Grease	PA	PCP 8.8 CRT 15	TCP 1.2 SFL 15
8 656	C	CHD		Timpreg Pol-Nu Type Preservative Grease	PA	PCP 8.8 TCP 1.2	CRT 15 SFL 15

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
PCP - pentachlorophenol plus related active chlorophenols (cont'd)							
10 617	C	CHD		Timpreg B Pol-Nu Type Wood Preservative Grease	PA	PCP 8.8 BNA 15.5	TCP 1.2
12 038	C	CHD		Timpreg B (Special) Wood Preservative Grease	PA	PCP 8.8 CRT 15.5	TCP 1.2 BNA 15.50
12 163	C	CHD		PQ-10 Liquid Fungicide for Lumber and Timber	EC	CUQ 5.0 TCP 2.4	PCP 17.6
14 904	C	CPT		Goodyear Penta Wood Preserver	SN	PCP 5.0	
14 905	C	CPT		Goodyear Wood Preserver Black	SN	PCP 5.0	
17 129	C	CUB	CAX	Cuprinol Penta No. 2	SN	PCP 4.75	
17 130	C	CUB	CAX	Cuprinol Penta No. 2 (WR)	SN	PCP 4.75	
15 341	C	DIV		Kleen-Phene Disinfectant	SN	BCP 1.79 PCP 0.46 SLS 4 TCP 0.063	IAL 10 PHA 1.02 SXS 1 TNM 1.17
8 404	C	DOO		CCC Pentol Wood Preservative for Field Cuts	SN	PCP 5	
12 534	C	DOO		Domtar Pentachlorophenol - Industrial Wood Preservative	GR	PCP 96	
11 974	C	DOW		Dowicide EC-7 Antimicrobial	GR	PCP 88	TCP 12
14 120	D	DUK		Woodsol Paintable Penta Clear	SN	PCP 4.8	
7 270	D	DUR		Wood Preservative Clear	SN	PCP 5	
14 054	C	DUR		Wood Preservative Clear	SN	PCP 4.85	
15 036	D	GHC		Protox Clear (Clair)	SN	PCP 5	
17 006	D	GHC		Protox Preservatif Pour Bois - Brun	SN	PCP 5.0	
9 110	D	HOS		Super Solignum 10-10 Clear Wood Preservative	SN	PCP 4.8	
12 510	D	HOS		Super Solignum Wood Preservative Stain 10-14, Walnut	SN	PCP 3.1	

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
PCP - pentachlorophenol plus related active chlorophenols (cont'd)							
12 512	D	HOS		Super Solignum Wood Preservative Stain 10-16, Teakwood	SN	PCP 3.1	
12 513	D	HOS		Super Solignum Wood Preservative Stain 10-15, Black	SN	PCP 3.1	
12 514	D	HOS		Super Solignum Wood Preservative Stain 10-200, Bungalo White	SN	PCP 3.1	
12 515	D	HOS		Super Solignum Wood Preservative Stain 10-68, Straw	SN	PCP 3.1	
12 516	D	HOS		Super Solignum Wood Preservative Stain 10-66, Drift Wood	SN	PCP 3.1	
12 518	D	HOS		Super Solignum Wood Preservative Stain 10-63, Dark Brown	SN	PCP 3.1	
12 519	D	HOS		Super Solignum Wood Preservative Stain 10-62, Brunswick Green	SN	PCP 3.1	
12 520	D	HOS		Super Solignum Wood Preservative Stain 10-23, Mahogany	SN	PCP 3.1	
12 521	D	HOS		Super Solignum Wood Preservative Stain 10-22, Cedar	SN	PCP 3.1	
12 522	D	HOS		Super Solignum Wood Preservative Stain 10-21, Redwood	SN	PCP 3.1	
6 948	D	LAT		Later's Pentachlorophenol SN Ready-to-use Wood Preservative	SN	PCP 5	
6 950	C	LAT		Later's Pentachlorophenol Wood Preservative 1-10 Liquid Concentrate	SN	PCP 40	
10 320	D	LAV		Laurentide Paint Wood Preservative Clear G-14	SN	PCP 4.8	
11 713	D	LEG		Rez Penta Wood Preservative Clear	SN	PCP 5	
11 714	D	LEG		Rez Penta Wood Preservative Green	SN	PCP 5	
6 410	D	NNP		Tim-Ber-Lox Fungicide Wood Preservative Green 4410	SN	PCP 4.75	

