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Chlorophenols and Their Impurities in the Canadian Environment

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Environmental Impact Control Directorate
March 1981

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CHLOROPHENOLS AND THEIR IMPURITIES IN THE CANADIAN ENVIRONMENT

by

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ABSTRACT

As one of the substances in Category II of Environment Canada's List of Priority Chemicals, the chlorophenols (CPs) have been reviewed to bring together, from various sources, detailed information on their entry, presence, transport, fate, and effects in the Canadian environment. This technical review has examined not only the CPs but also their impurities, primarily the polychlorinated dibenzo-p-dioxins (PCDDs) and the polychlorinated dibenzofurans (PCDFs).

Approximately 3.4 million kg of CPs are used annually in Canada. Their uses, which depend on their biological activity, range from preservatives in woods and paints, to anti-microbials in industrial cooling systems and in papermaking. In Canada, CPs are classed as pesticides and, as such, their industrial and agricultural uses are regulated by Agriculture Canada under the Pest Control Products Act.

Although there are eight CPs which have commercial acceptance, only 2,4-dichlorophenol (2,4-DCP), 2,3,4,6-tetrachlorophenol (2,3,4,6-TTCP), and pentachlorophenol (PCP) are manufactured in Canada; the remainder are imported. The sodium salts of TTCP and PCP are formulated in Canada, as well as being imported.

Entry of CPs into the environment has occurred at the sites where CPs have been used, such as wood treatment plants and pulp and paper plants. CPs can be generated as a result of aqueous chlorination of organic compounds during treatment of potable water and wastewater and can also occur as metabolites of chlorobenzenes.

CPs are ubiquitous in Canada. They have been identified in samples of water, snow melt, sediment, aquatic biota, and agricultural produce, as well as in man.

The chemistry of the CPs, PCDDs and PCDFs has been reviewed, as has the current methodology for analysis for these compounds. Information on the acute and chronic toxicity and toxicology of these compounds in terrestrial and aquatic organisms has also been presented. Although all toxicological effects cannot be directly attributable to particular CP, PCDD, or PCDF isomers, they have been identified in this review as such when and as reported in the literature.

The fate of the CPs, PCDDs, and PCDFs in the Canadian environment are of concern and have been reviewed under such headings as generation, degradation, transport, bioconcentration, and modelling.

Use of CPs by various sectors of industry has resulted, naturally, in the generation of CP contaminated wastes. Information on their management has been reviewed and profiled.

Summaries of current (1979) and proposed (1981) use claims compiled and accepted by Agriculture Canada for products containing CPs have been included in this review.

Conclusions based on the evidence in this review are listed following the Table of Contents.

RÉSUMÉ

Étant une des substances de la Catégorie II de la liste des produits chimiques prioritaires d'Environnement Canada, les chlorophénols (CPs) ont été étudiés pour rassembler, de diverses sources, de l'information détaillée sur leur entrée, leur présence, leur transport, leur sort et leurs effets dans l'environnement Canadien. Cette étude technique a examiné non seulement les CPs mais aussi leur impuretés, principalement les dibenzo-p-dioxins polychlorés (PCDDs) et les dibenzofurans polychlorés (PCDFs).

Approximativement 3.4 million kg de CPs sont utilisés annuellement au Canada. Leur utilisation qui dépend de leur activité biologique, s'étend des préservatifs pour le bois et la peinture aux antimicrobes dans les systèmes de refroidissement industriels et la fabrication du papier. Au Canada, les CPs sont classés comme pesticides et comme tels, leurs utilisations industrielles et agricoles sont règlementées par Agriculture Canada sous la Loi sur les produits antiparasitaires.

Même s'il y a 8 CPs acceptés commercialement, seulement trois sont manufacturés au Canada, 2,4-dichlorophénol (2,4-DCP), 2,3,4,6-tétrachlorophénol (2,3,4,6-TTCP) et pentachlorophénol (PCP); les autres sont importés. Les sels de sodium du TTCP et du PCP ont été formulés au Canada.

L'entrée des CPs dans l'environnement s'est produite aux emplacements où les CPs ont été utilisés, comme les usines de traitement du bois et les usines de pulpe et papier. Les CPs peuvent être générés comme résultat de la chlorination aqueuse de composés organiques durant le traitement de l'eau potable et de l'eau résiduaire et peut aussi se produire comme métabolites des chlorobenzenes.

Les CPs se trouvent partout au Canada. Ils ont été identifiés dans des échantillons d'eau, de neige fondue, de sédiment, d'organismes aquatiques, de produits agricoles et dans l'homme.

La chimie des CPs, des PCDDs et des PCDFs a été étudiée ainsi que la méthodologie courante pour l'analyse de ces composés. L'information sur la toxicité aiguë et chronique et sur la toxicologie de ces composés dans les organismes terrestres et aquatiques a aussi été présentée. Même si tous les effets toxicologiques ne peuvent être directement attribués à un isomère de CP ou PCDD ou PCDF particulier, ils ont été identifiés dans cette étude comme tel quand et comme reportés dans la littérature.

Le sort des CPs, des PCDDs et des PCDFs dans l'environnement canadien est d'intérêt et a été étudié sous des entêtes telles que génération, dégradation, transport, bioconcentration et modelage.

L'utilisation des CPs par des secteurs variés de l'industrie conduit, naturellement, à la génération de rebuts contaminés de CPs. L'information sur leur gestion a été étudiée et profilée.

Des résumés des demandes d'utilisation courantes et proposées, compilées et acceptées par Agriculture Canada pour les produits contenant les CPs ont été inclus dans cette étude.

Les conclusions basées sur l'évidence dans cette étude sont dressées à la suite de la Table des matières.

Ottawa, Ontario
K1A 1C8

May 1, 1980

4402-78/C67

Dr. J.E. Brydon
Director
Contaminants Control Branch
Environmental Impact Control
Directorate
Environment Canada
14th Floor, Place Vincent Massey
Hull, Quebec

Dear Dr. Brydon:

Re: Letter of Transmittal of Technical Review Report on Chlorophenols

In November, 1977, I was assigned the task of reviewing the literature on the chlorophenols (CPs) and their impurities, particularly the chlorinated dibenzo-p-dioxins. CPs and their associated impurities were listed in Category III and are now listed in Category II of the List of Priority Chemicals. I am pleased now to convey to you with this letter of transmittal the final version of the EPS Technical Review Report "Chlorophenols and their Impurities in the Canadian Environment".

As evidenced by the information in this document, CPs are persistent in the environment, bioaccumulative, highly toxic to aquatic organisms and apparently ubiquitous in the Canadian environment. This Review, when coupled with the forthcoming companion document from Health and Welfare Canada, will serve as a basis for decisions on the CPs and their associated impurities by the DOE/NH&W Environmental Contaminants Committee.

Many persons have contributed to this Review by providing both information and expertise, but in particular I would like to acknowledge the generous assistance of Mr. Michael Gilbertson during the development of this Review.

Yours sincerely,

Dr. P.A. Jones
Assessment Coordination Division
Contaminants Control Branch
Environmental Impact Control
Directorate

A TECHNICAL REVIEW REPORT ON THE CHLOROPHENOLS AND THEIR IMPURITIES IN THE CANADIAN ENVIRONMENT

TERMS OF REFERENCE

The Contaminants Control Branch (CCB) of the Environmental Protection Service (EPS) identified the need for a Technical Review Report concerning chlorinated phenols in mid-1977. The report responds to the identification of chlorophenols as a class of chemicals that require priority assessment under the Environmental Contaminants Program. The following terms of reference were distributed to the Department of Fisheries and Environment/Department of National Health and Welfare Environmental Contaminants Committee:

1) To examine and evaluate information in the literature on the chlorophenols under the main subject headings of:
   a) toxicity of the compounds to organisms in the environment, and
   b) fate of the compounds following their release into the environment.
2) To obtain and review unpublished information on the chlorophenols, presently available within the Department of Fisheries and Environment.
3) To obtain information on current laboratory and field research programs, including international, national, and provincial, which will further add to the background knowledge on the chlorophenols and their impurities, in particular, the chlorodibenzo-p-dioxins and the chlorodibenzofurans.
4) To review the chlorophenols as to their Canadian production, importation, uses, and routes of entry into the environment.
5) To review information on formation of chlorophenols outside the normal routes of manufacture.
6) To examine information on the levels of the chlorophenols in the various ecosystems in Canada.
7) To examine the presently available information, both published and unpublished, on the presence of impurities, and their levels in the commercial chlorophenol products used in Canada.

8) To review information on the formation, bioaccumulation, and persistence, of biologically active transformation products as a result of degradation, or metabolism, of the chlorophenols in the environment.

9) To make recommendations:

   a) on whether further information should be generated, through research and field projects, by the government, and whether this research should be within the Department of Fisheries and the Environment or inter-departmental.

   b) on whether contaminants associated with the chlorophenols are at such a level in the environment that they are, or will present, an environmental hazard, and, if so, what controls should be instituted through existing authorities.

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ABREVIATIONS

Chlorophenols

m-chlorophenol (3-chlorophenol)	m-CP (3-CP)
o-chlorophenol (2-chlorophenol)	o-CP (2-CP)
p-chlorophenol (4-chlorophenol)	p-CP (4-CP)
dichlorophenol	DCP
trichlorophenol	TCP
tetrachlorophenol	TTCP
pentachlorophenol	PCP

Sodium salts of chlorophenols

sodium trichlorophenate	NaTCP
sodium tetrachlorophenate	NaTTCP
sodium pentachlorophenate	NaPCP
potassium pentachlorophenate	KPCP

Predioxins

4-chloro-2-(2,4-dichlorophenoxy) phenol	Cl_3-predioxin
4,5,6-trichloro-2-(2,4-dichlorophenoxy)phenol	Cl_5-predioxin

Chlorodibenzo-p-dioxins

dibenzo-p-dioxin	DD
chlorodibenzo-p-dioxin	CDD
dichlorodibenzo-p-dioxin	DCDD
trichlorodibenzo-p-dioxin	TriCDD
tetrachlorodibenzo-p-dioxin	TCDD
pentachlorodibenzo-p-dioxin	PnCDD
hexachlorodibenzo-p-dioxin	HCDD
heptachlorodibenzo-p-dioxin	HpCDD

octachlorodibenzo-p-dioxin	OCDD
polychlorodibenzo-p-dioxin	PCDD

Chlorodibenzofurans

chlorodibenzofuran	CDF
dichlorodibenzofuran	DCDF
trichlorodibenzofuran	TriCDF
tetrachlorodibenzofuran	TCDF
pentachlorodibenzofuran	PnCDF
hexachlorodibenzofuran	HCDF
heptachlorodibenzofuran	HpCDF
octachlorodibenzofuran	OCDF
polychlorodibenzofuran	PCDF

Miscellaneous - chemicals

hexachlorobenzene	HCB
tetrachlorodiphenyl ether	TCPE
2,4,5-trichlorophenoxyacetic acid	2,4,5-T
2,4,-dichlorophenoxyacetic acid	2,4-D
adenosinetriphosphatase	ATPase
deoxyribonucleic acid	DNA
polychlorinated biphenyls	PCBs
polybrominated biphenyls	PBBs
4,4'-dichlorodiphenylethylene	DDE

Miscellaneous - general

gas chromatograph	gc
gas-liquid chromatograph	glc
electron-capture	ec
mass spectrophotometer	ms
thin layer chromatography	tlc

parts per million (10^{-6}) (mg/kg, mg/L, or µg/g)	ppm
parts per billion (10^{-9}) (µg/kg or µg/L)	ppb
parts per trillion (10^{-12}) (ng/kg or ng/L)	ppt
micrograms	µg
nanogram	ng
picogram	pg
active ingredient	ai
volume per volume	V/V
per os (oral administration, by the mouth)	po
hour	h
week	wk
year	yr

CONCLUSIONS

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 activity 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 molecules (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 is 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. 4.2.1.1).

39) PCPs have been implicated in a few cases of industrial poisonings in Canada as the result of mishandling of these toxic materials (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 indicate 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 organims. 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. 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, ie, 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) PCDDs 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).

1 INTRODUCTION AND OVERVIEW

1.1 Background and Overview

The chlorophenols (CPs) had been listed by the Department of the Environment (DOE) - National Health and Welfare (NHW) Environmental Contaminants Committee in Category III of the List of Priority Chemicals (Canada Gazette, May 20, 1978. 3011 - 3015). Category III substances are those "which the government believes may pose a significant danger to the environment or human health, or about which further detailed information, including toxicology and amounts used is required." It was primarily for the latter reason that a technical review report was required. The CPs were reassigned to Category II in the List of Priority Chemicals - 1979 by the DOE/NHW Environmental Contaminants Committee (Canada Gazette, December 1, 1979. 7365-7370). Category II substances are "Those substances which are being investigated to determine the nature and extent of the danger to human health or the environment and the appropriate means to alleviate that danger."

The CPs included in this review were mono-, di-, tri-, tetra-, and pentachlorophenol. Of the 19 CP isomers available to industry, seven have commercial utility. They include: o-chlorophenol (2-CP); p-chlorophenol (4-CP); 2,4-dichlorophenol (2,4-DCP); 2,4,5-trichlorophenol (2,4,5-TCP); 2,4,6-trichlorophenol (2,4,6-TCP); 2,3,4,6-tetrachlorophenol (2,3,4,6-TTCP); and pentachlorophenol (PCP). All seven are marketed and used in Canada, although only three of the CPs are produced in Canada (Table 1). An additional CP, m-chlorophenol (3-CP), has limited commercial utility. Uniroyal Chemical Division of Uniroyal Limited produces 2,4-DCP, 2,3,4,6-TTCP, and PCP at Clover Bar, Alberta (Sect. 2.1.1). Production of these CPs at this facility is by catalytic chlorination of phenol; all other isomers used in Canada are imported.

An alternative method used in Europe for production of some CPs is by the alkaline hydrolysis of chlorobenzenes, including production of PCP from hexachlorobenzene (HCB). This production process leads to the presence of chlorobenzenes as impurities in CPs (Sect. 2.1). Canadian requirements for CPs, approximately 3.4 million kg, necessitates the importation of approximately 2.1 million kg of CPs per year (Sect. 2.1.3).

The CPs can be classed as biocides or broad spectrum pesticides. They have biological activity as bactericides, slimicides, fungicides, herbicides, and insecticides.

TABLE 1 THE COMMERCIAL CHLOROPHENOLS USED OR MARKETED IN CANADA

Chemical	Suppliers/Principals[1]
m-chlorophenol (3-CP)	Bayer (Canada) Ltd. 7600 Trans-Canada Highway Pointe Claire, Que. H9R 1C8
o-chlorophenol (2-CP)	Bayer (Canada) Ltd.
p-chlorophenol (4-CP)	Bayer (Canada) Ltd. Japan Chemicals Ltd. 940 Alness St. Unit 10 Downsview, Ont. M3J 2R9
2,4-dichlorophenol (2,4-DCP)	Bayer (Canada) Ltd. Dow Chemical of Canada Ltd. P.O. Box 1012, Hgwy. 40 Sarnia, Ont. N7T 7K1
	* Uniroyal Chemical Division of Uniroyal Ltd. Erb St. Elmira, Ont. N3B 3A3 Clover Bar, Alta.
2,4,5-trichlorophenol (2,4,5-TCP)	Atlantic Trading Co. 3335 Yonge St., Suite 404 Toronto, Ont. M4N 2M2
	Bayer (Canada) Ltd. Dow Chemical of Canada Ltd.
	Record Chemical Co., Inc. 840 Montee De Liesse Montreal, Que. H4T 1N8
2,4,6-trichlorophenol (2,4,6-TCP)	Bayer (Canada) Ltd.
	Dow Chemical of Canada Ltd.
	Tennant Charles & Co. (Canada) Ltd. 34 Clayson Rd. Weston, Ont. M9M 2G8
2,3,4,6-tetrachlorophenol (2,3,4,6-TTCP)	Dow Chemical of Canada Ltd.
	* Uniroyal Chemical Division of Uniroyal Ltd. Clover Bar, Alta.

TABLE 1 (Cont'd) THE COMMERCIAL CHLOROPHENOLS USED OR MARKETED IN CANADA

Chemical	Suppliers/Principals[1]
pentachlorophenol (PCP)	Canada Colors & Chemicals Ltd. 160 Bloor St. E. Toronto, Ont. M4W 1C6
	Domtar Chemicals Ltd. 395 Maisonneuve Blvd. W. Montreal, Que. H3A 1L6
	Dow Chemical of Canada Ltd.
	Lawrason S. F. & Co. Ltd. 180 Adelaide St. S. Box 2425 London, Ont. N6A 4G3
	May and Baker (Canada) Ltd. 3300 Côte Vertu, Suite 202 St. Laurent, Montreal H4R 2B7
	Stanchem Div. PPG Ind. Canada Ltd. 5029 Ambroise St. Montreal, Que. H4C 2E9
	* Uniroyal Chemical Division of Uniroyal Ltd. Clover Bar, Alta.
	Van Waters and Rogers Ltd. 980 Van Horne Way Richmond, B.C. V6X 1W5
Sodium pentachlorophenate (NaPCP)	Canada Colors & Chemicals Ltd.
	Dow Chemical of Canada Ltd.
	Harrisons & Crosfield (Canada) Ltd. 4 Banigan Dr. Toronto, Ont. M4H 1G1
	Kingsley & Keith (Canada) Ltd. 310 Victoria Ave. Montreal, P.Q. H3Z 2M9

TABLE 1 (Cont'd) THE COMMERCIAL CHLOROPHENOLS USED OR MARKETED IN CANADA

Chemical	Suppliers/Principals[1]
	May and Baker (Canada) Ltd. 3300 Côte Vertu, Suite 202 St. Laurent, Montreal H4R 2B7
	Reichhold Chemicals Limited P.O. Box 130 Port Moody, B.C. V3H 3E1
	Van Waters and Rogers Ltd.

* = manufacturer locality, plant location is underlined
From: In part, Canadian Chemical Processing, Chemical Buyers Guide / 1979
[1] Formulators and all importers are not included

They are used as preservatives in woods, paints, drilling muds, photographic solutions, hides and leathers, and textiles; and as antimicrobials in industrial cooling systems, and pulp and paper mill systems. They are also used in the agricultural sector as herbicides and insecticides. Their greatest utility is for wood preservation. An estimated 1.29 million kg or 95% of the 1976 Canadian production of PCP was for wood preservation (Sect. 2.2.1). In addition to the agricultural and industrial uses of CPs, which are regulated by Agriculture Canada under the Pest Control Products Act, CPs also find use in health care and veterinary products and in disinfectants. The use of CPs in these classes of products is regulated by Health and Welfare Canada under the Food and Drug Act (Sect. 2.2.3).

As a result of process chemistry, all higher CPs, including TCP, TTCP, and PCP, contain the biologically active impurities polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) (Sect. 3). If CPs enter the environment, therefore, their environmental impact must be weighted to a lesser or greater degree by the presence and levels of the impurities, such as the hexa-, hepta-, and octa- CDDs, which are present in TTCP and PCP. The most toxic dioxin, the 2,3,7,8- isomer of TTCD does not occur in the most widely used CPs, TTCP or PCP, but may be present in TCP.

While CPs are only mildly toxic to terrestrial organisms, they are highly toxic to aquatic organisms with toxicity increasing as the number of chlorine atoms on the phenol ring increases (App. 3, Sect. 3.1.1.1 and App. 4, Sect. 4.1.1). This general

statement requires qualification. For example, in the case of mammalian toxicity from the mono-CPs, the toxicity may vary in accordance with the ring position of the chlorine atom: 2-CP and 3-CP are more toxic than 4-CP to rats, and toxicity may also vary with the route of administration of the CPs to the test animal. LC_{50} values derived from oral administration of the mono-CPs to rats are much lower than those resulting from interperitoneal or subcutaneous administration (Table A3-3).

Published acute toxicity data for the PCDDs in terrestrial animals are limited, except for the highly toxic TCDD. HCDD and OCDD are relatively non-toxic to rats and mice when administered at rates up to 1 g/kg. Research has shown that the toxicity of the PCDDs to mice and guinea pigs can be correlated with the degree of chlorination at the 2,3,7, or 8 position (App. 3, Sect. 3.1.2.1). Determination of acute toxicity levels of PCDD in fish is difficult because of an apparent delayed toxic response (App. 4, Sect. 4.1.4).

Although the mode of action of CPs is not fully understood, PCP is known as a potent uncoupler of oxidative phosphorylation when present in low concentrations and inhibits the same enzyme systems at high concentrations (App. 5, Sect. 5.1). It has also been postulated that part of the mammalian toxicity of CPs is due to membrane perturbation. The toxic action of the impurities in PCP - PCDDs and PCDFs - is still unknown (App. 5, Sect. 5.1).

Teratogenicity studies, with commercial grade and purified TTCP and PCP administered at a maximum tolerated dose of 30 mg/kg of TTCP and 50 mg/kg of PCP/day to pregnant Sprague-Dawley rats, have shown that neither compound was tera togenic, although PCP was highly embryolethal and embryotoxic. TTCP was not embryolethal and caused only a minimal degree of fetotoxicity (App. 3, Sect. 3.1.1.4).

Tumorigenic studies with the CPs have indicated a direct relationship between the presence of tumorigenic lesions and the isomeric structure of the chlorophenols. Treatment of mice with 2,4,5-TCP induced large numbers of papillomas, whereas treatment with 2,4,6-TCP or PCP did not result in abnormalities. Whether the induction of papillomas was due to the particular CP isomers or to the dioxin impurities present in the CPs has not been satisfactorily delineated (App. 3, Sect. 3.1.1.4).

A recent two year study on PCP in rats found that PCP was not carcinogenic when administered to rats in their diet on a chronic basis at dose levels sufficiently high to cause mild signs of toxicity (1, 3, 10, and 30 mg/kg/day).

When data from the 2-yr rat feeding study were evaluated for PCP effect on rat reproduction, it was noted that, except for a significant decrease in neonatal survival and growth among litters of females ingesting 30 mg PCP/kg/day, measures of reproduction capacity were unaffected by either the 10 mg or 30 mg/day dosage of PCP (App. 3, Sect. 3.1.1.4).

Various authors have pointed out that CDDs are not all alike in their toxicological properties. Although the symmetrical 2,3,7,8-TCDD is a known teratogen in mice and rats, the 1,2,3,4-TCDD is not embryotoxic at doses as high as 800 µg/kg/day. A recently completed 2-yr feeding study with Sprague-Dawley rats demonstrated a statistically significant increase ($p=0.05$) in tumors in rats fed as little as 5 ppt of 2,3,7,8-TCDD/gm in the diet compared to the control rats. HCDD and OCDD were identified as being non-teratogenic in the rat at 1.0 and 500 mg/kg/day, respectively; however, OCDD at the 500 mg/kg/day level did cause embryotoxicity in rats (App. 3, Sect. 3.1.2.6). The no-effect dose level for 2,3,7,8-TCDD ingested by rats for a lifetime is between 0.001 µg to 0.01 µg TCDD/kg/day (App. 3, Sect. 3.1.2.2).

The biochemical effects of only a few CDDs and CDFs have been reported. The 1,2,3,7,8,9- isomer of HCDD was identified as a factor in chick edema disease. Its lethality is low, however, compared to the tri-, and tetra- CDDs, which also cause chick edema disease. The 2,3,7,8-TCDD is a potent porphyrogen in male mice. Other documented effects are the liver changes which have been noted in female rats as a result of inclusion in their diet of CDDs and CDFs normally present in some PCP preparations (App. 3, Sect. 3.1.2.4).

CP use patterns and the disposal of wastes containing CPs are directly associated with the volume of CPs entering the environment (Sect. 4.0). As a result of this ingress, CPs and their impurities are probably ubiquitous in the Canadian environment, although few general surveys to identify their presence and levels have been carried out. One of the exceptions was a survey for CPs in the Great Lakes basin in 1977 by personnel from the Canada Center for Inland Waters. CP residues were detected in water and sediment samples from the Great Lakes and in effluents from municipal sewage treatment plants in Ontario (Sect. 5.1.1 and 5.1.2). Similar surveys have been undertaken jointly by Environment Canada and the Province of British Columbia to identify organic contaminants, including CPs, in the lower Fraser River and the Fraser Estuary with particular reference to industrial and municipal sources (Sect. 5.1.1 and 5.1.2). In the Maritime Provinces and in British Columbia CP residues have been detected in both

freshwater and marine fauna (Sect. 5.1.4.2). In this regard, research has shown that PCP can be quickly absorbed by fish and will accumulate in various organs, particularly the gall bladder (App. 5, Sect. 5.2). Although PCP can be rapidly excreted by fish, low residual levels of PCP may remain. Residues of CPs have also been detected in food and livestock feed that have been directly exposed to CPs (Sect. 5.2.6, and 5.2.7).

Both laboratory and field studies have shown that CPs are bioaccumulative in aquatic fauna (App. 8, Sect. 8.1).

Although CPs are bioaccumulative and persistent in the environment, degradation does occur through chemical, photochemical, and microbiological processes (App. 6, Sect. 6.1.1, 6.1.2, and 6.1.3, respectively). CP impurities in environmental media can be degraded through photolytic, thermal, and microbiological action (App. 7, Sect. 7.2.1, 7.2.2, and 7.2.3, respectively).

The economic benefits, associated with use of CPs as wood preservatives, have been documented (Sect. 2.2.1). In contrast, CPs have been implicated in incidents of food and feed contamination. Two examples of the latter, found in this report, are summarized briefly below.

In the winter of 1977 a shipment of western Canadian feed grains for consumption by livestock in eastern Canada became contaminated with PCP as a result of inadequate or improper cleaning of a railway box-car previously used for shipping PCP. Unwitting use of the contaminated grain by stock growers led to partial feed refusal by livestock and withholding of livestock and milk from market (Sect. 5.2.7).

The second example, which illustrates a continuing problem, both in Canada and in other countries, involves the use of CP contaminated wood shavings as livestock litter, particularly in poultry houses. Microorganisms, which use poultry droppings in the litter as substrate convert the CPs to chloroanisoles. The chloroanisoles, taken up by the poultry, probably through inhalation or dermal contact, have been identified as the compounds responsible for the tainting of poultry meat and eggs. If chloroanisole residues are present, even at very low levels, the tainted food is unacceptable for human consumption. This results in large financial losses in the poultry broiler industry, when it becomes necessary to destroy large numbers of birds.

1.2 Literature Cited and Referenced

The extensive amount of literature published on CPs and their impurities required that a screening system be employed. The modus operandi for including, or excluding, particular literature citations in this review included first, an examination of

information in recent review papers, supplemented with new material in references published, or unpublished, since the review articles. Secondly, information from older references has been included to supply the background material which is necessary to understand the current literature, or to supply basic information for those subjects on which no recent review papers were available. It was apparent that most of the research on CPs and their impurities had been conducted outside Canada and that there would be little Canadian content; however, if published Canadian research papers were available, they have been included as reference material.

The review information on toxicology, mode of action, and metabolism was selected to serve as background for an understanding of the impact of CPs and their impurities on fauna in the environment, rather than on human health. The information related to human health was selected as a sample of published literature, to indicate that CPs may affect human health.

Health and Welfare Canada is currently reviewing CPs from a human health standpoint.

1.3 Other Reviews and Publications

In the United States, the Environmental Protection Agency instituted a Rebuttable Presumption Against Registration and Continued Registration (RPAR) of pesticide products containing 2,4,5-trichlorophenol and its salts (F.R. 43(149):34026 - 34054, August 2, 1978. Part II). This RPAR was based on unfavorable risk criteria relative to oncogenicity and other chronic or delayed toxic effects, including fetotoxicity.

An RPAR was also set out against Wood Preservation Pesticides including those pesticide products containing pentachlorophenol (F.R. 43(202):48443 - 48478, October 18, 1978. Part II (cont.)). This RPAR was based on chronic or delayed toxic effects relating to teratogenic and/or fetotoxic effects in mammalian test species. The criteria documents upon which the RPARs were based have provided information not readily available elsewhere on 2,4,5-TCP and PCP and their impurities, particularly the dioxins.

Two other recent publications of merit were 1) "An Analysis of the Existing Wood Preserving Techniques and Possible Alternatives" authored by Fuller et al (1977) for the Mitre Corporation; and 2) a compilation of papers, which had been individually presented at a symposium on PCP, and were published in 1978 by Plenum Press under the title of "Pentachlorophenol: chemistry, pharmacology, and environmental toxicology", edited by K.R. Rao.

One other important review document was received from the Science Advisory Board of the U.S. E.P.A. They had compiled considerable information on the impurities, particularly the PCDDs, identified in PCP. The report, entitled "Report of the Ad Hoc Study Group on Pentachlorophenol Contaminants", was received in May 1979, with a publication date of December 29, 1978.

2 PRODUCTION AND USE OF CHLOROPHENOLS

As noted in Sect. 1.1, this technical review presents information on the chlorinated phenols and their impurities. The relationship between the chlorophenols and other phenolic compounds, as regards their structures, sources, and uses, is given in Table 2.

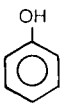
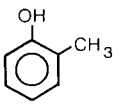
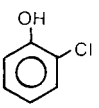
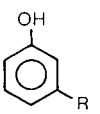
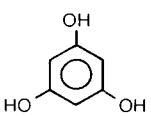
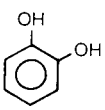
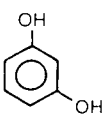
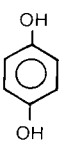
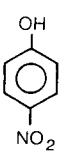
This chapter includes a brief summary of the processes for commercial production of CPs (Sect. 2.1), with emphasis on those processes currently used in North America. The bulk of the CPs used in Canada, primarily the di-, tetra-, and penta-CPs are produced in two Canadian and several American plants. The ratio between imported and Canadian produced CPs has been estimated at 30:70 (Sect. 2.1.3), with more than two-thirds of the Canadian imports coming from the United States (Table 7). In 1976 Canada used an estimated 3.4 million kg of CPs for various purposes, as outlined in this chapter.

2.1 Commercial Production of Chlorophenols

Von Rumker et al (1974) have adequately summarized the process for production of PCP by chlorination of phenol. Their description of the process, the reaction chemistry (Fig. 1) and the production and waste schematic (Fig. 2), as abstracted from Stoesser (1938), Shelton et al (1960), and Sittig (1967), are given below:

> "The chlorination is performed at substantially atmospheric pressure. The temperature of the phenol in the primary reactor at the start is in the range of 65-130°C (preferably 105°C) and is held in this range until the melting point of the product reaches 95°C. About three to four atoms of chlorine are combined at this point, the temperature is progressively increased to maintain a temperature of about 10°C over the product melting point, until the reaction is completed in 5-15 hr. The mixture is a liquid, and a solvent is not required, but the catalyst concentration is critical; about 0.0075 mol of anhydrous aluminum chloride is usually used per mol of phenol.
>
> "The off-gas from the chlorination reactor (largely HCl during the initial reaction and chlorine near the conclusion) is sent to a scrubber-reactor system (secondary reactor) containing excess phenol. It is held at a temperature such that the chlorine is almost completely reacted to give the lower chlorinated phenols, which may be either separated, purified and sold, or returned to be

TABLE 2 USE PATTERNS OF VARIOUS PHENOLIC COMPOUNDS (from Buikema et al, 1979) (from various sections of Kirk-Othmer Encyclopedia of Chemical Technology, 2nd Ed., John Wiley and Sons, New York).

Class of compound	Structure	Source/synthesis	Uses
Phenol		Cumene Benzene	53% phenolic 8% bisphenol A 7% alkylphenols 7% caprolactam 25% other
Cresols[1]		Petroleum or coal tar	28% phenolic resins 25% tricresylphosphate 10.7% disinfectants 8.9% antioxidants 8.4% engine and metal cleaners 7.1% ore-flotation 6.2% wire-enamel solvent 4% miscellaneous
Chlorophenols[1]		Phenol Chlorobenzene Nitrobenzene	Biocides and intermediates for biocides, wood preservation
Alkylphenols		Phenol	Antioxidants (BHT) Gasoline, oil Greases Plastics
Polyhydroxybenzenes (e.g., Pyrogallol Gallic acid Phloroglucinol)		Trinitrobenzoic acid	Pigments Medicines Photographic chemicals Dyes
Pyrocatechol		Natural resins Lignins	Antioxidants Dyes
Resorcinol		Halophenols Benzenedisulphonic acid Phenosulphonic acid Benzene	Antioxidants Eosin dyes Antiseptic Medicinals Explosives
Hydroquinone		Aniline	Photographic chemicals Antioxidants Medicinals
Nitrophenols[1]		Phenol Nitrochlorobenzene Benzene	Dyestuff Intermediates Explosives

[1] Various isomers are used; only one of the structural formulae is shown.

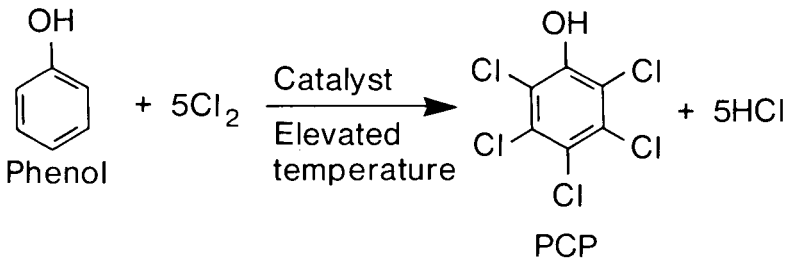


FIGURE 1 REACTION CHEMISTRY FOR PRODUCTION OF PENTACHLOROPHENOL BY CHLORINATION OF PHENOL

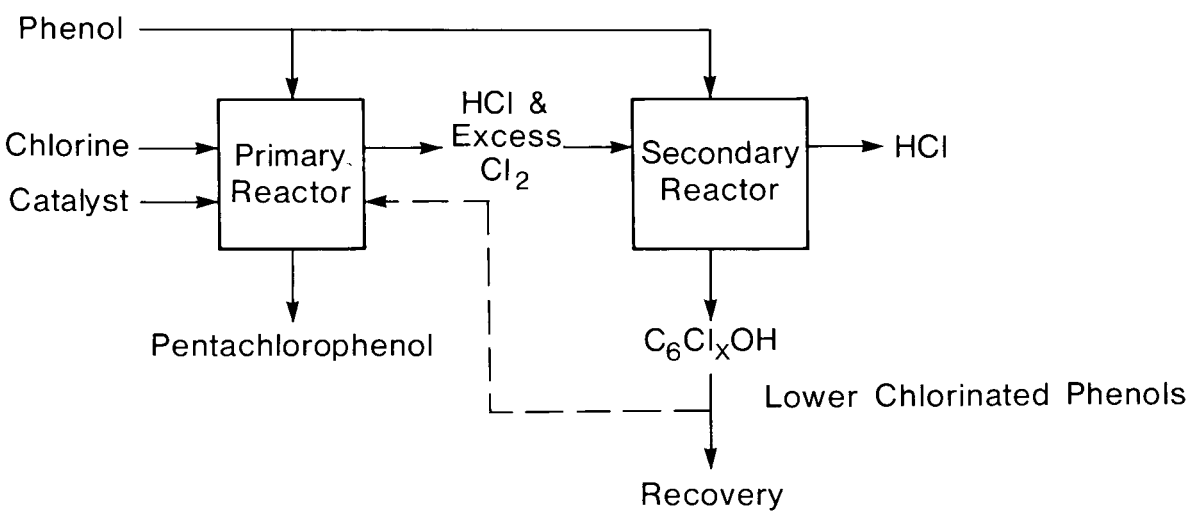


FIGURE 2 PRODUCTION AND WASTE SCHEMATIC FOR THE CHLOROPHENOLS (Adapted from von Rumker et al, 1974)

used as the primary pentachlorophenol reactor. The residual gas is substantially pure HCl."

In the residual gas, which can be used for production of industrial grade hydrochloric acid (HCl), otherwise known as muriatic acid, there may be small amounts of the lower chlorinated phenols. These can be removed by filtering the HCl through activated charcoal.

Since the chlorination of phenols, from mono-, di-, and tri-CPs through TTCPs to PCP is not a quantitative process, TTCPs are carried over into the PCPs. The TTCPs, of which specific isomers are usually not distinguished in formulations, are not considered as impurities but are listed as active ingredients (ai). Commercial grade PCP usually contains from 4 - 12% TTCP.

In Howard and Durkin (1973) a process flow chart indicates that production of PCP can be accomplished by hydrolysis of hexachlorobenzene (HCB). Von Rumker et al (1974), however, have indicated, after consultation with industry, that PCP has never been produced in the U.S. by that method, although patents covering the process have been assigned to Dow and Diamond Alkali (Sittig 1967).

Some of the PCP manufactured in Europe, which is imported into Canada, is derived from the alkaline hydrolysis of HCB at elevated temperatures and pressures (Rappe and Nilsson, 1972; Jensen and Renberg, 1972). This method can contribute to the presence of HCB in the PCP. Dutch regulations state that PCP should contain less than 5,000 ppm of HCB. The quantity of European PCP, which enters Canada and has been produced by alkaline hydrolysis of HCB, is unknown.

The lower chlorinated 2,4,5-TCP is produced by the alkaline hydrolysis of 1,2,4,5-tetrachlorobenzene (Rappe et al, 1978). Other lower chlorinated commercial CPs used as wood preservatives, including 2,4,6-TCP, potassium 2,3,4,6-TTCP, and 50% aqueous solution of potassium 2,4,6-TCP, are all manufactured by catalytic chlorination of phenol (Anderson et al, 1973; Nilsson and Renberg, 1974).

2.1.1 Production of Chlorophenols in Canada. Uniroyal Chemical Division of Uniroyal currently is the only producer of CPs in Canada. The plant which is located at Clover Bar, near Edmonton, Alberta, produces 2,4-DCP, 2,3,4,6-TTCP and PCP. The name-plate capacities for these CPs are given in Table 3. The Dow Chemical of Canada Limited CP plant at Fort Saskatchewan, also near Edmonton, Alberta, which had been limited to 2,4-DCP production, was dismantled in 1980.

TABLE 3 CANADIAN CHLOROPHENOL PRODUCERS, PLANT LOCATIONS AND CAPACITIES - 1980

Producer and location	Products Plant Capacity (kg) x 10^3		
	2,4-DCP	2,3,4,6-TTCP	PCP
Dow Chemical of Canada Ltd. Fort Saskatchewan, Alta.	*	NP	NP
Uniroyal Chemical Div. of Uniroyal Ltd. Clover Bar, Alta.	**	450	1,800

2,4-DCP = 2,4-dichlorophenol
2,3,4,6-TTCP = 2,3,4,6-tetrachlorophenol
PCP = pentachlorophenol
*Plant dismantled in late 1980
**Plant capacity not available
NP Not produced in Canada

Actual production, of course, fluctuates from year to year, and may be much below plant capacity.

During the production of DCP, usually about 93% is evolved as 2,4-DCP and the remaining 6 - 7% comes off as 2,6-DCP.

2.1.2 Production of Chlorophenols in the United States. The United States CP producers in 1978 are shown in Table 4 along with the plant locations. Production facilities for the lower CPs have undergone some changes. For example, in late 1978, a multimillion dollar modernization project was undertaken at the Monsanto CP plant at Sauget, Illinois, where CP production facilities are dedicated to o-CP, p-CP, and 2,4-DCP (Chemical Marketing Reporter, Oct. 23, 1978). Plant capacity data were not available for the lower CPs, although data were available for PCP (Table 5).