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
PCP - pentachlorophenol plus related active chlorophenols (cont'd)							
10 369	D	NNP		Tim-Ber-Lux Fungicidal Wood Preservative Clear 4413	SN	PCP 4.75	
12 374	D	OSD		Pentox Penta Green Wood Preservative	SN	PCP 5	
13 636	D	OSD		Pentox Wood Preservative Brown	SN	PCP 3.85	
17 047	C	OSD		Pentox 1 + 10 Penta	SN	PCP 40	
14 482	C	POS	FIT	24-12 Wood Preservative solution	SN	PCP 5	
14 077	C	POS	FIT	Osmose Osmoplastic Wood Preserving Compound	SU	CRT 20 KDC 3.1 SFL 43.7	DNP 2 PCP 2.21
16 864	C	POS	FIT	Osmoplastic - B Wood Preservative Compound	PA	BNA 15 CRT 15 PCP 10	
14 095	C	REB		Penta Preservative 1-10	SN	PCP 36.3	TCP 5
9 535	D	REC		Penta-Phenol Clear Paintable Wood Preservative and Primer-Sealer	SN	PCP 4.8	
12 800	C	RHC		RCL 49-162 Pentachlorophenol for Manufacturing Purposes Only	GR	PCP 96	
16 395	D	ROC		Woodlife Liquid Water Repellent Wood Preservative	SN	PCP 4.8	
8 227	D	ROR		ROZ-TOX Clear Wood Preservative and Sealer	SN	PCP 2.85	
10 633	C	SAJ		Sanitized Van Interior Aerosol	PP	PCP 0.1 MGK 1.67	PYR 0.5 PBU 1
16 490	C	SAJ		United Van Lines Sanitized Van Interior Spray	PP	MGK 1.67 PBU 1.0	PCP 0.1 PYR 0.5

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
PCP - pentachlorophenol plus related active chlorophenols (cont'd)							
8 789	C	STD		Stangard Penta Wood Preservative Concentrate 1-10	SN	PCP 41	
8 791	C	STD		Stangard Paintable Penta Clear Wood Preservative	SN	PCP 5	
8 799	C	STD		Stangard Penta WR Wood Preservative Concentrate 1-4	SN	PCP 21	
8 801	C	STD		Stangard Penta WR Water Repellent Wood Preservative	SN	PCP 5	
11 774	C	STD		Stangard Penta Green Wood Preservative	SN	PCP 5	
13 008	D	STD		Stangard Paintable Penta Clear Wood Preservative	SN	PCP 5	
13 010	D	STD		Stangard Penta WR Water Repellent Wood Preservative	SN	PCP 5	
13 091	D	STD		Stangard Penta Green Wood Preservative	SN	PCP 5	
13 618	C	STD		Stangard Penta Grease 10 Groundline Wood Preservative	PA	PCP 10	
15 987	C	STN		Horntox Clear Wood Preservative	SN	PCP 0.06 ZNN 2.0	
15 988	C	STN		Horntox Green Wood Preservative	SN	CUN 2.0 PCP 0.06	
3 608	C	TEI		Nevarot Water Repellent Wood Preservative	SN	PCP 4.75	
15 143	C	TIR	BAO	Pole Topper Fluid Wood Preservative	SN	PCP 8.8	TCP 1.2
15 144	C	TIR	BAO	Osmoband Wood Preservative Bandage	PA	CRT 15 SFL 20	PCP 8.8 TCP 1.2
16 915	C	TIR	BAO	PCP 1 to 10 Concentrate Wood Preservative	SN	PCP 35.86 TCP 4.17	
17 039	C	UNR		Uniroyal 17 039 Pentachlorophenol Oiled	SO	PCP 96	
10 925	C	VAR		Guardsman Penta Preservative 1-10	SN	PCP 43	
12 303	C	VAR		Guardsman Penta Preservative	SN	PCP 4.25	