By 1978 PCP plant capacity figures for the United States were changed markedly from previous years because the Monsanto capacity to produce about 11.8 million kg of PCP per year (about 1/3 of the total U.S. capacity) was phased out. Plant capacity at each of the three remaining producers - Dow, Reichhold, and Vulcan - had steadily increased since 1974. The Dow capacity had increased from 8.2 to 13.5 million kg per year while Reichhold and Vulcan went from an estimated 5.4 million kg per year each to 8.1 million kg and 9.0 million kg, respectively (Chemical Marketing

TABLE 4 UNITED STATES CHLOROPHENOL PRODUCERS, PLANT LOCATIONS, AND PRODUCTS[1]

Producer	Plant Location	Products[2]
Aldrich Chemical Co., Inc.	Milwaukee, WI	m-chlorophenol
		2,6-dichlorophenol
		3,4-dichlorophenol
		3,5-dichlorophenol
Dow Chemical, USA	Midland, MI	o-chlorophenol (2-chlorophenol)
		p-chlorophenol (4-chlorophenol)
		2,4-dichlorophenol
		2,4,5-trichlorophenol
		2,4,6-trichlorophenol
		pentachlorophenol
Eastman Kodak Co.	Rochester, NY	m-chlorophenol
Monsanto Co.		
Monsanto Chemical Intermediates Co.	Sauget, IL	o-chlorophenol (2-chlorophenol)
		p-chlorophenol (4-chlorophenol)
		2,4-dichlorophenol
Northwest Industries Inc. Velsicol Chemical Corp.	Beaumont, TX	2,5-dichlorophenol
Reichhold Chemicals, Inc.	Tacoma, WA	pentachlorophenol
R.S.A. Corporation	Ardsley, NY	m-chlorophenol
Specialty Organics, Inc.	Irwindale, CA	m-chlorophenol
		2,3-dichlorophenol
		2,6-dichlorophenol
Vertac, Inc. Transvaal, Inc.	Jacksonville, AR	2,4-dichlorophenol
		2,4,5-trichlorophenol
Vulcan Materials Co. Chemical Div.	Wichita, KS	pentachlorophenol

[1] 1977 Directory of Chemical Producers, and 1978 Supplement

[2] Chemicals listed may include those for captive use.

TABLE 5 PENTACHLOROPHENOL PRODUCERS, PLANT LOCATIONS AND CAPACITIES IN THE U.S. IN 1980[1]

Producer	Location	Capacity (kg) x 10^3
Dow Chemical U.S.	Midland, MI	13,500
Reichhold Chemicals, Inc.	Tacoma, WA	8,100
Vulcan Material Co. Chemical Div.	Wichita, KS	9,000

[1]Chemical Marketing Reporter, 218(16), October 20, 1980

Reporter 218(16), October 20, 1980. The Chemical profile in Chemical Marketing Reporter 218(16) indicated that Reichhold intended to expand to 13.5 million kg per year at Tacoma in 1981.

Current production and sales figures for the U.S. are incomplete; however, U.S. sales volumes, including both domestic and export, from 1972 - 1975 show a

TABLE 6 PENTACHLOROPHENOL PRODUCTION AND SALES IN THE U.S. 1972 - 1974

Year	Production (kg) x 10^3	Sales (kg) x 10^3	Reference
1972	22,545	21,933	U.S. Tariff Commission, Synthetic Organic Chemicals. U.S. Production and Sales 1972. T.C. Publ. 681
1973	21,140	22,197	U.S. International Trade Comm. Synthetic Organic Chemicals. U.S. Prod. and Sales 1973 U.S.I.T.C. Publ. 728
1974	23,756	24,435	U.S. International Trade Comm. Synthetic Organic Chemicals U.S. Prod. and Sales 1974 U.S.I.T.C. Publ. 776
1974		19,728	Mitre Corp., Fuller et al, 1977
1975		16,093	Mitre Corp., Fuller et al, 1977
1976	19,899		Chem. Mkt. Rpt. 215(15) April 9, 1979
1977	20,349		Chem. Mkt. Rpt. 215(15) April 9, 1979

fluctuation in annual sales from 16.1 million kg to 24.4 million kg (Table 6). Export sales were approximately 1% of domestic sales.

The latest marketing profile for PCP in the U.S. published by Chemical Marketing Reporter 218 (16) Oct. 20, 1980, indicated a growth of 1.1% per year during the past ten years. Consumption was expected to expand slowly and steadily, barring any government controls.

2.1.3 Importation of Chlorophenols into Canada. Imports of CPs from 1971 to 1979 (Table 7), fluctuated between a high of 3,469,400 kg in 1971 and a low of 926,000 kg in 1979.

Pentachlorophenol imports in 1979 of 625,600 kg (Table 8) were approximately 38% of the total CPs imported. Similar statistics prior to 1976 were not available. The majority of the imports have consistently come from the United States.

2.2 Use of Chlorophenols in Canada

2.2.1 Commercial. The CPs manufactured, imported, and marketed in Canada are used either directly as pesticides or biocides, or are used as intermediates in the manufacture of pesticides. For example, all of the 2,4-DCP production in Canada is used as an intermediate in the manufacture of 2,4-D phenoxy herbicides. Although it is not manufactured in Canada, 2,4,5-TCP is used in the United States in the manufacture of the insecticide ronnel (O,O-dimethyl O-(2,4,5-trichlorophenyl) phosphorothioate) which is used extensively in Canada on livestock. The 2,3,4,6- isomer of TTCP, which is produced in Canada also enters the pesticide market. It also usually occurs along with PCP as an active ingredient in wood preservatives. The quantities of di-, tri-, and tetra-CPs used in Canada have not been computed.

Although PCP and its sodium salt have a variety of applications as fungicides or biocides, which range from the preservation of wood to the treatment of water in cooling systems, the major use is as a wood preservative. This accounts for approximately 95% of the volume of PCP utilized in Canada. By way of comparison, the latest Chemical Marketing Reporter profile (Vol. 218 (16) Oct. 20, 1980 for PCP shows 79% of PCP production in the U.S. is used for wood preservation for poles, crossarms, and piles; 12% for sodium pentachlorophenate (NaPCP) production, and 19% for water treatment and other uses.

TABLE 7 IMPORTS OF CHLOROPHENOLS INTO CANADA, 1971 - 1979[1]

Country	1971[2] Quantity (kg) x 10^3	1971[2] Value ($) x 10^3	1972[2] Quantity (kg) x 10^3	1972[2] Value ($) x 10^3	1973[3] Quantity (kg) x 10^3	1973[3] Value ($) x 10^3
United States	3,422.8	1,598	2,085.1	941	2,164.6	1,206
France	24.9	12	50.1	33	19.1	7
Japan	-	-	11.6	4	-	-
Netherlands	-	-	0.3	1	0.3	1
Switzerland	0.6	2	-	-	-	-
United Kingdom	16.4	25	88.7	93	1.8	3
W. Germany	4.7	7	45.2	60	8.5	17
Total	3,469.4	1,644	2,281.0	1,132	2,194.3	1,234

Country	1974[3] Quantity (kg) x 10^3	1974[3] Value ($) x 10^3	1975[3] Quantity (kg) x 10^3	1975[3] Value ($) x 10^3	1976[3] Quantity (kg) x 10^3	1976[3] Value ($) x 10^3
United States	1,805.7	1,186	1,668.2	1,818	1,209.8	1,347
France	142.2	136	250.9	234	120.8	70
Japan	-	-	-	-	-	-
Netherlands	-	-	-	-	-	-
Switzerland	-	-	-	-	-	-
United Kingdom	670.7	346	26.1	132	206.2	552
W. Germany	9.1	16	14.8	47	107.1	142
Total	2,627.2	1,684	1,960.0	2,231	1,643.9	2,111

Country	1977[2] Quantity (kg) x 10^3	1977[2] Value ($) x 10^3	1978[3] Quantity (kg) x 10^3	1978[3] Value ($) x 10^3	1979[3] Quantity (kg) x 10^3	1979[3] Value ($) x 10^3
United States	1,081.7	1,347	-	-	-	-
France	23.4	48	-	-	-	-
Japan	-	-	-	-	-	-
Netherlands	-	-	-	-	-	-
Switzerland	-	-	-	-	-	-
United Kingdom	42.2	109	-	-	-	-
W. Germany	90.2	83	-	-	-	-
Total	1,237.5	1,587	1,573.0	1,894	926.0	1,385

1 Statistics Canada, Imports by Commodities and Countries
2 Through November
3 Through December

TABLE 8 IMPORTS OF PENTACHLOROPHENOL INTO CANADA, 1976 - 1979[1]

Country	1976		1977		1978		1979	
	Quantity (kg) x 10^3	Value ($) x 10^3	Quantity (kg) x10^3	Value ($) x 10^3	Quantity (kg) x 10^3	Value ($) x 10^3	Quantity (kg) x 10^3	Value ($) x 10^3
Unidentified	188	186	-	-	77	89	77	78
West Germany	-	-	66	70	-	-	-	-
United States	370	315	310	305	468	498	548	685
Total	558	501	377	375	545	587	625	763

[1]Statistics Canada, Imports by Commodities and Countries

The quantity of PCP used in Canada in 1976 has been estimated at 1,746,300 kg plus an additional 182,000 kg of NaPCP (Table 9), for a total of 1,928,300 kg. Of that amount, approximately 624,600 kg were imported, primarily from the United States (Table 8).

Further estimates indicate that the Prairies and Ontario have each used one-third of the total PCP marketed each year in Canada, with lesser amounts used in B.C., Quebec, the Maritimes, and the Territories, in that order.

Market survey data for NaPCP, the water soluble salt of PCP, were not available; however, approximations were made of the market share for each major sector (Table 9).

TABLE 9 AN ESTIMATION OF ANNUAL QUANTITY OF SODIUM PENTACHLOROPHENATE UTILIZED BY SECTORS OF INDUSTRY AND AGRICULTURE IN CANADA - 1976

Sector	% of Quantity	Quantity (kg)
Fungicides (e.g. mushroom house fungicide)	5	9 100
Water treatment	30	54 600
Wood treatment (sap stain inhibitor)	45	81 900
Leather and tanning industry	10	18 200
Miscellaneous	10	18 200
Total		182 000

The anti-microbials used as sap stain inhibitors and as water treatments usually contain NaPCP or NaTTCP as the major active ingredient. NaPCP may include 3% - 18% of NaTTCP and/or NaTCP. The sap stain inhibitors used in British Columbia are usually formulated from NaTTCP as the main active ingredient up to a level of 24.2% (Table A10-2). It should be noted that when sap stain inhibitors are discussed in this review distinction has been made as to whether the main active ingredient was NaPCP or NaTTCP.

Wood preservation is a major component of the Canadian forest products industry, which ranks first in export dollars earned for Canada. In fact, in 1976, the value of forest products exported totalled 6.5 billion dollars (Hansard, 1979). By 1979, the net contribution of forest products exports to Canada's balance of payments was $10.6 billion,

an increase of $2 billion over 1978 (Environment Canada, 1980a, 1980b). Therefore, it is of interest to present some background information on wood preservation treatments, such as types of materials and number of treatment plants, to have a better grasp of the financial impact the use of CPs has on the wood industry.

The necessity for treatment has been documented by Smith (1978) who has briefly summarized both the role played by the various fungi in the biodeterioration of wood and also the treatment systems available to combat the decay organisms. Treatments for long-term protection of wood are generally referred to as wood preservation techniques. Treatments for short-term protection are known as wood protection techniques. More details on the systems are provided by Arsenault (1978), Shields and Stranks (1978), and Smith (1978). Comparative costs, in 1974 dollars, for the various systems used in western Canada were given by Cooper (1974).

Cserjesi and Roff (1975) have pointed out that unseasoned wood with a moisture content of more than 20% (therefore susceptible to fungus attack) is shipped overseas in large volumes from the Pacific Northwest. Total lumber exports from B.C. in 1979 were approximately 9.95 billion bd.ft. The water soluble sodium salts of CPs are used exclusively to protect this wood from sap stain and mould fungi.

Based on data from various sources, including Statistics Canada and the B.C. Council of Forest Industries, it is estimated that during 1977 in British Columbia approximately 784 tonnes of NaTTCP:NaPCP/2.125:1 were used to treat 6.62 billion bd. ft. of lumber for short term protection against sap stain fungi. In 1977 approximately 55% of the 12.038 billion bd. ft. of lumber produced in B.C. was treated for sap stain control. It is also estimated that the use of sap stain control materials was at the same level or slightly higher in 1979.

The amount of CPs used for treatment of export lumber may decrease, since commencing in 1978 a higher percentage of the lumber exported to the United States will probably be kiln dried. The reasons for this are two-fold: a) U.S. Federal regulations governing wood house construction and the requirement for kiln dried lumber, and b) the need for Canadian lumber exporters to maintain a competitive position in the U.S. market. Because of the savings in freight costs lumber products destined for shipment by rail are usually dried to a moisture content of less than 20%, which is not conducive to the growth of sap stain and moulds.

Benefits derived from wood preservation far outweigh the initial costs incurred. Benefits occur in the area of conservation of resources including the energy

used in wood processing, forest land conserved, and lower replacement costs (Roche, 1965). These annual savings in 1974 dollars have been calculated by Hartford (1976) for the United states at $867.94/m^3$ ($24.58 per cu.ft.) versus treatment cost of $30.00 - $35.30/m^3$ ($.85 -$1.00 per cu. ft.).

The dollar savings per m^3 are an average of the replacement costs for wood used by utilities, railroads, and in construction. Replacement costs for wood are highest for the utilities, lowest for the railroads, and with those for construction falling midway between these two.

The two basic systems for treating wood with preservatives containing CPs are pressure and non-pressure systems (Table 10). The non-pressure systems listed in Table 10 refer only to those plants applying wood preservatives, not to those applying surface treatments with the Na salts of the CPs for wood protection. Those plants which have pressure treatment facilities can use water-borne preservatives, such as the water soluble formulations containing one or more toxic elements such as copper, zinc, chromium, arsenic, flourine and, in some cases, ammonium hydroxide. They can also use preservatives in oil or organic solvents. The two most widely used oil-borne treatments involve a) creosote-petroleum oil mixtures or b) PCP in oil. Both creosote and PCP are highly toxic to fungi (Shields, 1976).

Creosote is obtained as a by-product of coal-tar distillation, which comes from the coking ovens in the steel industry. Domtar, Inc. is the sole producer of creosote in Canada.

Creosote contains more than 160 compounds, each of which is in itself toxic to wood destroying organisms (Anonymous, 1971). Shields and Stranks (1978) in their summary of creosote's benefits and risks point out that while creosote is toxic to some types of sensitive fish, it is retained in treated wood for years at levels close to those immediately after treatment; therefore, it poses little risk to the aquatic environment. Creosote is widely used as a heavy-duty preservative where dark color and odor are not important factors (Smith 1978).

In the U.S., use of creosote for wood preservation far exceeds that of PCP. In 1972 an estimated 440 million kg of creosote was used compared to only 17.2 million kg of PCP, approximately 4% of the organic wood preservative market (von Rumker et al. 1974).

Pentachlorophenol has been used as a wood preservative since the 1940's, when it was introduced as an alternative and "clean system" to the commonly used creosote

TABLE 10 WOOD PRESERVING PLANTS IN CANADA IN 1979 - LOCALITY, TYPE AND PRESERVATIVES USED (Eastern Forest Products Laboratory, Fisheries and Environment Canada, Ottawa, Ontario).

Region	Pressure Treating									
	O	W	CO	OW	WF	COW	OWF	COWF	Non Pressure	Total
British Columbia	1				1	1	1	1	1	6
Alberta	2		1	2		1			1	7
Saskatchewan	3	1								4
Manitoba		3								3
Ontario		5			2	1	1	2	7	18
Quebec			1-MC			1	1	1		4
Atlantic Provinces		2	1			2				5
Total	6	11	3	2	3	6	3	4	9	47

Legend: C - creosote
O - oil-borne
W - water-borne
F - fire retardent
MC - methylene chloride

(Arsenault, 1978). Shields and Stranks (1978) note that PCP is not readily lost from treated wood. Its permanence is in direct relationship to the volatility of the carrier oil or solvent used; the lower the volatility of the carrier oil the lower the loss of PCP vapor. Treatments, considered clean, are used for wood going into service in windows, doors, plywoods, utility poles, etc. In addition to using light colored oils as carriers, PCP can also be deposited "cleanly" into wood by use of recoverable carriers such as LP gas and methylene chloride (Arsenault, 1978).

The lower chlorinated phenols, TTCP, TCP, and chloro-2-phenylphenol have been used for some special wood preservation applications. These compounds are less desirable than PCP because of one or more of the following factors: undesirable odors, higher volatility, more water soluble, and more likely to cause skin irritation. Both the tri-, and tetra-CPs are registered for use as wood preservatives when in combination with other compounds such as PCP (App. 10).

2.2.2 Agricultural. Agricultural uses parallel those industrial uses already discussed under Sect. 2.2.1, with the prevention of wood decay the primary reason for using PCP and TTCP treatments. In addition to this major use, PCP is also registered under the Pest Control Products Act to treat wood in farm buildings, fences, etc., to control various organisms such as powder post beetles, termites, carpenter ants, molds, chicken mites, and moss on roofs. In combination with specific herbicides it is registered for control of certain weeds. The quantities of PCP used in the agricultural sector are relatively small compared to industrial usage.

2.2.3 Domestic. In the domestic category of products registered under the PCP Act, PCP is one of the materials available to the home-owner for treatment of wood used in fences and building construction for prevention of decay. PCP is marketed both as the main active ingredient in wood preservatives for home use and also as an additive in products such as stains and paints. Domestic sales are a small portion of the overall PCP market, probably less than 1%.

In addition to the industrial, agricultural, and domestic uses of CPs which are regulated by Agriculture Canada, CPs are also used in health care (dental) products and in disinfectants for home, farm and hospital use. Products in these classes, which contain p-CP (4-CP) and PCP come under the jurisdiction of Health and Welfare Canada via the Food and Drug Act. Approximately one dozen products from an equal number of companies contain 1.0 to 100% p-CP in their formulations. Additionally, p-CP is used as an intermediate in the production of chlorophene (o-benzyl-p-CP), which is also used in

disinfectants. PCP is used in three dental care products manufactured by one company. Concentrations of PCP in these products are from 0.1 - 0.22%. Another compound, hexachlorophene (2,2'-methylene bis (3,4,6-TCP)), which is derived from the condensation of 2,4,5-TCP with formaldehyde in the presence of sulfuric acid, is used in disinfectants and sanitation products for hospital, home, and veterinary use. Hexachlorophene is a component of 42 products manufactured by 19 companies. It is incorporated in these products at concentrations of 0.006 - 3.0%. Hexachlorophene is said to contain 0.03 mg/kg or less of 2,3,7,8-TCDD (Rappe et al, 1979b). The volume of CPs used in health care, veterinary, and sanitation products is unknown. Quantities of hexachlorophene imported from the United States in 1976, 1977 and 1979 were 22, 103 and 10 metric tonne, respectively (Statistics Canada, Imports by Commodities and Countries).

3 IMPURITIES IN CHLOROPHENOLS

The presence of toxic impurities in CPs is usually a result of process chemistry. The elevated temperatures (above 95°C) required during the latter stage of chlorination of phenol to PCP, favor formation of polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF). These are tricyclic aromatic compounds which exhibit similar physical, chemical, and biological properties (App. 1, Sect. 1.2.3). These materials, sometimes listed as inerts, and also known as high boilers, are listed as impurities in CP formulations. Qualitative and quantitative data on impurities in CPs are presented in this section.

3.1 Polychlorinated Dibenzo-p-dioxins, Chlorodibenzofurans, and Chlorinated Diphenyl Ethers

PCDDs, PCDFs and polychlorinated diphenyl ethers (PCDPEs) have been identified as common impurities in commercial CPs (Nilsson and Renberg, 1974), (Jensen and Renberg, 1973), (Firestone et al, 1972), (Rappe et al, 1978). In addition, Buser (1976) confirmed that polychlorinated phenoxy phenols, polychlorinated benzenes, and polychlorinated biphenyls could occur as impurities in chlorinated phenols. Rappe et al (1979a) listed the possible number of positional PCDD and PCDF isomers as 75 and 135, respectively (Table 11). The structures and numbering systems for the PCDDs and PCDFs are illustrated in Fig. 3.

TABLE 11 POSSIBLE NUMBER OF POSITIONAL PCDD AND PCDF ISOMERS (Rappe et al, 1979a)

Chlorine substitution	Number of isomers	
	PCDDs	PCDFs
mono-	2	4
di-	10	16
tri-	14	28
tetra-	22	38
penta-	14	28
hexa-	10	16
hepta-	2	4
octa-	1	1
Total	75	135

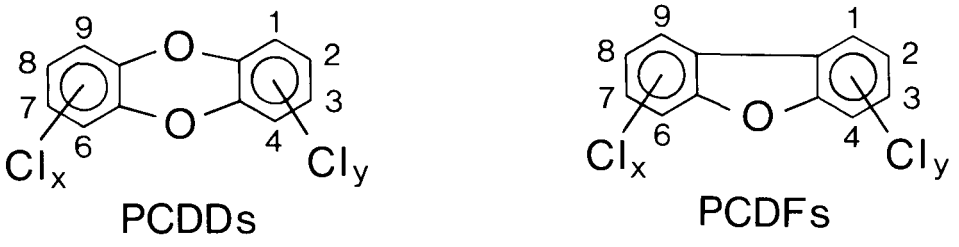


FIGURE 3 STRUCTURES AND NUMBERING SYSTEMS FOR THE PCDDs AND PCDFs (Rappe et al, 1979)

Firestone et al (1972) reported on the quantitative analysis for PCDDs in the CPs (Table 12), and also identified PCDFs and PCDPEs in the CPs (Tables 13, 14).