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
PCP - pentachlorophenol plus related active chlorophenols (cont'd)							
12 319	C	VET	ARH	Mystox LSE Bacteriostatic and Fungistatic Additive	EC	PCF 25	PCP 0.5
16 917	C	WAB		18-116R Wood Sealer T-678 Gold	SU	PCP 4.39	
14 204	D	WEW		Woodlife Liquid Water Repellent Wood Preservative	SN	PCP 5	
14 205	C	WEW		Woodlife Liquid Water Repellent Wood Preservative	SN	PCP 5	
14 206	C	WEW		Woodlife 3:1 Concentrate Wood Preservative	SN	PCP 16.3	
SPC - sodium pentachlorophenate plus sodium salts of other chlorophenols							
13 483	C	BEZ		Betz Slimicide A-9	SN	SPC 27.6 SDD 4.0 IAL 10	STD 9.1 QAC 5.0
8 146	C	CHD		Chapman Permatox 10-S	SG	BNS 57	SPC 36
15 574	C	CHD		Napclor-S Antimicrobial	GR	SPC 90	
12 867	C	DEC		Dearcide 712 Liquid Cooling Water Microbistat	SN	SPC 32	STD 8.0
11 992	C	DOW		Dowicide G-ST Antimicrobial	SG	SPC 90	SHO 1.5
16 841	C	NAC		Chem-Aqua 400	SN	SPC 5	
17 005	D	PAA	JOB	Ready-To-Use Moss Stop	SN	SPC 3.6	
13 297	C	PBA		Slimicide Formula Y-100 Pellets	PE	SPC 90	
14 076	C	SAN		Sanfax Pinefax Liquid Disinfectant	SN	IAL 13.5 TYT 2.66 SBC 1.1 SXS 2.8	POI 5 SPC 0.55 SBD 2.81
13 955	C	SAT	SAJ	Sanitized Brand SPI	SN	SPC 10	

TABLE A3-1 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF FEBRUARY 1, 1983 (cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
SPC - sodium pentachlorophenate plus sodium salts of other chlorophenols (cont'd)							
16 863	C	TIR	TIS	Patox Pole Treating Wrap Type I	IF	CRT 11.0 KCR 12.5 SFL 37.1	SPC 8.5 STC 1.0

1 Registration Number
2 Market (C = Commercial, D = Domestic)
3 Registrant (Section 2.2.1)
4 Agent (Section 2.2.2)
5 Formulation (Section 2.2.4)
6 Guarantee (% Active Ingredient, unless otherwise stated).

Code definitions (Section 2.2.3).