Woolson et al (1972) identified hexa-, hepta-, and octachlorodibenzo-p-dioxins (HCDD, HpCDD, and OCDD, respectively) in TCP, TTCP, and PCP. They state that the TCP samples contained only small amounts of HCDD and no sample contained over 10 ppm. TTCP contained less than 100 ppm of HCDD, HpCDD, and OCDD, while six of the 20 PCP samples contained >100 ppm to <1000 ppm of the HpCDD and OCDD isomers. No TCDD was detected in any samples of CPs at levels above 0.5 ppm, the minimum level of detection.

Villanueva et al (1973) reported on the level of HCDD, HpCDD and OCDD in samples of technical PCP (86% PCP) and in analytical grade PCP (95% PCP). The technical product was more contaminated than the analytical one by factors of 1400, 600, and 539 for the HCDD, HpCDD and OCDD, respectively.

TABLE 12 POLYCHLORODIBENZO-p-DIOXINS IN MONO-, DI-, TRI-, TETRA-, AND PENTACHLOROPHENOLS BY ELECTRON CAPTURE GAS CHROMATOGRAPHY[a] (Firestone et al, 1972)

Sample[b]	Mfr.	Date Recd	Dioxin	Found (ppm)
1 2-CP		4/67	none	-
2 2,4-DCP		4/70	none	-
3 2,6-DCP		-	none	-
4 2,4,5-TCP-Na	1	9/67	none	-
5 2,4,5-TCP-Na	1	6/69	2,7-dichloro 2,3,7,8-tetrachloro[d]	0.72 1.4
6 2,4,5-TCP	1	6/69	1,3,6,8-tetrachloro[d] 2,3,7,8-tetrachloro[d]	0.30 6.2
7 2,4,5-TCP	2	7/70	pentachloro	1.5
8 2,4,5-TCP	2	7/70	none	-
9 2,4,5-TCP	3	7/70	2,3,7,8-tetrachloro	0.07
10 2,4,6-TCP		-	2,3,7-trichloro 1,3,6,8-tetrachloro	93 49
11 2,3,4,6-TCP	1	-	hexachloro[d] hexachloro[d] heptachloro[d] octachloro	15 14 5.1 0.17
12 2,3,4,6-TCP	c	3/67	hexachloro[d]	4.1
13 2,3,4,6-TCP	c	-	none	-
14 PCP-Na	1	9/67	hexa[d] hepta[d] hepta[d] octa	14 5.4 9.1 3.8
15 PCP-Na	5	6/69	hexa[d] hepta[d] hepta[d] octa	20 1.3 10 3.3
16 PCP	4	5/70	hexa[d] hexa[d] hepta[d] hepta[d] octa	0.96 38 10 39 15
17 PCP	4	7/70	hexa[d] hepta[d]	35 23
18 PCP		3/67	hexa hexa	0.03 0.14

TABLE 12 (Continued) POLYCHLORODIBENZO-p-DIOXINS IN MONO-, DI-, TRI-, TETRA-, AND PENTACHLOROPHENOLS BY ELECTRON CAPTURE GAS CHROMATOGRAPHY[a] (Firestone et al, 1972)

Sample[b]	Mfr.	Date Recd	Dioxin	Found (ppm)
19 PCP	5	6/69	hexa[d]	13
			hepta[d]	12
			hepta[d]	35
20 PCP	6	5/70	hexa[d]	0.91
			hepta[d]	0.50
			hepta[d]	1.6
			octa[d]	5.3
21 PCP	7	7/70	hexa[d]	15
			hepta[d]	23
			octa	15

[a]See text for conditions. (Firestone et al, 1972).

[b]2-CP = 2-chlorophenol; 2,4-DCP = 2,4-dichlorophenol; 2,6-DCP = 2,6-dichlorophenol; 2,4,5-TCP-Na = 2,4,5-trichlorophenol sodium salt; 2,4,5-TCP = 2,4,5-trichlorophenol; 2,4,6-TCP = 2,4,6-trichlorophenol; 2,3,4,6-TCP = 2,3,4,6-tetrachlorophenol; PCP-Na = pentachlorophenol sodium salt; and PCP = pentachlorophenol

[c]Obtained from laboratory chemical supplier.

[d]Confirmed by combined gas chromatography-mass spectrometry.

TABLE 13 DETECTION OF POLYCHLORODIBENZOFURANS IN CHLOROPHENOLS BY COMBINED GAS CHROMATOGRAPHY-MASS SPECTROMETRY[a] (Firestone et al, 1972)

Sample[b]		Chlorofurans 3 Cl	4 Cl	5 Cl	6 Cl	7 Cl	8 Cl
1	2-CP		+				
2	2,4-CDP						
3	2,6-DCP						
4	2,4,5-TCP-Na						
5	2,4,5-TCP-Na	+[c]	+[d]	+[e]			
6	2,4,5-TCP		+[d]				
7	2,4,5-TCP						
8	2,4,5-TCP	+					
9	2,4,5-TCP						
10	2,4,6-TCP		+	+	+		
11	2,3,4,6-TCP				+	+	+
12	2,3,4,6-TCP			+	+		
13	2,3,4,6-TCP		+		+		
14	PCP-Na				+	+	
15	PCP-Na			+	+	+	
16	PCP				+	+	
17	PCP				+		
18	PCP						
19	PCP		+	+	+	+	
20	PCP				+	+	+
21	PCP				+		

[a] See text for conditions (Firestone et al, 1972).

[b] See Table 12 footnote b for description of sample abbreviations.

[c] Data suggest component is a trichlorodimethoxy-dibenzofuran.

[d] Data suggest component is a tetrachlorodimethoxy-dibenzofuran.

[e] Data suggest component is a pentachlorodimethoxy-benzofuran.

TABLE 14 DETECTION OF POLYCHLORODIPHENYL ETHERS IN CHLOROPHENOLS BY COMBINED GAS CHROMATOGRAPHY-MASS SPECTROMETRY[a] (Firestone et al, 1972)

Sample[b]		Polychlorodiphenyl Ethers							
		3 Cl	4 Cl	5 Cl	6 Cl	7 Cl	8 Cl	9 Cl	10 Cl
1	2-CP								
2	2,4-DCP								
3	2,6-DCP								
4	2,4,5-TCP-Na								
5	2,4,5-TCP-Na				+[c]				
6	2,4,5-TCP								
7	2,4,5-TCP								
8	2,4,5-TCP								
9	2,4,5-TCP								
10	2,4,6-TCP	+	+	+	+	+	+		
11	2,3,4,6-TCP			+	+	+	+		
12	2,3,4,6-TCP[d]			+	+	+	+		
13	2,3,4,6-TCP			+	+	+	+		
14	PCP-Na				+	+	+		
15	PCP-Na			+	+	+	+	+	+
16	PCP				e	+	+	+	
17	PCP			+	+	+	+	+	
18	PCP								
19	PCP		+	+	+	+			
20	PCP				+	+	+	+	
21	PCP				e	+	+	+	

[a]See text conditions (Firestone et al, 1972).

[b]See Table 12 footnote b for description of sample abbreviations.

[c]Data suggest component is a hexachloromethoxydiphenyl ether.

[d]Data suggest that tetrachlorobenzofuran and pentachloroanisole are also present.

[e]Data suggest that a hexachlorohydroxybiphenyl is present.

Johnson et al (1973) reported on the levels of HCDD and OCDD in commercial grade PCP. HpCDDs, and HCDF, HpCDF, and OCDFs were identified qualitatively but lack of appropriate standards did not allow quantitative analyses. As expected, Johnson et al (1973) did not detect any 2,3,7,8-TCDD in the samples because the appropriate precursors are not present in PCP.

Nilsson and Renberg (1974) reported on impurities found in three chlorinated phenol formulations, 2,3,4,6-potassium TTCP; 2,4,6-TCP and 2,4,6-potassium TCP, which are manufactured from phenol by direct chlorination. Quantitative data were obtained by use of gc-tlc and combined gc-ms analysis. The main impurities (1 - 5%) were chlorinated 2-hydroxydiphenyl ethers (pre-dioxins), precursors to CDDs, and 4-hydroxydiphenyl ethers (isopredioxins). Further impurities identified were chlorinated dihydroxybiphenyls, and PCDFs with the PCDFs at levels of 10 - 100 ppm, PCDPEs estimated to be in the range of 100 - 1000 ppm.

Nilsson and Renberg (1974) point out that the concentration of the highly toxic CDFs was at the same level as had been found in some polychlorobiphenyl (PCB) formulations. Identification and levels of PCDFs in American PCBs have been reported by Bowes et al (1975a, 1975b). Nilsson and Renberg (1974) suggested that the dihydroxybiphenyls and the diphenyl ethers were possible precursors of CDFs, both in the industrial process and in the environment.

Goldstein et al (1977) quantitatively analyzed a sample of technical PCP from Monsanto used in a study on effects of contaminants in PCP on hepatic drug-metabolizing enzymes and porphyria (Table 15). They noted that TCDD and PnCDD were not detected in the technical PCP sample by gc-ms equipment at a detection limit of 0.1 ppm. Since the Monsanto PCP is no longer produced, the contaminant levels in their product are primarily of academic interest.

A process for synthesizing an improved PCP, low in impurities, has been developed by Dow (Watson and Kobel, 1974a, 1974b), (Yoshimine and Kobel, 1974). The compositions of the commercial and improved, PCPs are compared in Table 16.

Data, previously published by Buser and Bosshardt (1976), were presented by Firestone (1977) to indicate levels of PCDDs and PCDFs in PCP from a domestic PCP supplier, Dow Chemical Co. (Table 17).

Firestone (1977) also presented information on the results of analyses of hexa- and octachlorodibenzo-p-dioxin in various U.S. domestic PCPs (Table 18).

TABLE 15 CHEMICAL ANALYSIS* OF TECHNICAL PENTACHLOROPHENOL (excerpted from Goldstein et al, 1977)

Compound	Concentration
Pentachlorophenol	84.6%
Tetrachlorophenol	3%
Hexachlorodibenzo-p-dioxin	8 ppm
Heptachlorodibenzo-p-dioxin	520 ppm
Octachlorodibenzo-p-dioxin	1380 ppm
Tetrachlorodibenzofuran	≤4 ppm
Pentachlorodibenzofuran	40 ppm
Hexachlorodibenzofuran	90 ppm
Heptachlorodibenzofuran	400 ppm
Octachlorodibenzofuran	260 ppm

*Analysis by gc-ms. The lower detection limit was 0.1 ppm.

TABLE 16 COMPOSITION OF COMMERCIAL AND IMPROVED PENTACHLOROPHENOL (Firestone, 1977)

Component	Commercial[a]	Improved[b]
Pentachlorophenol	88.4%	89.8%
Tetrachlorophenol	4.4%	10.1%
Trichlorophenol	< 0.1%	< 0.1%
Chlorinated phenoxyphenols	< 6.2%	-
Octachlorodibenzo-p-dioxin	2500 ppm	15.0 ppm
Heptachlorodibenzo-p-dioxins	125 ppm	6.5 ppm
Hexachlorodibenzo-p-dioxins	4 ppm	1.0 ppm
Octachlorodibenzofurans	80 ppm	< 1 ppm
Heptachlorodibenzofurans	80 ppm	1.8 ppm
Hexachlorodibenzofurans	30 ppm	< 1 ppm

[a]Dowicide 7, sample 9522A.

[b]Dowicide EC-7.

TABLE 17 CHLORODIOXINS AND CHLOROFURANS IN DOW PCP PRODUCTS (Firestone, 1977)

Sample	PCDD[a] (ppm)			PCDF[b] (ppm)				
	Hexa-	Hepta-	Octa-	Tetra-	Penta-	Hexa-	Hepta-	Octa-
PCP (EC-7)	0.15	1.1	5.5	0.45	0.03	0.3	0.5	0.2
PCP (EC-7)	0.03	0.6	8.0	<0.02	< 0.03	< 0.03	< 0.1	< 0.1
PCP[c]	9.5	125	160	<0.02	0.05	15	95	105
PCP[c]	9.1	180	280	0.05	0.25	36	320	210
PCP-Na[c,d]	3.4	40	115	<0.02	0.05	11	50	24
PCP	10.0	130	210	0.20	0.20	13	70	55
PCP	5.4	130	370	0.07	0.20	9	60	65

[a]PCDD = polychlorodibenzo-p-dioxin.

[b]PCDF = polychlorodibenzofuran.

[c]A Dow product; supplied by Fluka, a laboratory chemical supplier.

[d]PCP-Na = sodium pentachlorophenate.

TABLE 18 HEXA- AND OCTACHLORODIOXINS IN DOMESTIC PCPs (Firestone, 1977)

Sample	Mfr.	Hexachlorodioxin (ppm)[a]	Octachlorodioxin (ppm)[b]
1	Vulcan	10	1700
2	Vulcan	ND[c]	ND
3	Vulcan	15	2500
4	Vulcan	16	3600
5	Reichhold	20	700
6	Reichhold	17	600
7	Reichhold	23	900
8	Reichhold	ND	ND
9	Monsanto	15	1400
10	Monsanto	12	1100
11	Monsanto	15	1900
12	Dow	ND	2
13	Dow	ND	2
14	Dow	ND	ND
15	Dow	16	1500
16	Dow	16	1800
17	Dow	21	3400

[a]Detection limit 0.3 ppm, except for Sample 8 which is 2 ppm.

[b]Detection limit 1 ppm, except for Sample 8 which is 6 ppm.

[c]ND = not detected.

Firestone (1977) compiled the known information on levels of the individual HCDD and HpCDD isomers in PCP and NaPCP samples from Dow; however, the analysis did not include the improved PCP (Dow EC-7) formulation currently under development.

Buser (1976) and Buser and Bosshardt (1976) reported the presence of a TCDD at levels of <0.01 to 0.23 ppm in samples of commercial PCP. In the same samples of PCP they had also detected penta- to octa-CDD.

Rappe et al (1978) analyzed for PCDF content, in 2,4,6-TCP and 2,3,4,6-TTCP, two of the most commonly used CP formulations on the Scandinavian market. In addition a PCP from the U.S. was analyzed. (Note: although all the products analyzed were referred to as chlorophenates, they may or may not have been the alkali salts of their respective CPs). Both the TCP and TTCP were prepared by the chlorination of phenol. The production method for the PCP was not known. Rappe et al (1978) noted that although the CPs differed as to origin and synthesis, the same PnCDF, HCDF, and HpCDF isomers were found as the main PCDF components in all three samples, although in somewhat different proportions. The main isomers were 1,2,4,6,8-PnCDF, 1,2,3,4,6,8-, 1,2,4,6,7,8- and 1,2,4,6,8,9-HCDF, and 1,2,3,4,6,7,8- and 1,2,3,4,6,8,9-HpCDF. Combined levels which had been determined for the various PCDFs are given in Table 19.

TABLE 19 LEVELS OF POLYCHLORINATED DIBENZOFURANS (PCDFs) AND TOTAL POLYCHLORINATED DIBENZO-p-DIOXINS (PCDDs) IN CHLORINATED PHENOLS (μg/g = ppm) (Rappe et al, 1978)

		PCDFs					Totals	
Chlorophenol	Origin	tetra-	penta-	hexa-	hepta-	octa	PCDFs	PCDDs
2,4,6-tri	Scand.[b]	1.5	17.5	36	4.8	-	60	< 3
2,3,4,6-tetra	Scand.	< 0.5[a]	10	70	70	10	160	12
penta	U.S.	0.9	4	32	120	130	280	1000

a This product had a high level of polychlorinated diphenyl ethers (PCDPEs). The quantification of tetra-CDFs is disturbed by the interference of hexa-CDPEs.

b Scandinavia

4 ROUTES OF ENTRY OF CHLOROPHENOLS INTO THE ENVIRONMENT

The most obvious routes of entry for CPs into the environment are at the wood preserving plant sites which are usually located close to water. This section includes comparative information on the processes used for treating wood and an indication as to the volume of waste water generated. Information is also included on the less obvious routes in which CPs can enter or be formed in the environment, such as via the chlorination of water.

4.1 Primary Routes

4.1.1 Wood Preserving Plants, and their Treatment Systems. Since the CPs are primarily used as anti-microbials or preservatives in the wood processing industry, it is logical to consider the possibilities for escape of these compounds into the environment at one or more points along the manufacturing or wood processing stream, as well as from treating wood at in-service sites.

As with all other chemical compounds, CPs may enter the environment at the manufacturing or use site through either intentional disposal or accidental dispersal. In the case of the former, it may be done with or without knowledge of the consequences. Although it is difficult to document, this type of deliberate disposal can occur in the manufacturing process, at wood treatment plants or during on-site treatments with wood preservatives. Intentional release of CPs into the environment can also occur when wastes, which have not been fully treated, are discarded.

Environmental contamination by accidental dispersal of CP products may occur, from manufacturing mishaps, such as overflows, spills from ruptured or broken processing equipment and lines, and from wastes from plant clean-up operations. Leakage from broken containers during warehousing, storage and shipping operations may also be a cause of contamination. The volumes of product involved in such an occurrence are likely to be small, but it may be significant as a point source for a local area, particularly if it becomes part of an uncontrolled and untreated effluent going into a drainage system.

Shields (1976) states that non pressure preservation treatments, usually carried out on-site in forests, fields, mills and construction sites, but also at treating plants, can cause local pollution. However, data on environmental impact are lacking. On the other hand, Shields (1976) states that "Pressure treatments usually impose a sequence of hydrostatic pressures and vacuum on the wood. Since these treatments can only be done

at commercial plants, information on waste disposal and treatment procedures is generally more readily available".

Arsenault (1978) has summarized the various processes available for both conditioning and treating wood. Conditioning is usually carried out in the retort in which the wood is impregnated. The processes which include CPs as part of the treatment are as follows:

1) Boultinizing- a drying process using heat and vacuum, normally used for Douglas fir poles. "This method of conditioning wood in the treating cylinder consists of heating wood while submerged in hot creosote or penta/oil solution while the cylinder is evacuated. The preservative is heated to 180-210°F before any vacuum is applied. Since the boiling point of water is lower under a partial vacuum (22 in.Hg or more) than at atmospheric pressure, drying can proceed rather rapidly at temperatures below 212°F. Generally the temperature of the oil is 210 to 220°F for a time period of anywhere from 10 hours to 50 hours depending on the moisture content and size of the poles. Longer heating times result in all of the ultimate checking occurring in the cylinder before treatment which insures adequate penetration into the checks and longer service life of the poles."
2) Steam conditioning- not a drying process, though some water is removed in the process. This process can be used for green wood pretreatment, if it is followed by penta/hot oil under pressure. Live steam is injected into a closed retort which contains poles, piling or lumber. The steam is followed by a vacuum, until sufficient moisture has been removed. The wood is then ready for treatment.
3) Thermal process- The wood is immersed in a hot penta/oil solution, followed by a cold solution which results in a partial vacuum in the cells of the wood. This system is used for cedar poles and other forest products, made from Douglas fir, and various species of larch, pine, and cedar.
4) Pressure system- a "full cell" process using penta/oil solution as in the Bethell process, and the "empty-cell" system as in the Rueping and Lowry processes.

The process waters from the wood treatment plants are sources of CP pollution if the waters are not treated either biologically or chemically (App. 9).

Contamination of process water from pressure treatment cylinders occurs when condensate from a steaming cycle flows down the walls of the retort and entraps residues of PCP from the previous preservative treatment cycle and then leaves the cylinder through the steam trap. Steaming generates more wastewater than Boultinizing

although both processes are primary sources of potential polluting materials (Shields, 1976).

Another main source of contaminated waters is the cooling waters from barometric condensers. Shields (1976 indicates that the volume of water from this source is frequently too large to be treated economically.

Secondary sources for contaminated water include vacuum water, storm water from areas saturated with preservatives, wash water, boiler blowdown water, and condensate from heating coils (Shields, 1976).

Some of the many variables which affect the volume of wastewater from wood preservation plants include the volume of wood preserved, the amount of green wood conditioning required, the type of preservative used, whether or not barometric condensers are used, the amount of dilution from precipitation, soil percolation, and spills (Shields, 1976).

Shields (1976), who considers that retort condensation waters are the most important source of contaminated water, estimates that there is usually less than 68,200 L (68 m^3) per day of wastewater from a plant employing open steam conditioning. Contaminated water from a closed steaming system would be in the order of 4,500 to 9,100 L/day/retort (Richardson, 1978; Shields, 1976). Not more than 9,100 L (9 m^3) per day of wastewater should be produced at a Boultonizing plant.

Customarily, wastewaters are described according to their BOD, COD, pH, and phenol, oil, and solids content. Effluent from Canadian wood preserving plants, in the majority of cases has either not been analyzed for levels of CPs, including PCP, or the findings have not been reported in the literature.

This lack of current, specific information on quantity and quality of effluent from the wood preservation plants has been noted by the Water Pollution Control Directorate, Environmental Protection Service, Environment Canada, in a draft report (1977-80) on "A Preliminary Discussion Paper on Environmental Controls for the Canadian Wood Preservation and Protection Industry".

4.1.2 Wood Protection Facilities. Wood protection (surface treatment of wood for sap stain protection and mold control) is carried out with water soluble salt formulations of CPs at sawmills and lumber export terminals. Applications of chemicals to the wood are made by either dipping or spraying, with dipping being the most common method in 1980. Dipping of lumber may be done by use of drive-in lumber carriers, crane-dip, automatic conveyor, or other equipment. In British Columbia the dipping systems

utilizing CPs have been identified by the Environmental Protection Service, Environment Canada, as a serious environmental hazard due in part to their inadequate design and operation, and in part to the large number of facilities located at environmentally vulnerable sites. Incidents harmful to the environment and involving CPs in dip tank operations in British Columbia are reported in Sect. 5.1.1.

4.2 Other Routes

4.2.1 In-Service Treatments with Preservatives. When CP wood preservatives are applied on-site to wood products already in-service, there is always the potential for contamination of the environment. Not many of these occurrences have been documented, which may or may not, reflect the true situation. However, there was one case-history recorded in 1972 from British Columbia. A hydro pole was treated in place with a PCP preservative, and a nearby stream received a shock load of PCP, resulting in a fish kill for a distance of 800 m downstream (Alderdice 1978, personal communication).

4.2.2 Petrochemical Drilling Fluids - Dispersal from Sumps. Another source of environmental contamination by CPs is the petrochemical drilling fluids, which include bactericides to prevent fermentation of the polysaccharides, starch and XC polymer (Land, 1974) (Falk and Lawrence, 1973). When NaPCP is used for this purpose it is maintained at a concentration of 700 -1400 ppm in the drilling fluid, which is the same concentration as for formaldehyde when it is used for the same purpose. The used drilling fluids and associated wastes are contained in large excavations or sumps. Sumps will vary in size according to effluent storage needs; volumes of drilling wastes in sumps following a partial season's operations, might fall in the range of 2,800 - 11,300 m^3. The sumps are often subject to flooding and washing-out with the resultant release of toxic materials to the surface waters of the area. No specific data are available on the volume of NaPCP used in drilling fluids. As an example of the amount of drilling fluids used during a partial season of drilling, two sites in the Canadian Arctic from May to mid-August 1972, used 1.1 to 1.2 x 10^6 kg (Falk and Lawrence, 1973). This amount represents the total kilograms of drilling fluid components, including water.

4.2.3 Aqueous Chlorination. In 1977 the production of chlorine in Canada was 9.0 x 10^8 kg while in the United States it was 9.6 x 10^9 kg, for a total of 10.5 x 10^9 kg (Donnan, 1979). The amount of chlorine used by the pulp and paper industry has been estimated for Canada and the United States as 60% and 15%, respectively, of their total production, or 5.3 x 10^8 kg and 1.4 x 10^9 kg, respectively (Donnan, 1979). In Canada

approximately 1.7% (1.5×10^7 kg) of the chlorine produced is used for sanitary purposes including treatment of potable water, wastewater, swimming pools, cooling water circuits, food packaging process water and in household sanitation products (Donnan, 1979); however, other estimates state water treatment in Canada could utilize 9×10^7 kg of chlorine/yr (Anonymous, 1976). In the United States, approximately 4% (3.8×10^8 kg) of the chlorine production is used for these types of water treatment (White, 1976).

The total amount per year of residual chlorine discharged to Canadian waters is unknown as is the amount of this chlorine incorporated into organic molecules present in these surface waters.

A review of aqueous chlorination of organic compounds and their chemical reactivity and effects on environmental quality has been authored by R.C. Pierce (1978) of the National Research Council. He states that the interaction of aqueous chlorine with appropriate organic molecules will produce halogenated organic compounds including halophenols and related derivatives, halogenated nitrogenous substances such as chlorinated amino acids and nucleic acid bases, and trihalogenated methane derivatives. Arsenault (1976) has commented on the ease of chlorination of phenol by noting that municipal chlorination of drinking water can give rise to CPs in the ppb range. In addition to formation of the lower chlorinated CPs, a 10 ppm level of chlorine can chlorinate 1 ppm of naturally occurring phenol and generate 0.2 ppb of PCP. The halophenols, of which the CPs are a part, have potential adverse biological effects. They tend to reduce biodegradability, increase bioaccumulation and are already, at their present levels in water and commercial protein products from these waters, affecting taste and odour. Pierce (1978) anticipated that toxicity problems would also be traced to these compounds. In addition to current research on the release, environmental stability, quantification, and effect on environmental quality of halophenols in a freshwater environment, Pierce (1978) recommended that a productive area of research would be in dealing with the chemistry and toxicity of halogens and halogen containing organic compounds in the marine environment because of increasing use of seawater for disposal of chlorinated wastes and chlorinated cooling waters.

Also, Gibson and Bourquin (1977) noted that "Much information is needed on the quantity and types of halogenated products generated in estuarine and marine waters before a proper assessment of their fate is possible". These authors further stated that in addition to organochlorine compounds from industry and agriculture being artifically introduced into the environment in enormous quantities, "Compounds containing the

carbon-halogen bond are widespread in the environment being naturally formed by marine algae, certain other plants and soil microorganisms."

4.2.4 Incineration. Olie et al (1977) reported on an investigation of environmental loading with organic pollutants by waste products of municipal incinerators. They observed that the most abundant chlorine containing compounds in condensate from flue gas were the di-, tri-, and tetra- CPs. Smaller quantities of the PCDDs were also detected (Sect. 6.2). Although the authors were unable to estimate the quantities of CPs or PCDDs which might be leaving the stacks of incinerators because of the many variables involved, they did conclude that: "Municipal incinerators and other combustion processes may be a source of some of the organochlorine compounds in the environment."

5 RESIDUES OF CHLOROPHENOLS AND THEIR TRANSFORMATION PRODUCTS IN THE ENVIRONMENT

The available information and data on residues of CPs in the Canadian environment, both aquatic and terrestrial, are presented in this section. Information from other geographical areas, particularly the United States, is also included to help indicate the magnitude of the residue problem and to demonstrate the ubiquitous nature of CPs in the environment, including the atmosphere.

5.1 Residues in Aquatic Systems

The relationship between the physical characteristics of the CPs, such as solubilities (App. Sect. 1.1.2), the transport of the CPs in an aquatic environment, and their uptake and concentration in aquatic organisms can only be understood following the detection and quantification of CP residues. The information on residues in the aquatic system will be presented under the general headings of water, sediments, plants, and animals.

5.1.1 Water

Canada

The available information on levels of CPs in surface waters of Canada is extremely limited, considering the numbers of wood treatment plants and other industries which have been using CPs for several years. This deficiency of data would indicate an apparent lack of monitoring for these compounds and/or the present and past difficulty of identification of individual organic compounds in environmental samples.

In the spring of 1969, the effluent from a Domtar wood-preserving plant in Newcastle, N.B., was monitored and analyzed almost daily for two months during the time period of the plant's operation. PCP levels ranged from a trace (<0.5 mg/L) to 18.3 mg/L (Zitko and Carson, 1969). Other CPs tentatively identified in effluent samples included 2,3,4,6-TTCP and 2,4,5-TCP. Following the installation of additional treatment facilities, effluents were brought down to non-toxic levels.

PCP was detected in water samples from the Salmon River, Truro, N.S. The samples, which were taken below a pole and lumber treating plant in May and July 1976, had PCP levels of 0.38 and 0.008 µg/L, respectively (NAQUADAT File, Water Quality Branch, Inland Waters Directorate, Environment Canada, Ottawa).

Bacon (1978) identified CPs in the effluent of a Kraft pulp mill at St. John, N.B., during an investigation into the bioaccumulation of toxic compounds in pulpmill effluent by aquatic organisms in the receiving waters (see Sect. 5.1.4.1, and Sect. 5.1.4.2). Samples from the Kraft mill effluent consisted of the combined chlorination and extraction effluents from the first stage of the bleaching process. The organic extract (oil) from the Kraft mill effluent, whose initial pH was approximately 2.4, contained various levels of 2,4-DCP and 2,4,6-TCP (Table 20). PCP was not identified in the effluent. No chlorinated compounds were detected in the receiving water samples. Bacon (1978) attributed the non-detection of CPs in the receiving water samples to the very large tidal flushing in the study area resulting in a very large dilution factor for any compounds released into the water. The study area was unique in that it was situated in the lower St. John River estuary, partially occluded by a sill, and subjected to a reverse flow action with a large inflow of seawater. Therefore effluent from the Kraft mill (Mill II, Figure 4) situated near the sill is carried both upstream and downstream.

Other areas in Canada where CPs have been identified in water samples, and where the data have been published, are in Ontario and British Columbia.

In 1977, as part of a Great Lakes Survey for organic compounds that impart taste and odour problems, two sets of water samples taken by the Water Resources Branch of the Ontario Ministry of the Environment were analyzed for phenols and acids. Robinson and Smillie (1977) reported that in 11 samples from the St. Mary's River, i.e., the outlet of Lake Superior, in the vicinity of the Algoma Pulp and Paper Mill, no CPs were identified. It should be noted that their analytical method would not show p-substituted phenols (Fox, 1978b). Of 10 water samples from Thunder Bay (Lake Superior), near the Abitibi Power and Paper Mill, one sample had 4 µg/L of DCP and two samples had 3 and 23 µg/L of TCP.

A more extensive survey was conducted in 1977 by the Canada Center for Inland Waters to determine the presence and levels of PCP in the Great Lakes Basin. Results of the survey were summarized by Fox (1978a) as follows:

> "In 1977, 85 whole bulk water samples from stream mouths, nearshore areas adjacent to stream mouths and interconnecting rivers and channels on the Canadian shores of the Great Lakes were analyzed for pentachlorophenol. Levels of pentachlorophenol ranging from <5 ng/L to 1400 ng/L (with transient highs of up to 23,000 ng/L after periods of heavy rainfall) were observed. Only 8 sites produced samples with no detectable pentachlorophenol. The highest

TABLE 20 CHLOROPHENOLS IN KRAFT PULP MILL EFFLUENT NEAR ST. JOHN, N.B. (adapted from Bacon, 1978)

Source[1] and sample date	mg oil (organic extract) in Bleachery Effluent/L	Chlorophenol concentration (µg/g oil) 2,4-DCP	2,4,6-TCP
Kraft after 30% ClO_2, Feb. 28, 1978	220	131	3.7
Kraft, Jan. 13, 1978	190	195	47
Kraft, 15% ClO_2, June 1977	200	422	131
Kraft, Aug. 28, 1977	478	655	117
Kraft, before 15% ClO_2, Jan. 1978	50	31	0.6
Kraft, after 15% ClO_2, Jan. 1978	195	182	11

[1]Samples were taken from effluent either prior to or following chlorination and caustic extraction; DCP - dichlorophenol, TCP - trichlorophenol.

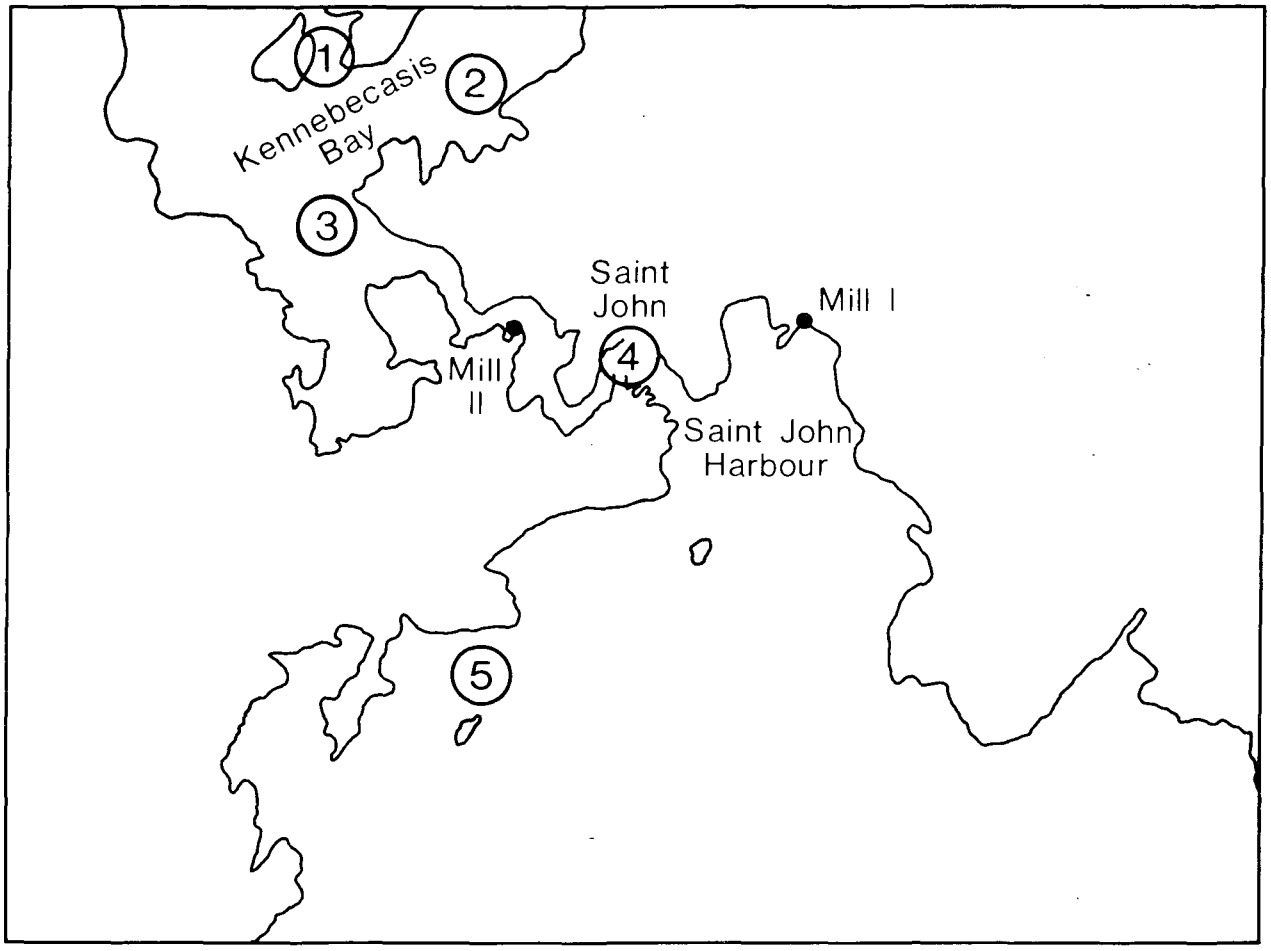


FIGURE 4 MAP OF VICINITY OF ST. JOHN, N.B., SHOWING SAMPLE SITES USED IN STUDY OF BIOACCUMULATION OF TOXIC COMPOUNDS (Bacon, 1978)

Sites in brackish water above the sill:

(1) Milkish Channel
(2) Goat Island
(3) Boar's Head

Site in marine water in the harbour:

(4) Harbour Bridge

Sites in marine water outside the harbour:

(5) Manawagonish Island

Mill I Ground wood pulp mill

Mill II Kraft mill

levels occurred in watersheds along the Lake Erie and Lake Ontario shorelines (Figures 5-9).

"Thirteen sewage effluent samples from 7 treatment plants in southern Ontario were also analyzed for pentachlorophenol which was observed in all samples, ranging from 65 ng/L to 1,300 ng/L." (Figure 10).

As one part of a study on toxic substances in the Great Lakes, water samples obtained in June 1978 from Thunder Bay, Marathon and the Michipicoten areas of Lake Superior were analyzed for PCP, for both "dissolved" and suspended loads (Strachan, 1979b). The average concentration of PCP in the lake water at Thunder Bay and Marathon was approximately 11.0 µg/L and at Michipicoten it was approximately 29.0 µg/L. These figures are above "background interference" and are statistically significant.

As an illustration of levels of PCP that could occur in ground water as a result of seepage of PCP from a wood treatment facility, ground water extracted by well point system on-site at the Abitibi-Price Northern Wood Preservers Ltd., Thunder Bay, had PCP concentrations of 2.05 - 3.35 mg/L (Thompson et al, 1978).

Snow pack samples collected during the winter of 1977-78 from 19 locations in Ontario were analyzed for the presence of toxic substances including PCP (Strachan, 1979a). Trace to micro amounts of PCP, ie. <0.001 µg/L to 0.003 µg/L of snow melt, were detected in samples from 8 of 19 sites. Sites which were positive for PCP ranged from Pt. Pelee National Park in the southern-most tip of Ontario to Fushimi Lake Provincial Park, near Hearst, and Kettle Lake Provincial Park, near Timmins, both of which are in the Hudson Bay watershed. It was concluded that the presence of PCP in the snow demonstrated that aerial transport of PCP occurs year-round.

In British Columbia in 1972, as a result of a fish kill from misapplication of PCP in oil to a hydro power pole near the Little Campbell River in Surrey, water samples were taken to establish levels of PCP remaining following the fish kill. Two days after the introduction of the PCP to the stream, water samples from 27 m downstream from the pole treatment site contained 53.75 ppm of PCP. Seven days later water samples from the same site contained 80 ppb of PCP (Alderdice, 1978).