3.2 Codes- Pest Control Products Act Product Registrations

3.2.1 Chlorophenol Products Registrants

Code	Registrant Name and Address
ALC	Alchem Ltd., P.O. Box 5002, 1055 Truman St., Burlington, Ont. L7R 3Y9
BEG	Behr Process Corp., Box 1287, 1603 W. Alton Ave., Santa Ana, Calif. 92702 U.S.A.
BEN	Benjamin Moore and Co. Ltd., 15 Lloyd Ave., Toronto, Ontario M6N 1G9
BEZ	Betz Laboratories Ltd., 1173 Teron Rd., P.O. Box 13020, Kanata, Ottawa, Ontario K2K 1X3
CAO	Canadian Adhesives Ltd., 420 Marien Ave., Montreal East, Que. H1B 4V6
CAV	Canadian Germicide Co. Ltd., 591 The Queensway, Toronto, Ont. M8Y 1J8
CBE	Canadian Tire Corp. Ltd., 2180 Yonge St., Toronto, Ont. M4W 2H3
CEP	Century Paint & Wallpaper Ltd., 1514 Merivale Rd., Ottawa, Ont. K2G 3J6
CHD	Chapman Chemical (Canada) Ltd., Suite 3900 - 1155 Dorchester Blvd. W., Montreal, Que. H3B 3V2
CPT	Consolidated Protective, Coatings Ltd., 2300 Schenker St., LaSalle, P.Q. H8N 1A2
CUB	Cuprinol Ltd., Adderwell, Frome, Somerset, England
DEC	Dearborn Chemicals, 3451 Erindale Station Rd., Mississauga, Ont.
DIM	Diachem of B.C. Ltd., 5289 Regent St., Burnaby, B.C. V5C 4H4
DIV	Diversey (Canada) Ltd., 2645 Royal Windsor Dr., Clarkson Postal Station, Mississauga, Ont. L5J 1L1
DOO	Domtar Chemicals Ltd., Wood Preserving Div., 395 de Maisonneuve Blvd. W., Montreal, Que. H3A 1L9
DOW	Dow Chemical of Canada, Ltd., P.O. Box 1012, Hgwy. 40, Sarnia, Ont. N7T 7K7
DRC	Drew Chemical Ltd., 1 Drew Court, Ajax, Ont. L1S 2E5
DUK	Dussek Bros. (Canada) Ltd., P.O. Box 385, Belleville, Ontario K8N 5A5
DUR	Dural Products Ltd., 550 Marshall Ave., Dorval, Que. H9P 1C9
GHC	Gibson - Homans of Canada Ltd., 101 de la Berre, Boucherville, Que. J4B 2X6
HOS	House of Sturgeon (National) Ltd., 200 Norelco Dr., Weston, Ont. M9L 1S4
LAT	Later Chemicals Ltd., 12080 Horseshoe Way, Richmond, B.C. V7A 4V5
LAV	Laurentide Chemicals Inc., 4650 12e Ave., Shawinigan-Sud, Que. G9N 6V9
LEG	Lepage's Ltd., 50 West Dr., Bramalea, Ont. L6T 2J4

NAC	National Chemsearch of Canada Ltd., 245 Orenda Rd., Bramalea, Ont. L6T 1E7
NNP	Northern Paint Co. Ltd., 394 Gertrude Ave., Winnipeg, Man. R3L 0M6
OSD	Osmose Wood Preserving Co. of Canada Ltd., 1080 Pratt Ave., Montreal, Que. H2V 2V2
PAA	Pace National Corp., 500 - 7th Ave. S., Kirkland, WA. 90833 U.S.A.
PBA	Perolin-Bird Archer Ltd., 100-2nd St., Cobourg, Ont. K9A 4M2
POS	Pole Sprayers of Canada Ltd., 980 Ellicott Street, Buffalo, N.Y. 14209 U.S.A.
REB	Record Chemical Co. (Western) Ltd., 3905 E. 1st Ave., Burnaby, B.C. V5C 3W3
REC	Record Chemical Co. Inc., 840 Montee de Liesse Rd., Montreal, Que. H4T 1N8
RHC	Reichhold Chemicals Ltd., P.O. Box 130, Port Moody, B.C. V3H 3E1
ROC	Roberts Co. Canada Ltd., 2070 Steels Rd., Bramalea, Ont. L6T 1A7
ROK	Robinson and Webber Ltd., 1569 Orange St., Winnipeg, Man. R3E 3B5
ROR	Frank T. Ross and Sons 1962 Ltd., Box 248, West Hill, Ont. M1E 4R5
SAJ	Sanitized Process (Canada) Ltd., Ste. 1700, 2200 Yonge St., Toronto, Ont. M4S 2C6
SAN	Sanfax Industries Ltd., 1650 South Service Road, Dorval 760, Que. H9P 1H9
SAT	Sanitized Incorp., 605-3rd Ave., New York, New York, 10016 U.S.A.
STD	Stan Chem Ltd., 681 Plinquet ST., Winnipeg, Man. R2J 2X2
STN	Sternson Ltd., 22 Mohawk St., P.O. Box 130, Brantford, Ont. N3T 5N1
TEI	Texas Refinery Corp. of Canada Ltd., 25 Industrial St., Toronto, Ont. M4G 1Z2
TIR	Timber Specialties Div., Pole Sprayers of Canada Ltd., 980 Ellicott St. Buffalo, N.Y. 14209, U.S.A.
UNR	Uniroyal Chemical, Div. of Uniroyal Ltd., Erb St., Elmira, Ont. N3B 3A3
VAR	Van Waters and Rogers Ltd., P.O. Box 2009, Vancouver, B.C. V6B 3R2
VET	Ventron Corporation, Congress Street, Beverly, Mass. 01915 U.S.A.
WAB	Walker Bros., ltd., 5684 Beresford St. Burnaby, B.C. V5J 1J2
WEW	Weldwood of Canada Ltd., 1055 W. Hastings, Vancouver, B.C. V6D 3V8