In addition to the fish kill in the Little Campbell River there have been three other reported fish kills in B.C. salmon waters in the 1957 - 1973 period, which were attributed to CPs. These were reported by McKenzie et al (1975) from data compiled by the Fisheries Service, Environment Canada. In 1963, PCP was suspected in a fish kill in

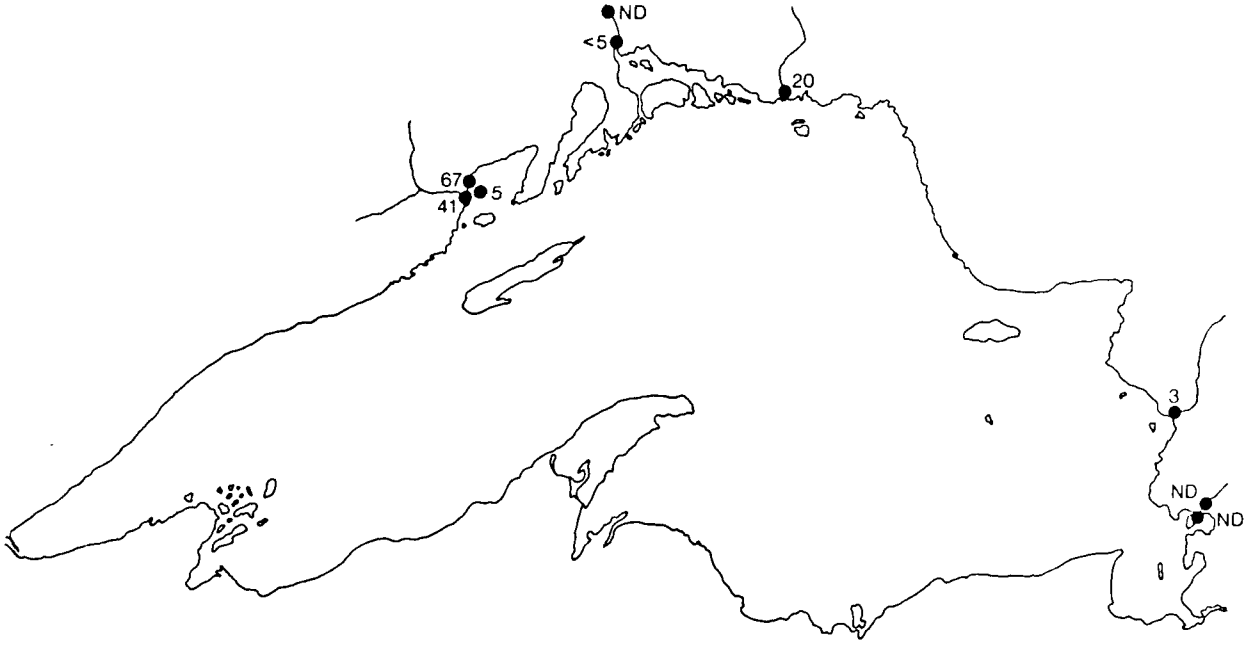


FIGURE 5 PENTACHLOROPHENOL IN THE LAKE SUPERIOR BASIN
(Fox, 1978a)
(pentachlorophenol levels expressed as ng/L of bulk whole water; ND = not detectable)

FIGURE 6 PENTACHLOROPHENOL IN THE LAKE HURON BASIN
(Fox, 1978a)
(pentachlorophenol levels expressed as ng/L of bulk whole water;
ND = not detectable)

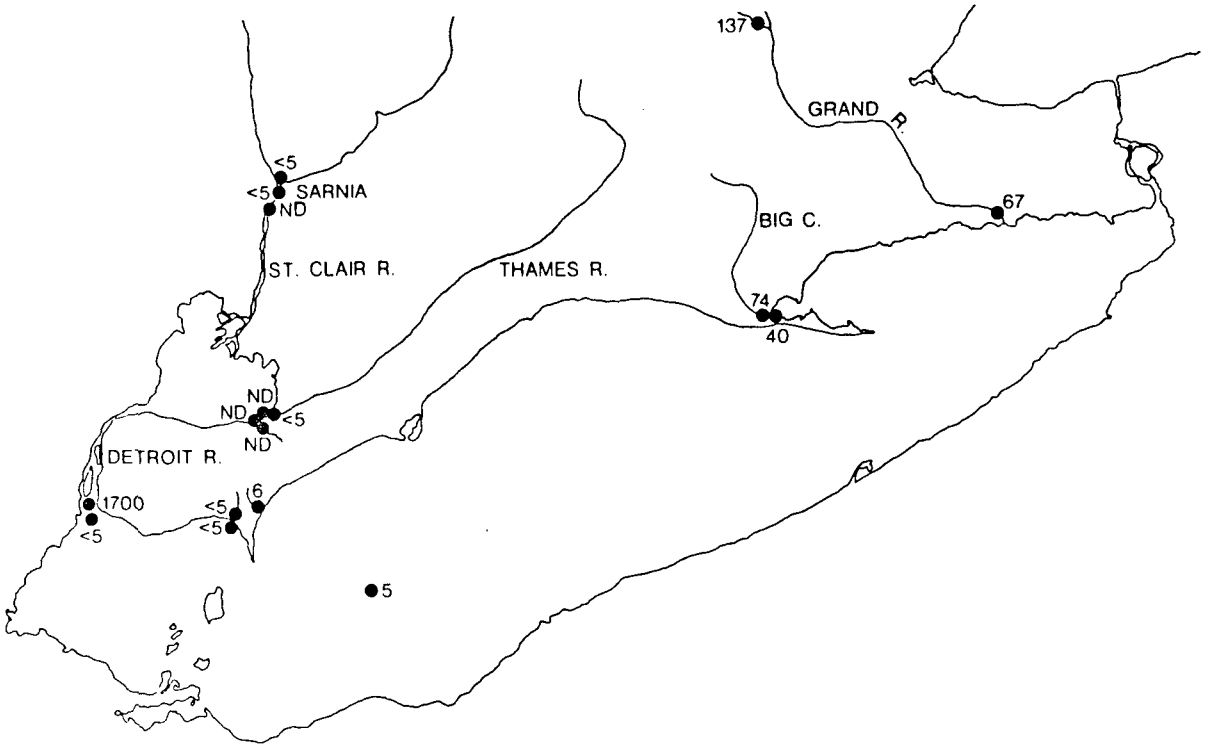


FIGURE 7 PENTACHLOROPHENOL IN THE LAKE ERIE AND LAKE ST. CLAIR BASINS (Fox, 1978a)
(pentachlorophenol levels expressed as ng/L of bulk whole water; ND = not detectable)

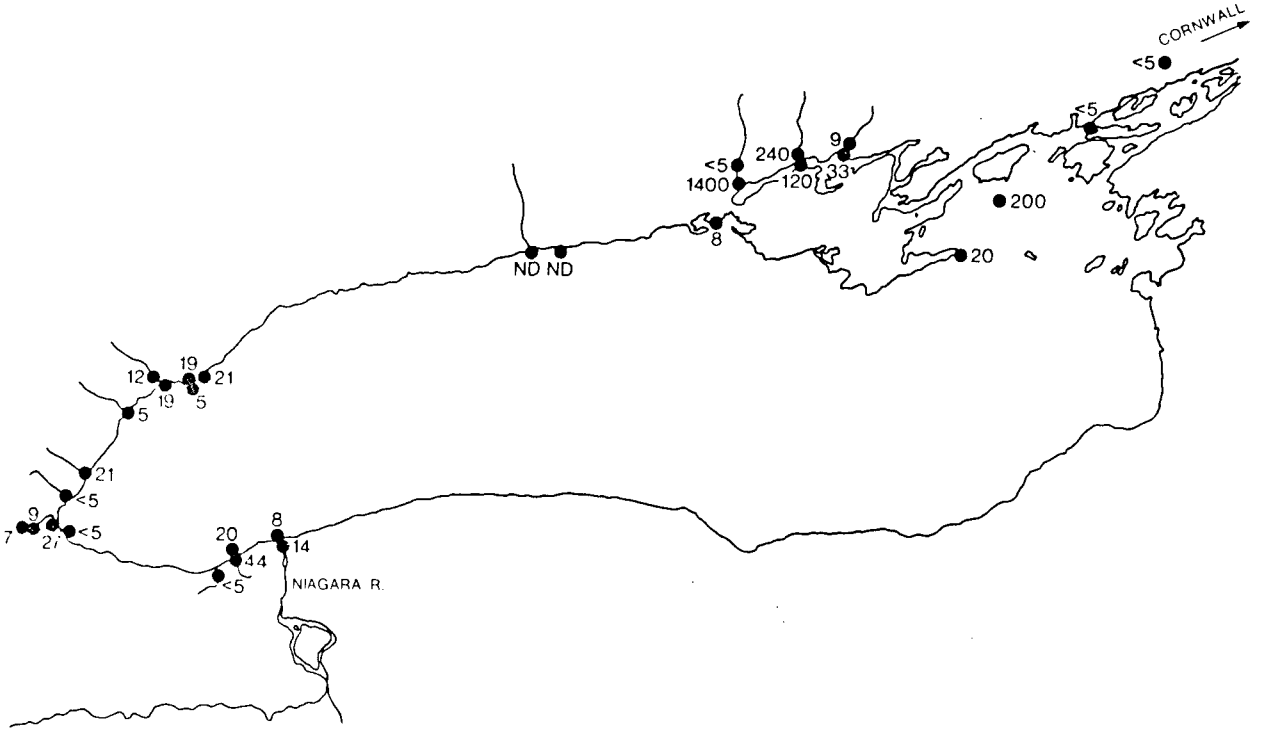


FIGURE 8 PENTACHLOROPHENOL IN THE LAKE ONTARIO BASIN
(Fox, 1978a)
(pentachlorophenol levels expressed as ng/L of bulk whole water;
ND = not detectable)

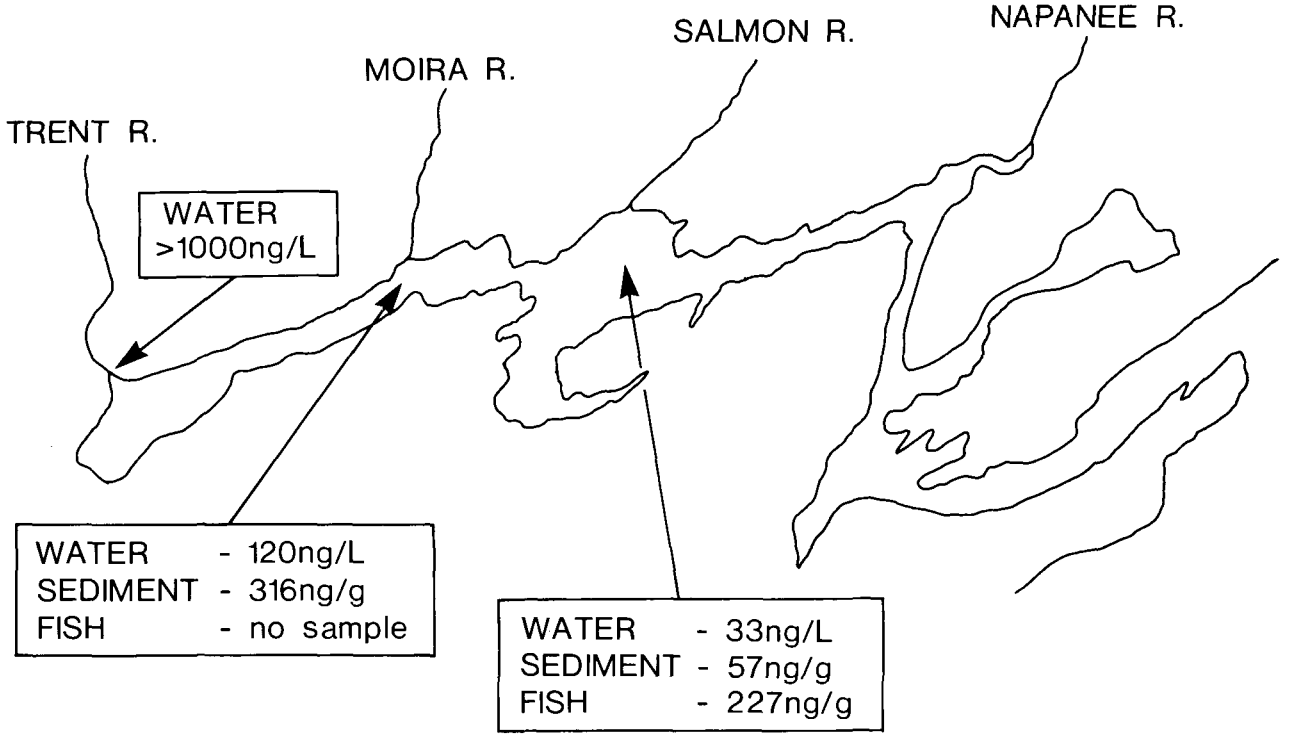


FIGURE 9 PENTACHLOROPHENOL IN BULK WATER, SURFACE SEDIMENT (dry wt.) AND FISH (brown bullhead, wet wt.) IN THE BAY OF QUINTE (Fox, 1978a)

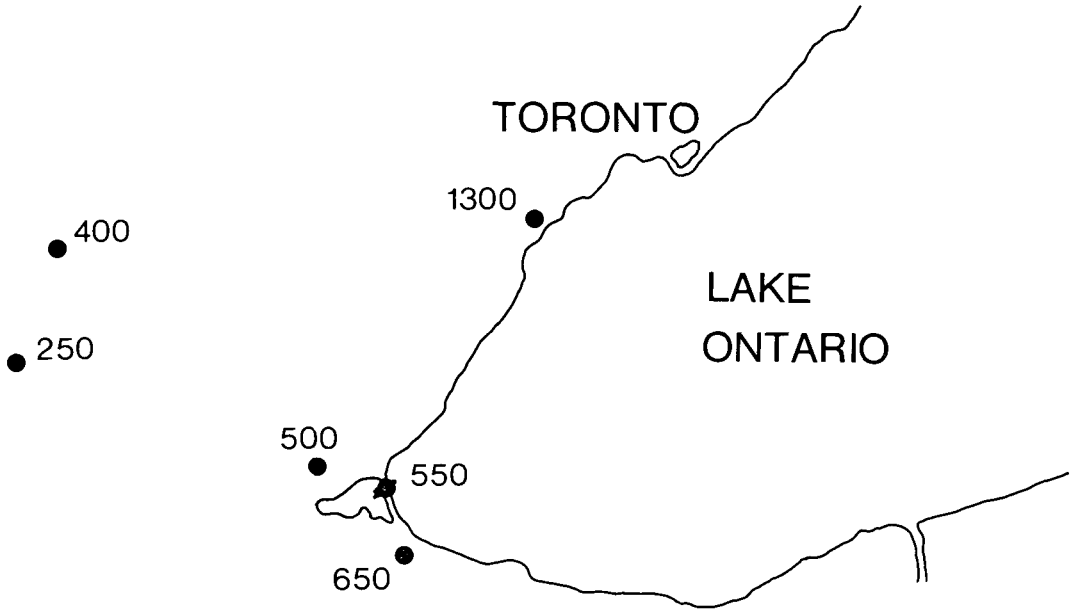


FIGURE 10 PENTACHLOROPHENOL IN FINAL SEWAGE EFFLUENT (ng/L) (Fox, 1978a)

the Sooke Basin, southern Vancouver Island; in 1972, another fish kill occurred in Victoria Harbour, which was also attributed to PCP. One additional instance was a fish kill attributed to PCP and TTCP in Mamquam Channel, Howe Sound. Except for the Little Campbell River incident there is no indication that the water in which the kills occurred was analyzed for the toxic materials.

The following report on the disposal of PCP contaminated ship ballast from a ship in for refit in Vancouver, B.C., was recounted during a round-table discussion at a symposium on PCP (Conklin and Fox, 1978):

> "An unusual incident involving PCP with no known resultant hazards was the case of a privately owned United States oil well drilling ship. The ship was brought into Vancouver to be refitted for drilling in the Arctic. The ship had a permanent type ballast consisting of barite ($BaSO_4$) with a water solution of Na-PCP and paraformaldehyde. The concentration of PCP in the ballast was 55 ppm. The ship's hull had to be restructured to withstand the severe operating conditions. Along with the structural changes, 2,000 tons of the ballast had to be removed and antifreeze solution added to the remaining ballast. After all possible alternatives for dumping the material were analyzed, the decision was made to dispose of the ballast at sea. The solution was diluted and dumped over 2,000 square miles of ocean 250 miles off the coast."

During the winter of 1978 - 79, groundwater entering the Okanagan River leading to Skaha Lake, south of Penticton, B.C., was contaminated with CPs as a result of leakage of TTCP/PCP from a defective, newly-constructed and untested, concrete dip tank at a sawmill. An estimated 18,200 L of 1% solution (10,000 ppm) of TTCP/PCP were lost from the tank. Drilled wells were used to both monitor CP levels in the groundwater and for interception and pumping-out of the groundwater between the spill site and the Okanagan River. Concentrations of CPs ranged from 0.001 to 61.0 ppm in the groundwater. Nearly 4.5×10^6 L of pumped contaminated water were trucked to the Penticton sewage treatment plant for treatment, including filtering through charcoal filters. Levels of CPs in samples of surface water from the Okanagan River and Skaha Lake ranged from 0.005 to 1.1 ppm, with one anomalous value of 18.09 ppm of CPs recorded from river water (Environment Canada, 1978; Cluett, 1979).

In the fall of 1978 a monitoring program was initiated in selected B.C. coastal receiving waters (Fig. 11, 12) to determine the environmental contamination resulting

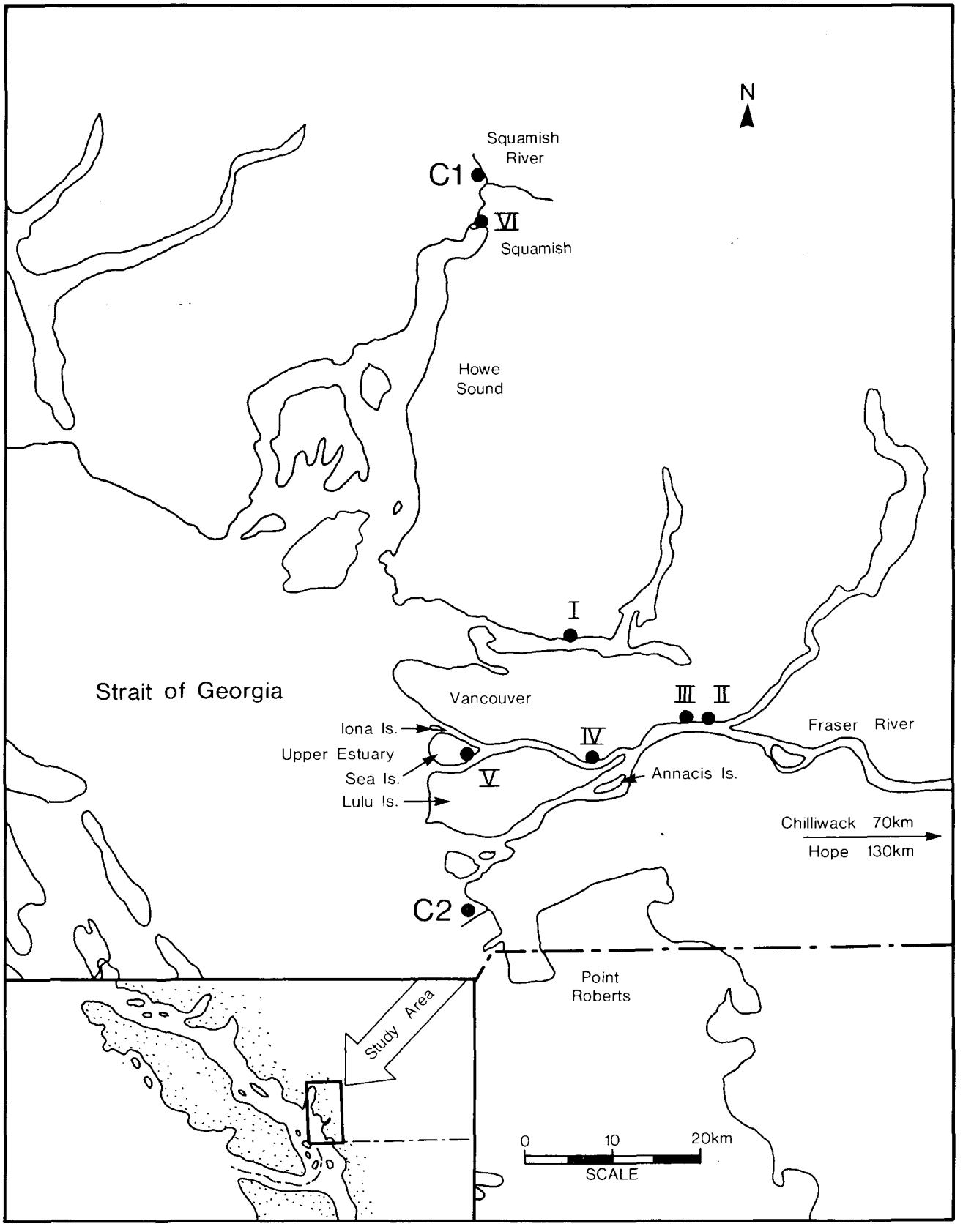


FIGURE 11 LOCATIONAL MAP OF THE LOWER MAINLAND, BRITISH COLUMBIA, SHOWING SAMPLE SITES. (Environment Canada, 1979)