3.2.2 Registrants' Canadian Agents

Code	Agent and Address
ARH	LK Archer, 407 Oakdale Crescent, Thunder Bay, Ont.
BAO	W.E. Bateman, 347 Bay St., Suite 304, Toronto, Ont. M5H 2R8

BPR	Behr Process of Canada, 4624 - 11th St. N.E., Calgary, Atla. T2E 2W7
CAX	Hoechst Canada Inc., 100 Tempo Ave., Willowdale, Ont. M2H 2N8
FIT	Art W. Fish. P.O. Box 88, Bonnie Dr. Route 1, Winfield, B.C. V0H 2C0
JOB	Joseph Chan, 1111 Beach Ave., Ste. 2005, Vancouver, B.C.
SAJ	Sanitized Process Canada Ltd., Suite 1700, 2200 Younge St., Toronto, Ont. M4S 2C6

3.2.3 Chlorophenol Products Active Ingredients

Code	Active Ingredient
BCP	*o*-benzyl-*p*-chlorophenol
BNA	borax, anhydrous
BNS	borax
BTO	bis (tri-*n*-butyltin) oxide
CRT	creosote
CUN	copper as elemental, present as copper naphthenate
CUQ	copper - 8 - quinolinolate
DNP	dinitrophenol
IAL	isopropyl alcohol
KCR	potassium chromate
KDC	pottassium dichromate
KTC	pottassium tetrachlorophenate
MGK	*n*-octyl bicycloheptene dicarboximide
NAB	nabam
PBU	piperonyl butoxide (technical)
PCF	fatty acid esters of pentachlorophenol (e.g. pentachlorophenol laurate)
PCP	pentachlorophenol
PHA	phosphoric acid
PML	phenylmercuric lactate
POI	pine oil
PYR	pyrethrins
QAC	n-alkyl (50% C14, 40% C12, 10% C16) dimethyl benzyl ammonium chloride
SBC	sodium *o*-benzyl-*p*-chlorophenate
SBD	sodium *p*-tert-butylphenate
SDD	sodium dimethyldithiocarbamate
SFL	sodium fluoride

SHO	sodium hydroxide
SLS	sodium lauryl sulfate
SMM	sodium metaborate octahydrate
SPC	sodium pentachlorophenate
STC	sodium tetrachlorophenate
STD	sodium trichlorophenate
SXS	sodium toluene sulfonate, or sodium xylene sulfonate
TCH	2,4,5-trichlorophenol
TCP	tetrachlorophenol
TNM	trisodium nitrilotriacetate monohydrate
TYT	tetrasodium ethylenediaminetetraacetate
ZNN	zinc as elemental, present as zinc naphthenate

3.2.4 Formulations

Code	Chlorinated Phenol Products Formulations
EC	emulsifiable concentrate
GR	granular
IF	impregnated fabric
PA	paste
PE	pellet
PP	pressurized product
SG	soluble granule
SN	solution
SO	solid
SU	suspension

APPENDIX 4 **HISTORICAL REVIEW: An Annotated Chronology of Chlorophenol Incidents and Regulatory Action - A Canadian Perspective**

1941 - The initial registration of a wood preservation product containing PCP was granted by the Canada Department of Agriculture to A.D. Chapman and Co., Inc. for Permatox 10S, Registration No. 1170.

May 12, 1971 - Meat Inspection Circular No. 111, Health of Animals Branch, Agriculture Canada, entitled "Use of Pentachlorophenols in Treating Hides." Recommendation: to stop all use of PCP products for preserving hides.

October, 1973 - Dow Chemical of Canada Ltd., closes PCP plant at Ft. Saskatchewan, Alberta.

April, 1974 - Sampling program set up to investigate dioxin content in PCP. Centered mainly on detection of the 2,3,7,8-TCDD isomer. No positive results found.

August, 1975 - Odour problem in B.C. mills regarding application of PCP sapstain chemicals.

April, 1976 - Environmental Contaminants Act promulgated.

June, 1976 - Request from Environment Canada for list of problem chemicals for consideration as Priority Chemicals under the Environmental Contaminants Act. Chlorophenols suggested by many agencies/depts, including Agriculture Canada.

Fall, 1976 - Chlorophenols scheduled for in-depth re-evaluation by Agriculture Canada.

Feb.-March, 1977 - Rail transportation incident regarding feed oats contaminated with PCP. Ritchie Feed & Seed, Ottawa, Ontario.

Spring, 1977 - PCP contamination of Michigan cattle. PCP pressure treated wood used in construction of barns and to treat other wooden structures on farm.

March, 1977 - First List of Priority Chemicals released by Environment Canada. Chlorophenols Listed in Category III., which is the category for those substances which may pose a significant danger to human health or the environment and about which further information is required.

September, 1977 - Tallow oil/feed ration problem in Quebec. Analysis of the feed ingredients showed the tallow oil to contain 0.3-0.4 ppm PCP and 0.03 ppm OCDD. A raw hide processor was using an unregistered chlorophenol antimicrobial agent for the prevention of the deterioration of hides and treating solutions during pretanning and tanning steps. Product withdrawn from use.

November 15, 1977 - Environment Canada initiates a technical review of the environmental aspects of the class of chemicals, chlorophenols and their impurities.

March-April 1978 - Monsanto PCP manufacturing plant at Sauget, Illinois, closed.

August, 1978 - Monsanto PCP manufacturing plant at Newport, Wales (U.K.), closed.

October, 1978 - Investigation initiated regarding "off flavour" musty odour of Ontario broiler chickens associated with the presence of PCP residues in litter/bedding (wood shavings).

October 18, 1978 - U.S. EPA RPAR Notice issued (PD 1).

October 27, 1978 - PCP Spill - seepage into ground water, Pentiction, B.C.

January 5, 1979 - Agriculture Canada memo to basic manufacturers of the chlorophenols noting regulatory status under review during 1979 and 1980.

January 10, 1979 - Agriculture Canada memo to federal and provincial agencies requesting comments on the chlorophenols.

January 1979 - Regulatory action by Agriculture Canada: (1) suspension of pentachlorophenol as a wood preservative for use in the interior of poultry houses; (2) suspension of PCP as a disinfectant and insecticide to kill chicken mites in poultry houses; and (3) limitation of the use of PCP and SPC in leather tanning operations. Limitation: Do not use in the pretanning operations of curing, liming, soaking and pickling from which by-product fats result.

August 7, 1979 - First Agriculture Canada memo to registrants (R-1-79) issued regarding notification that the regulatory status of products containing chlorophenols were subject to review during 1979 and 1980.

August 9, 1979 - The penultimate draft of the Environment Canada document on chlorophenols was distributed to the Department of the Environment/National Health and Welfare Environmental Contaminants Committee (DOE/NH&W ECC) on August 3, 1979. Based on recommendations which accompanied the report the committee recommended that additional information on the commercial flow of chlorophenols in Canada should be obtained through a notice issued under the Environmental Contaminants Act.

September 24, 1979 - Second Agriculture Canada memo to Registrants (R-1-79) outlining proposed suspensions of uses, new limitations and new cautionary statements for labels of products containing chlorophenols.

December 1, 1979 - Chlorophenols listed in Category II of the DOE/NH&W Environmental Contaminants Act Priority Chemicals - 1979. Category II is for those substances which are being investigated to determine the nature and the extent of the danger to human health or the environment and the appropriate means to alleviate that danger.

January 21, 1980 - Commencing on this date notices under Paragraph 4(1)b of the Environmental Contaminants Act were sent out by Environment Canada, initially to Canadian manufacturers of chlorophenols, then to wood preservers and finally in August, 1980, to Pest Control Products Act registrants of products containing chlorophenols, and to the registrant's distributors. The purpose was to obtain information on the commercial flow of chlorophenols in Canada.

May 1, 1980 - The final draft of the Environment Canada technical review on chlorophenols was transmitted to the Chairman of the DOE/NH&W ECC.