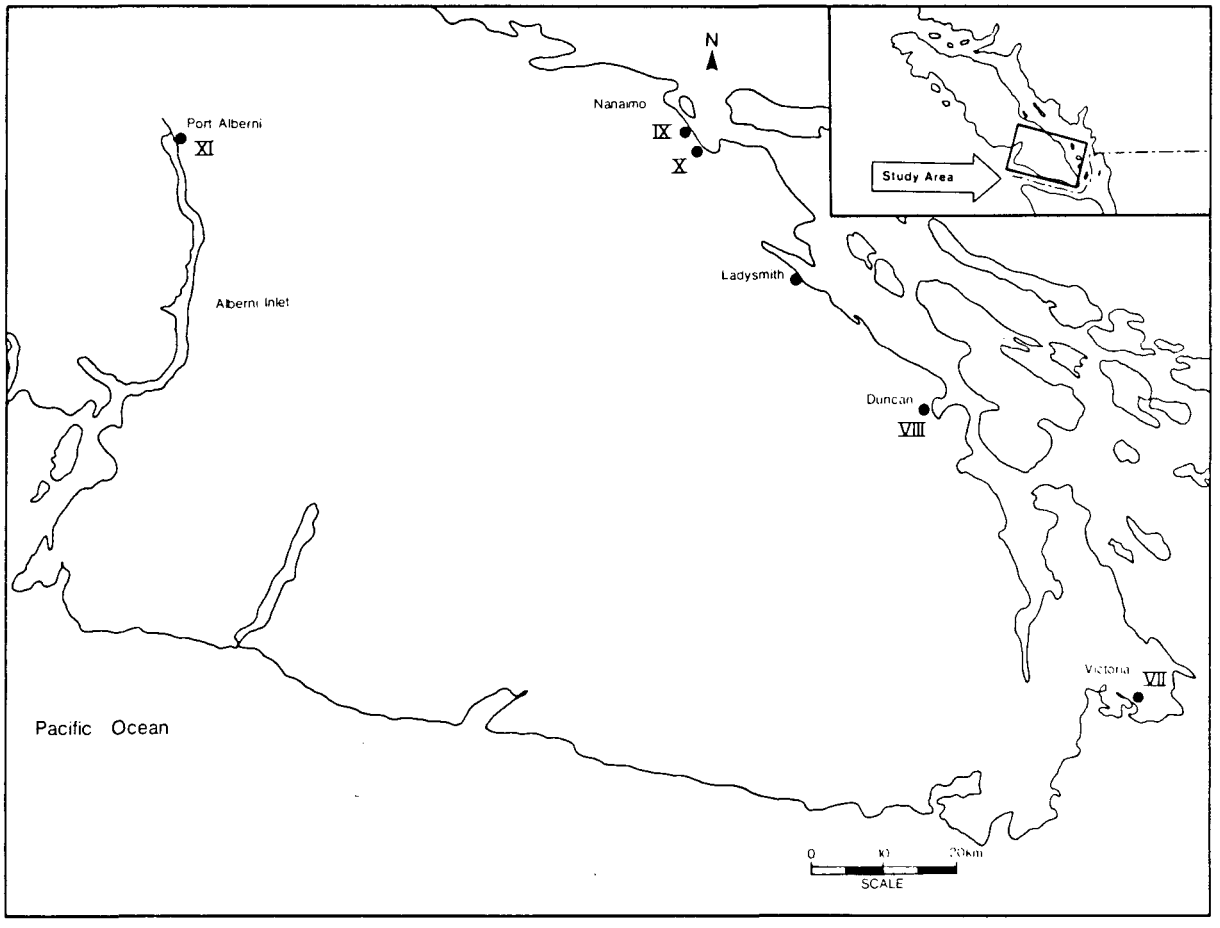


FIGURE 12 LOCATIONAL MAP OF SOUTHERN VANCOUVER ISLAND, BRITISH COLUMBIA, SHOWING SAMPLE SITES. (Environment Canada, 1979)

from the use of CPs in the wood preservation and wood protection industries (Environment Canada, 1979). Analyses for CPs, from DCP through PCP, were completed for samples of surface water, effluent, sediments (Sect. 5.1.2), and biota (fish, molluscs, and crustaceans) (Sect. 5.1.4.1, 5.1.4.2). No DCPs or TCPs were detected in the surface water or effluent samples. Both PCP and TTCP were present in the aquatic environment at all sites monitored, although they were not specifically detected in the surface water samples for Site VII, at Victoria, Vancouver Is. (Table 21). In the surface water samples at the other sites, concentrations (µg/L) of PCP in fresh and salt water ranged from trace amounts to 0.28 µg/L and 7.3 µg/L, respectively; similarly, for TTCP amounts present ranged from trace to 1.0 µg/L and 5.2 µg/L, respectively.

Effluents were sampled only at Vancouver Island sites. At two of the four sites effluents had comparatively high concentrations (µg/L) of PCP (225 and 2760 µg/L) and even greater concentrations of TTCP (530 and 8270 µg/L) (Table 21).

A cooperative study to identify and report on sources and levels of toxic contaminants in the lower Fraser River and Estuary was undertaken by Environment Canada and the British Columbia Ministry of the Environment. In a report in the Water Quality Series, prepared by C.L. Garrett (1980), data from both agencies has been utilized to document levels of toxic organic contaminants, including CPs, not only in environmental samples (Sect. 5.1.2, Sect. 5.1.4.2, Table 27) but also in industrial effluents, in waste disposal systems, and in landfill leachates (Table 22). The anomalous high level of PCP (6000 ppb) quantified in the drainage ditch sample from Coast Laminated Timber required verification, because CPs are not known to be used by Coast Laminated Timber. It was later determined that the high level of PCP was the result of an overflow from a wastewater tank used for holding glue washdown water prior to disposal by a septic tank company. Although the holding and disposal problem was corrected, the glue washdown water was confirmed to contain high levels of PCP (92 ppm) (Garrett, C.L. 1980. Personal communication).. Levels of CPs in industrial discharges were generally below 7.0 ppb except for CPs in drainage ditch water samples from a site on Sea Island, Richmond, which at the time was occupied by a pesticide packaging and formulating plant, Later Chemicals (near Site V, Fig. 11). Samples of drainage ditch water taken during the winter of 1977-78 had elevated levels of various isomers of the CPs: 135 to 2400 ppb of 2,4,5-TCP; 81 to 3120 ppb of 2,4,6-TCP; 96 to 166 ppb of 2,3,4,6-TTCP, and 1125 to 2520 ppb of PCP (Table 22). Recently, following abandonment of the plant, monitoring of soils, sediments, and water at the site showed that PCP, if present, was below detection

TABLE 21 CHLOROPHENOL CONCENTRATIONS (ppb) IN SEDIMENT, SURFACE WATER, AND EFFLUENT ASSOCIATED WITH THE WOOD PRESERVATION INDUSTRY AT FRESHWATER AND MARINE SITES IN BRITISH COLUMBIA (Adapted from Environment Canada, 1979)

Site		Pentachlorophenol				Tetrachlorophenol				Trichlorophenol	
		Sediment (µg/kg dry wt.)		Surface Water µg/L	Effluent µg/L	Sediment (µg/kg dry wt.)		Surface Water µg/L	Effluent µg/L	Sediment (µg/kg dry wt.)	
		Average	Range			Average	Range			Average	Range
FRESHWATER											
II	Fraser R., Coquitlam	35.0	10-70	0.28		28.0	10-60	0.10		ND	ND
III	Fraser R., Coquitlam	10.8	ND-30	0.25		27.4	6-80	1.0		ND	ND-10
IV	Fraser R., Burnaby	18.1	ND-90	TR		21.9	TR-90	0.30		ND	ND
V	Fraser R., Sea Island	TR	TR	TR		10.0	TR-15	0.20		ND	ND
MARINE											
I	Burrard Inlet, N. Vancouver	34.7	ND-240	0.75		39.8	ND-280	1.3		ND	ND
VI	Squamish R., Squamish	52.8	14-84	2.4		98.7	46-220	5.2		52.1	5-150
VII	Victoria	106.6	TR-500	ND		272.1	9-1600	ND		91.0	TR-170
VIII	Cowichan Bay	16.0	ND-75	TR	0.56	19.5	11-44	0.09	1.2	ND	ND
IX	Naniamo	42.0	ND-170	TR	225	65.4	15-290	0.06	530	ND	ND
X	Naniamo	13.1	ND-67	3.1	ND	22.8	9-71	3.3	2.7	ND	ND
XI	Pt. Alberni	187.9	ND-590	7.3	2760	157.3	54-370	0.22	8270	37.3	7-96

ND = Non Detectable (approx. 1/4 of limit of quantitation)
TR = Trace (present, but below level of quantitation)
Limit of quantitation - chlorophenols-sediment 5 ppb, water 0.05 ppb.

TABLE 22 LEVELS OF CHLOROPHENOLS (ppb) IN INDUSTRIAL AND MUNICIPAL DISCHARGES IN THE GREATER VANCOUVER AREA (Garrett, 1980)

Site	4-CP	2,4-DCP	Compound 2,6-DCP	2,3,4-TCP	2,4,5-TCP	2,4,6-TCP	3,4,5-TCP	2,3,4,6-TTCP	2,3,4,5-TTCP	2,3,5,6-TTCP	PCP
1) Scott Paper						5.4		0.2			0.2
2) MacMillan Bloedel											
a) before composite sampler								0.2			0.2
b) cooling water discharge to drainage ditch						TR		6.0			1.2
c) after retention tank											
3) B.C. Forest Products								0.7			0.2
4) Crown Zellerbach											
a) Coast Wood Prod. Div.								3.0			1.3
b) Richmond Lumber Mill					TR	TR		0.8			1.6
5) Domtar N.W. Preservers											
a) Ditch at railroad											
b) Refuse site					4.2			38.6		35.0	12.0
6) Coast Laminated Timber		TR	2.4	0.2	2.3	1.0	0.7	2100.0	TR	TR	6000.0
7) Belkin Packaging					TR	TR		7.2			5.4
8) Ioco Oil Refinery											
a) Main discharge pipe						0.3		0.4			4.9
b) after separator No. 4											3.6
9) Dow Chemical						TR		0.2			1.4
10) Reichold Chemicals								0.3			0.3
11) Later Chemicals											
a) S.W.Ditch	150.0	330.0	220.0		2400.0	3120.0		96.0			2520.0
b) N.W.Ditch		51.0		3.6	135.0	81.0		166.0	12.0		1125.0
12) Richmond Landfill Leachate											
a) Drainage ditch					TR	TR		0.3			1.2
b) Ditch just above discharge to Fraser River			2.0		0.8				0.09	1.2	1.4
13) Burns Bog Landfill											
a) N.W.Ditch					TR	TR		0.2			0.6
b) N.W. & S.E. Ditches combined				TR	TR	TR					1.6

TABLE 22 LEVELS OF CHLOROPHENOLS (ppb) IN INDUSTRIAL AND MUNICIPAL DISCHARGES IN THE GREATER VANCOUVER AREA (Garrett, 1980) (Cont'd)

Site	Compound 4-CP	2,4-DCP	2,6-DCP	2,3,4-TCP	2,4,5-TCP	2,4,6-TCP	3,4,5-TCP	2,3,4,6-TTCP	2,3,4,5-TTCP	2,3,5,6-TTCP	PCP
14) N. of Richmond Landfill Standing water (illegal hog fuel dump - 8931 River Rd)								0.4			6.0
15) Vito Steel Boat and Barge Co. Ltd. Bog water								0.5			
16) Lougheed Mills, Surrey Ditch - (133 St. and 116 A)					0.7		0.3	1.2			2.4
17) Vendev Landfill Ditch on Lougheed Hgwy.								0.8			0.9
18) Braid St. Landfill, Coquitlam a) Swamp at toe of landfill					1.7	1.0	0.5	3.2	0.6		7.2
b) Combined leachate at pumphouse			1.2		0.4			1.8	0.3	1.5	15.0
c) Ditch on westside			5.6	0.3	1.5	0.4	0.8	7.4	1.4		42.5

Note: TR = Trace
CP = monochlorophenol, DCP = dichlorophenol, TCP = trichlorophenol, TTCP = tetrachlorophenol, PCP = pentachlorophenol
Blank space = below limit of detection

limits (Garrett, 1980). The levels (dry weight basis) of TTCPs and TCPs detected in the samples of soils/sediments from the yard area were 2000 ppb of TTCPs and 180 ppb of TCPs. In soils/sediments from the ditches, there were 360 ppb of TTCPs and 90 ppb of TCPs. Levels of these coumpounds, identified in samples of standing water from the yard area, were 120 ppb of TTCPs and 190 ppb of TCPs. Drainage water samples from the ditches contained 300 ppb of TTCPs and 150 ppb of TCPs (Garrett, 1980). For comparison, leachates from landfills had levels of CPs from <1.0 to 42.0 ppb (Table 22).

Levels of PCP and 2,3,4,6-TTCP were quantified in municipal sewage treatment plant influents and effluents at the Annacis Island, Iona Island, and Lulu Island facilities and at the Ladner sewage lagoon and have been tabulated by Garrett (1980) (Table 23). Generally, levels were below 10 ppb except for 2,3,4,6-TTCP identified at levels of 13.2 and 28.3 ppb in two samples of Annacis Island effluent. With these exceptions, reductions in levels of PCP and 2,3,4,6-TTCP were noted between influents and effluents at the plants. Various TCPs were identified at low (TR-1.2 ppb) levels in both influents and effluents although data were limited (Table 23).

United States

The long-term effect of a CP on water quality was observed in 1945 near Montibello, California, when the groundwater supply of that city became contaminated with 2,4-DCP. Swenson (1962) reviewed the incident and noted that off-flavor and bad odor from the 2,4-DCP persisted for 4 to 5 years. He had observed that the original dilution of 2,4-DCP to sewage water was probably 1 to 10 million, which was 10 times above the threshold level.

The presence of PCP in the low parts-per-trillion (ppt) range in non-potable waters in Oahu, Hawaii, were noted in a study by Bevenue et al (1972a). A water sample from a sewage outfall from Honolulu contained 2600 ppt PCP. The authors suggested that the presence of PCP resulted from use of this pesticide for control of termites. A wood treatment plant was the source of 143 ppt of PCP in water samples in a drainage ditch, and in a stream near the lumber yard there were 168 ppt of PCP.

Bevenue et al (1972b), who reviewed the literature on organochlorine pesticide residues in rain water, observed in 1971 - 72, levels of 2 to 270 ppt of PCP in rain water samples from Oahu. Of further interest were the detections of 14 ppt of PCP in snow from Mauna Kea Summit (3505 m) and 10 ppt PCP in Lake Waiau (fed almost exclusively by the Summit snows) on the island of Hawaii.

TABLE 23 CHLOROPHENOLS IN GREATER VANCOUVER MUNICIPAL SEWAGE TREATMENT PLANTS INFLUENTS AND EFFLUENTS (ppb) (Garrett, 1980)

Chlorinated phenol	Sewage Treatment Plant												
	Annacis Island				Iona Island				Lulu Island				Ladner
	Influent		Effluent		Influent		Effluent		Influent		Effluent		Sewage lagoon
	No. 1	No. 2	No. 1*	No. 2	No. 1	No. 2	No. 1*	No. 2	No. 1	No. 2	No. 1*	No. 2	Effluent
2,3,4-TCP											TR		
2,4,5-TCP									TR		TR	TR	
2,4,6-TCP	0.7		0.7	1.2			TR		0.09	0.9	TR	0.1	
3,4,5-TCP									TR		TR		
2,3,4,6-TTCP	8.7	10.8	28.3	13.2	1.4	1.1	1.0	0.7	0.6	10.0	0.6	1.7	
PCP	7.8	12.0	4.7	1.2	1.3	2.0	1.4	1.2	4.5	2.8	1.1	3.0	0.5

TR = Trace, TCP = trichlorophenol, TTCP = tetrachlorophenol, PCP = pentachlorophenol
Blank spaces - below limits of detection
*Results for effluent sample No. 1 from each plant based on 2 replicates

Concentrations of significant amounts of PCP were identified by Buhler et al (1973) by gc and confirmed by ms in samples of sewage influent and effluent from three Oregon cities and in Willamette River water and treated discharge water. PCP levels in sewage influent were 1 - 5 ppb, while in sewage effluent they were 1 - 4 ppb. Conventional processing of river water reduced levels of PCP by 60%, leaving 40% of the PCP in the finished drinking water.

Fountaine et al (1976) reported that due to industrial discharge over many years, Naylor's Run Creek in Haverford Township, a suburban area 10 miles from Philadelphia, PA, had accumulated dangerous levels of PCP. Water samples taken in late August and September 1974 showed levels of PCP from 0.05 to 10.5 ppm. The authors suggested steps that could be taken to reduce the level of PCP which came from both factory discharge and from ground seepage; the latter probably originated from gross spillage of PCP at the factory area in the early years of operation.

Pierce et al (1977) in 1975 - 76, followed levels of PCP in a man-made lake following an overflow from a pole-treatment plant waste holding pond in December 1974. They noted that lethal concentrations of PCP reached the lake immediately after the spill, but within two months levels were down to 6 to 19 ppb, in the lake water. Their summation of the results were as follows:

> "These results indicate a short residence time for PCP in the water column as supported by a rapid repopulation of fish in the lake by the time of the first sampling (February, 1975). The low level PCP concentrations found in the water throughout the study were probably due to the continuous influx from contaminated water shed areas. This is supported by anomolously high PCP concentrations observed in February, 1976, which followed a two month period of high rainfall. Surface water samples were analyzed periodically, but did not show a marked increase in PCP content over bulk water as had been expected, even in the presence of a visible oil sheen."

Pierce and Victor (1978) reported that routine sampling of the lake continued both prior to and following a second overflow in December 1976, with the following results:

> "PCP appeared to be uniformly distributed throughout the lake exhibiting background levels (0.3 ppb) in October, 1976, increasing in January, 1977 immediately after the spill, and decreasing to 5 to 10 ppb by April, 1977".

In their report Pierce and Victor (1978) noted the concentration in the lake water of the PCP degradation products, PCP-OCH_3, 2,3,4,5-TTCP, and 2,3,5,6-TTCP (App. 6, Sect. 6.1.1.1). In summary, the authors noted that although the 2,3,4,5-TTCP isomer was present in concentrations equal to or greater than the 2,3,5,6-TTCP isomer in some samples, it exhibited a low response to gc-ec and was difficult to quantify in many instances. They also observed that varying quantities of the methyl ether (anisole) of both TTCP isomers were also observed but proved difficult to quantify, due to low concentrations and interference from naturally-occurring substances. The 2,3,5,6-TTCP concentration in the lake water from August 1976 to April 1977 varied between 0.03 to 2.0 ppb. Although the concentration of pentachloroanisole (PCP-OCH_3) in the lake water, during the same period, ranged from 0.002 to 1.94 ppb, with the high value recorded from samples following a spill of effluent from the plant holding pond in December 1976, the concentration had remained at relatively constant low levels, which the authors suggest was due to the low solubility of PCP-OCH_3 in water.

Europe

Ernst and Weber (1978a) reported levels of PCP detected in water samples obtained in 1976-77 from the Weser River downstream from Bremen, and in the Weser and Elbe Estuaries and the German Bight. High levels, up to 500 ng/L, in the upper part of the estuary dropped to near the detection limit in the German Bight. The input of river-borne PCP in the estuary was demonstrated by the highest levels of PCP occurring at low tide. From the data available the Elbe River may have been contributing as much PCP as the Weser River. Using an average PCP level of 100 ng/L and a water flow of 300 m^3/s, a rough estimate of the input of PCP via the Weser River to the German Bight during the investigation period would be 1000 kg/yr.

In addition to PCP in waters in the study area associated with the Weser River, six lower chlorinated phenols were identified and quantified. Weber and Ernst (1978a) reported that on an average the sum of lower chlorinated phenols in estuarine waters amounted to 20% of PCP; 2,3,5,6- and/or 2,3,4,6-TTCP and 2,4,6-TCP predominated with lesser amounts of 2,4,5-TCP; 2,6-, 2,5-, and/or 2,4-DCP. A review by Buikema et al (1979) surveyed the pertinent world literature on phenolics in the aquatic ecosystem. Included in their summary tables were levels of mono-, di-, and tri-chlorophenolic compounds reported in 1975 in water samples from three rivers in the Netherlands - the Meuse, Rhine, and Schiede - and from the North Sea coast at

Scheveningen, just north of the Mass-Waal-Rhine Estuaries in the Netherlands. Levels for the CPs were: 1 - 20 µg mono-CPs/L, 0.01 - 1.5 µg DCPs/L, and 0.003 - 0.1 µg TCPs/L.