June 26, 1980 - Regulatory action, Health and Welfare Canada. Section B.01.046(1) of the Food and Drug regulations amended to add "(p) chlorinated dibenzo-*p*-dioxins." Thus, a food is considered adulterated if any quantity of any chlorinated dioxin is found to be present.

June, 1980 - Dow Chemical of Canada Ltd. closes chlorophenoxy plant at Ft. Saskatchewan, Alberta. Plant dismantled by December, 1980.

September, 1980 - PCP contamination of wood shavings; broiler chicken production problems in Quebec.

September, 1980 - PCP contamination of poultry feed, Winkler, Manitoba, due to use of chlorophenol antimicrobial in production of tallow oil.

October, 1980 - Dow PCP manufacturing plant at Midland, Michigan, closes.

October, 1980 - The British Columbia Federal-Provincial Wood Protection Task Force was set in place. The Task Force, which is still functional, is composed of representatives of federal and provincial agencies, the B.C. forest industry, and labour unions. Under their sponsorship a Code of Good Practice incorporating Best Practicable Control Technology is being developed for the design and operation of lumber treatment facilities for the control of sapstain and mold fungi.

November 28, 1980 - Trade memo (T-1-229) released by Agriculture Canada, titled "Changes in the Regulatory Status of the Chlorophenols."

January 1, 1981 - Regulatory Action by Agriculture Canada: (1) suspension of chlorophenol products carrying label instructions for use as wood preservatives and/or wood stains for INTERIOR home use; (2) suspension of products containing sodium pentachlorophenate (SPC) for use as fungicides in mushroom houses and on tools for mush-

room culture; (3) suspension of products containing PCP for use as wood preservatives on wooden food containers and on horticultural lumber; (4) suspension of products containing PCP for use as wood preservatives on above-ground interior woodwork of farm buildings; (5) suspension of all chlorophenol products carrying label instructions for use as microbiocides in curing hides; (6) suspension of products labelled for use as herbicides and soil sterilants, except those labelled for destruction of moss on roofs; (7) suspension of chlorophenol products for use as slimicides in pulp and paper mill operations; (8) suspension of all DOMESTIC class products for application by SPRAY methods.

January 6, 1981 - News Release by Agriculture Canada, entitled "Use of Chlorophenols Restricted."

January, 1981 - U.S. EPA RPAR document (PD 2/3) on Wood Preservatives released.

June 4, 1981 - Environment Canada document "Chlorophenols and Their Impurities in the Canadian Environment" released publicly. The French translation became available in December, 1981.

June 16, 1981 - The draft Environment Canada recommendations of August 3, 1979, concerning the collection of further information and development of controls for chlorophenols were modified in light of the January 1, 1981, regulatory action by Canada Department of Agriculture. The revised draft recommendations were developed for future discussion by the DOE/NH&W Toxic Chemicals Committee.

October 20, 1981 - Canada Safety Council releases Hazard Warning Notice on Pentachlorophenol (PCP).

November 19, 1981 - The first meeting of the DOE/NH&W Ministers Advisory Committee on Dioxins. The membership of this Committee, formed under subsection 3(4) of the Environmental Contaminants Act, is composed of persons from outside Government, who are to advise the Ministers on the risks to humans and non-human targets associated with the presence of chlorinated dioxins in the environment.

January 4, 1982 - NRC (Canada) Dioxin documents released: (1) Polychlorinated Dibenzo-*p*-dioxins: Criteria for Their Effects on Man and His Environment" (2) "Polychlorinated Dibenzo-*p*-dioxins: Limitations to the Current Analytical Techniques".

May 10, 1982 - NRC (Canada) document, "Chlorinated Phenols: Criteria for Environmental Quality" released.

March 10, 1983 - The first draft of the Health and Welfare Canada report "Chlorophenols and Their Impurities: A Health Hazard Evaluation",

prepared in the Bureau of Chemical Hazards was distributed to the DOE/NH&W Toxic Chemicals Committee.

September 15, 1983 - The final draft of the 1983 supplement to the Environment Canada report "Chlorophenols and Their Impurities in the Canadian Environment" was completed.

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