5.1.2 Sediments. In a review of the world literature on PCP residues in bottom waters and in sediments, Strufe (1968) referred to several research papers where it was reported that PCP and NaPCP were strongly adsorbed to mud in river water (mud content 1500 - 1600 mg%) within hours, eg. 80% loss of active ingredient to mud after 216 h (9 days) at 18°C in one study and 65% loss from 7 ppm of NaPCP by adsorption to mud in 20 h at 22-23°C in another study. In these and similar studies he quoted, circumstantial evidence indicated adsorption of PCP by mud and sediments, since the measurements made were loss of active ingredient from the water and not the amount of active ingredient gained by the sediment. However, in one study cited, where the NaPCP content of the river bed was measured at three sites, up to 900 m from the injection sites, following a molluscicide operation, the NaPCP present in the sediment was at a maximum, 25 ppm, 24 h after the application, but decreased to near zero within 9 days.

Canada

In an abstract of a report on PCP in the Great Lakes Basin (Sect. 5.1.1) Fox (1978a) noted the PCP levels in sediment, and he also included a summary statment based on levels of PCP in both water and sediment, as follows:

> "In the Bay of Quinte on the north shore of Lake Ontario, with the highest observed input of pentachlorophenol, sediments contained >300 ng/g (dry weight) and fish contained >200 ng/g (whole fish, wet weight).
>
> "The study indicates widespread contamination of the Great Lakes Basin with pentachlorophenol, the levels of which can be related to the extent of urban and industrial development of individual watersheds. A significant quantity of pentachlorophenol is discharged into the watersheds from sewage treatment plants."

Strachan (1979b) reported on concentrations of PCP detected in sediments in Lake Superior. The sediment samples collected in June 1978 were complementary to water samples from the same sites (Sect. 5.1.1). The average concentrations (range in parentheses) of PCP in sediment samples from Thunder Bay, Marathon, and Michipicoten were 16.9 (0.83 - 100.9), 7.3 (0.61 - 31.68), and 2.3 (0.99 - 6.78) µg PCP per gram of sediment (dry wt.), respectively.

In a study of chlorinated compounds in the St. John River estuary (Sect. 5.1.1), Bacon (1978) noted that the sediment samples analyzed for chlorinated compounds showed only trace amounts of PCP and trichloroguaiacol which could not be confirmed by mass fragmentography.

In the monitoring program for CPs in the aquatic environment associated with B.C. wood preservation plants (Sect. 5.1.1), PCP and TTCP were identified and quantified in sediment samples from all sites, while TCP was present in sediment samples from 4 of the 11 sites (Environment Canada, 1979). CP concentrations (µg/kg dry wt.) in sediments varied from non-detectable or trace to maximums of 590 ppb PCP, 1600 ppb TTCP, and 170 ppb TCP. TTCP was detected at similar or greater concentrations than PCP at all sites (Table 21).

Samples of sediments from drainage ditches at a former pesticide plant at Sea Island (Sect. 5.1.1) contained TTCP (0.36 ppm) and TCP (0.09 ppm). PCP, if present in the sediment samples, was below the detection limits (Garrett, 1980).

United States

Following an overflow of wood treatment wastes from a pole-treating company's wastewater holding pond in December 1974, Pierce et al (1977) studied PCP levels in water (Sect. 5.1.1) and sediment in the affected freshwater ecosystem. Their studies were summarized as follows:

> "Sediments contained much larger amounts of PCP (up to 1,200 ppb) than were found in the water column. Samples of stream sediments near the spill source contained over ten times that found in other sediments in February, April and June, 1975. By December, 1975, the PCP content of stream sediments showed a drastic reduction probably due to resuspension and redistribution of PCP-containing sediments. The concentrations in lake sediments remained relatively constant throughout the study."

In a subsequent report after continued monitoring of PCP levels in the lake water and sediment, and following a second spill in December 1976, Pierce and Victor (1978) stated that:

> "Suspended particulates generally contained less than ten percent of the PCP in the water column. Although particulate PCP was not so abundant as dissolved PCP, it may be important for the transport of PCP to the sediment. Sediment contained a higher concentration (500 ppb average) of PCP in August

and October, 1976, and, rather than exhibiting an increase immediately after the spill, the sediment samples collected in January, 1977 showed a slight decrease in PCP content (200 ppb average) indicating a residence time of PCP in the water column of over a week before incorporation into sediment. Lake sediment showed an increase near the mouth of the stream in February, 1977 (1,500 ppb), but in April the concentration in sediment at all sites was back to the January level (200 ppb average). This reduction could be due to degradation or to the influx of uncontaminated sediment from soil erosion."

5.1.3 Plants. No residue data on CPs in aquatic plants in Canada could be located. A report by Pierce et al (1977) referred to concentrations of PCP in leaf litter and vegetation along a stream which included drainage from an industrial site (Sect. 5.1.1 and 5.1.2). Although not precisely an aquatic situation, because of the close relationship of the PCP in the leaf litter and the stream, the study is referred to here rather than under the terrestrial ecosystem section of this report. High concentrations of PCP, 1680 to 6400 ppb, were detected in leaf litter from the stream bank throughout the study, from February 1975 to May 1976. They reported that PCP contaminated leaf litter released 10% of the PCP to water after a 24 h equilibrium period. Their conclusions were as follows:

"These results indicate that PCP associated with leaf litter and vegetation along the bank of a stream may be released over a long period of time, serving as a source for chronic PCP contamination of the aquatic environment."

5.1.4 Animals

5.1.4.1 Invertebrates

Canada

Although there is considerable information on toxicity of CPs to invertebrates (App. 4, Sect. 4.1.2), little information is available on residues of CPs in invertebrates from a Canadian aquatic environment. There is a current study on the fate of PCP in biota, including invertebrates, in the Bay of Quinte, Lake Ontario (Fox, 1978).

Bacon (1978) reported on levels of 2,4-DCP; 2,4,6-TCP, and PCP, detected and quantified by glc and gc-ms, in samples of clam, Mya arenaria, and sandshrimp, Crangon septemspinosa, taken in pulpmill effluent receiving waters near St. John, N.B., in 1977 (Figure 4) (Table 24) (Sect. 5.1.1). Although the percent recoveries were somewhat low (40-45%), TCP and PCP were detected in the lipids of the majority of samples.

TABLE 24 CHLOROPHENOLS IN MARINE ORGANISMS IN PULPMILL EFFLUENT RECEIVING WATERS NEAR ST. JOHN, N.B. (Adapted from Bacon, 1978).

Organism	Type of sample	Location of sampling station	Sampling date (1977)	Lipid wt in 5g sample (mg)	CP conc. (µg/g lipid as methyl ethers) 2,4-DCP	2,4,6-TCP	PCP
Invertebrates							
Clam (Mya arenaria)		Off Boars Head	June 15	19.7	N/D*	N/D	0.43
		Harbour Bridge	July 14	64.3	Detect.**	0.56	0.83
		Harbour Bridge	Aug. 23	74.2	N/D	0.123	2.3
Sandshrimp (Crangon septemspinosa)	body	Off Boars Head	June 15	62.1	Detect.	0.74	2.4
Vertebrates							
Winter flounder (Pseudopleuronectes americanus)	muscle	Off Boars Head	June 15	17.6	3.7	0.12	6.2
	muscle	Off Milkish Channel	June 23	31	Detect.	1.85	1.63
	muscle	Off Milkish Channel	July 5	21.6	2.5	0.29	7.9
	viscera	Off Milkish Channel	June 23	109.1	Detect.	1.41	0.49
	skin/fat	Off Milkish Channel	June 23	106.4	Detect.	1.41	0.49
	liver	Off Milkish Channel	June 23	92.9	0.7	3.48	1.3
Gaspereau (alosa pseudoharengus)	muscle	Off Boars Head	June 15	84.3	Detect.	N/D	0.82
	liver	Off Boars Head	Aug. 24	1321	0.29	0.02	0.22
Shad (alosa sapidissima)	liver	Off Boars Head	Aug. 24	691	0.52	0.017	0.81
	liver	Off Goat Island	Sept. 7	452	Detect.	0.027	0.58
Smelt (Osmerus mordax)	muscle	Off Boars Head	June 15	21.5	9	0.43	4.04
	muscle	Off Milkish Channel	June 23	24.7	1.1	0.25	5.6
	viscera	Off Milkish Channel	June 23	158.5	Detect.	2.3	0.37
	skin/fat	Off Milkish Channel	June 23	63.5	Detect.	0.67	0.35
	liver	Manawagonish Island	Aug. 31	123.2	Detect.	0.062	1.44
Spiny dogfish (Squalus acanthias)							
Sturgeon (Acipenser oxyrhynchus)	liver	Off Boars Head	June 15	509.3	0.37	0.028	0.26
Tomcod (Microgadus tomcod)	muscle	Off Boars Head	June 15	52.3	3.73	1.82	3.4
	muscle	Off Milkish Channel	June 23	53.4	Detect.	2.29	0.43
	muscle	Off Milkish Channel	July 5	7.7	2	0.33	5.36
	viscera	Off Milkish Channel	June 23	176.8	Detect.	3.8	0.75
	skin/fat	Off Milkish Channel	June 23	77.3	Detect.	2.1	1.0
	liver	Off Milkish Channel	June 23	1641	0.74	0.39	0.17

*N/D = not detected

**Detected; not quantified due to interference

Note: CP - Chlorinated phenol, DCP - Dichlorophenol, TCP - Trichlorophenol, PCP - Pentachlorophenol
Total recoveries from Sephadex and silica gel: DCP 45%, TCP 40%.

In the fall of 1978 invertebrates from receiving waters for effluents from wood preservation and wood protection plants in British Columbia were monitored for presence of CPs (Sect. 5.1.1 and 5.1.2) (Environment Canada, 1979). Although desired species of invertebrates were not always available at all sites, invertebrate species monitored included crayfish (Pacifastacus sp.) from freshwater sites, and crabs (Cancer spp.) and clams (Macoma spp.) from marine sites. The average concentration (µg/kg wet wt.) of PCP and TTCP in the tissues of the crabs and molluscs ranged from non-detectable to a maximum concentration of 17 ppb PCP and 20 ppb TTCP (Table 25). TCP was not detected in the invertebrate samples.

Europe

During a study on CPs in the Weser Estuary and German Bight (Ernst and Weber, 1978a; Weber and Ernst, 1978a) (Sect. 5.1.1), Ernst and Weber (1978b) also investigated occurrence of CPs in bottom living animals of the Weser Estuary. They reported CP levels in the polychaete, Lanice conchilega, and an actinian, Sagartia troglodytes. Analysis by gc-ec and gc-ms indicated average levels of CPs in L. conchilega on a wet-weight basis as follows: 117.5 ng PCP/g, 67 ng 2,3,4,6- and/or 2,3,5,6-TTCP/g, 7 ng 2,3,4,5-TTCP/g, 26 ng 2,4,6-TCP/g, 19.3 ng 2,4,5-TCP/g, and 11.8 ng 2,4- and/or 2,5-DCP/g. In comparison to levels of PCP in L. conchilega, levels of PCP in S. troglodytes were much lower, with an overall average of 4.6 ng PCP/g, wet weight. It was noted that during sampling S. troglodytes were tightly attached to L. conchilega and thus exposed to the same environmental conditions. Ernst and Weber (1978b) noted that on the basis of an average PCP concentration of 0.04 ng/L in the water at the sampling location, bioconcentration factors for PCP in S. troglodytes were calculated to be approximately 70 - 180, whereas L. conchilega exhibited those from 2600 - 8500.

At the same time that the bioaccumulation of CPs in L. conchilega was being investigated by Ernst and Weber (1978b), they detected mono-, di-, and tribromophenols in this organism (Weber and Ernst, 1978b). The 2,4,6-tribromophenol content was in the range of 0.6 to 2.3 mg/kg wet weight, with comparable amounts of mono- and dibromo compounds. Water analysis in the environment of the polychaete population indicated presence of <1 - 2 ng/L of 2,4,6-tribromophenol. Weber and Ernst (1978b) suggested that the brominated compounds were not likely to be river-borne and bioaccumulated from water; rather, they were naturally occurring secondary metabolites that were either

TABLE 25 AVERAGE CONCENTRATIONS (ppb) OF PENTACHLOROPHENOL (PCP) AND TETRACHLOROPHENOL (TTCP) IN THE TISSUES OF FISH, CRABS, AND MOLLUSCS COLLECTED AT FRESHWATER AND MARINE SITES NEAR WOOD PRESERVATION PLANTS IN BRITISH COLUMBIA
(Adapted from Environment Canada, 1979)

			Principal Species-Concentrations of CPs in Tissues (ug/kg wet wt.)																	
			Cancer magister* (Dungeness crab)		Cancer productus (Red rock crab)		Cottus asper (Prickly sculpins - freshwater)				Leptocottus armattus (Staghorn sculpins - marine)				Macoma sp. (prob. balthica) (clams)		Pacifastacus sp. (prob. leniusculus) (crayfish)		
SITE		Contaminant	Chelaped Muscle	n	Chelaped muscle	n	liver	n	Skeletal muscle	n	liver	n	Skeletal muscle	n		n	pincer muscle		n
FRESHWATER																				
II	Fraser R., Coquitlam	PCP					140	7	40	7							ND		7
		TTCP					89	7	100	7							ND		7
III	Fraser R., Coquitlam	PCP					600	2	14	2							ND		10
		TTCP					320	2	80	2							TR		10
IV	Fraser R., Burnaby	PCP					300	4	74	4	100	6	TR	6			TR		2
		TTCP					96	4	10	4	74	6	TR	6			TR		2
V	Fraser R., Sea Island	PCP					TR	5	12	5	470	19	5	19					
		TTCP					82	5	5	5	480	19	8	19					
MARINE																				
I	Burrard Inlet, N. Van.	PCP			TR	10					24	7	TR	7					
		TTCP			6	10					69	7	6	7					
VI	Squamish R., Squamish	PCP	ND	10							35	4	TR	4					
		TTCP	8	10							63	4	9	4					
VII	Victoria	PCP	TR	6	7	11					26	3	13	3					
		TTCP	TR	6	TR	11					470	3	8	3					
VIII	Cowichan Bay	PCP	16	8							TR	10	TR	10					
		TTCP	20	8							29	10	10	10					
IX	Naniamo	PCP	TR	10															
		TTCP	7	10															
X	Naniamo	PCP	17	2							2100	7	TR	7	ND	340			
		TTCP	5	2							1600	7	8	7	12	340			
XI	Pt. Alberni	PCP	8	2							640	1	84	1					
		TTCP	TR	2							430	1	34	1					
Average Concentrations		PCP	7		4		260		35		425		13		ND		TR		
		TTCP	7		4		147		49		402		11		12		TR		

*At Site X the sample consisted of 1 C. magister and 1 Hemigrapsus nudus (shore crab)

n = number of samples analyzed

ND = Non-detectable (approx. 1/4 of limit of quantification) (Limits of quantification for CPs in tissue 5 ppb)

TR = Trace (present, but below level of quantification)

synthesized by L. conchilega itself or - less likely - ingested with food. Weber and Ernst (1978b) concluded with a cautionary note that:

> "Furthermore, natural occurrence of bromophenols in L. conchilega should be considered if bioaccumulation of anthropogenic chlorophenols is evaluated from toxicologic and environmental considerations."

South America

In 1971 in Surinam, S.A., a study, which had been supported by the Canadian Wildlife Service, investigated the effect of pesticides, including NaPCP, on fauna on an 8,000 ha rice growing area (Vermeer et al, 1974). Part of the pest control program had included spraying of NaPCP for reducing the population of water snails, Pomacea glauca and P. lineata. NaPCP had been applied at the rate of 3.5 - 4 kg of 85% active material per 20 L of water per hectare, just prior to the commencement of each of two rice growing seasons, in April and again in October.

Following sample collecting, processing and shipping, residue analyses were carried out at the Ontario Research Foundation. Vermeer et al (1974) reported that mean wet-weight residue levels of PCP in composite samples of Pomacea snails were 36.8 ppm. Snails were the major food component of several species of birds frequenting the rice fields (Sect. 5.2.4).

5.1.4.2 Vertebrates. Few data on residues of PCP in aquatic vertebrate fauna exist. The situation was somewhat comparable to that in the previous section on invertebrates; i.e. much toxicity data was available (Sect. 6.1.3 and 6.1.4) but residue data were scarce until recently, which may, in part, be a reflection of the analytical problems.

Canada

PCP levels in aquatic fauna from several locations on the east coast of Canada were recorded by Zitko et al (1974) (Table 26). To provide a base for evaluation of the residue levels, Zitko et al (1974) pointed out that Stark (1969) had reported that guppies (Lebistes reticulatus) killed by PCP within 18 h contained approximately 100 µg/g wet weight of PCP.

Bacon (1978) reported levels of CPs in tissues of vertebrate aquatic organisms from the St. John River estuary (Table 24), as part of an overall study on "Bioaccumulation of Toxic Compounds in Pulpmill Effluents by Organisms in Receiving Waters" (Sect. 5.1.1, 5.1.2, and 5.1.4.1). The CPs detected in the tissues and quantified at what Bacon

TABLE 26 CONCENTRATIONS OF PENTACHLOROPHENOL IN AQUATIC FAUNA AND COMMERCIAL FISH FOOD (Adapted from Zitko et al, 1974)

Sample Name	Location	Year	Weight (g)	Pentachlorophenol (ng/g wet weight)[1]
Cod (*Gadus morhua*)	St. Croix estuary, N.B., Maine	1972	486.8	0.82
Winter flounder (*Pseudopleuronectes americanus*)	St. Croix estuary, N.B., Maine	1972	157.8	1.77
	St. John estuary, N.B.	1972	128.2	3.99
Sea raven (*Hemipterus americanus*)	St. Croix estuary, N.B., Maine	1972	875.9	0.5
Silver hake (*Merluccius bilinearis*)	St. John estuary, N.B.	1972	214.0	1.75
Atlantic salmon (juvenile)	From hatcheries	1973	50.3	1.26
			8.5	0.54
White shark liver (*Carcharodon carcharias*)	Landed Leonardville, N.B.	1971	-	10.83
Double-crested cormorant egg (*Phalacrocorax auritus*)	White Horse (Bay of Fundy)	1973	46.2	0.36
Herring gull egg (*Larus argentatus*)	Hospital Island, Passamaquoddy Bay, N.S.	1973	96.1	0.51
Commercial fish food			-	2.23

[1]For fish samples, with the exception of white shark liver, subsamples taken from two specimens were combined for analysis. Whole salmon, and muscle tissue of the other fish were analyzed.

(1978) considered prominent levels were 2,4-DCP, 2,4,6-TCP, and PCP; although no CPs were detected in water samples and no PCP was detected in the effluent from the mills (Sect. 5.1.1). Bacon (1978) remarked on the inconsistencies of the CP levels in the analyzed tissues as follows: "The lower concentrations in the tomcod liver and the higher concentrations in the smelt muscle indicate the complexity of the metabolic aspects for which there is yet virtually no published data." It had been anticipated that the higher concentrations of CPs would be in the livers.

Fox (1978a), as previously mentioned (Sect. 5.1.1), had reported that fish from the Bay of Quinte, on the north shore of Lake Ontario, contained >200 ng/g of PCP (whole fish, wet wt.).

Strachan (1979c) in a partial and prepublication report on levels of PCP in three species of fish taken from Lake Superior in 1978 noted that the ranges in concentrations of PCP in the whole fish samples were as follows: lake trout (Salvelinus nanaycush), 0.1 - 1.0 ppm; siscowet or fatty lake trout (Salvelinus nanaycush siscowet), 0.1 - 1.0 ppm; and lake whitefish (Coregonus clupeaformis), 0.02 - 0.60 ppm. The fish were obtained from the same areas of Lake Superior from which water and sediment samples were obtained, ie. Thunder Bay, Michipicoten Bay, and the Marathon area. Various factors including small sample size preclude identifying the particular waters from which the fish were taken; therefore, the fish are only referred to as being from Lake Superior (Sect. 5.1.1 and Sect. 5.1.2).

PCP at 26 ppb was detected and confirmed by gc-ms in a 50 g tissue sample of flounder collected in November 1978 at Sturgeon Bank, B.C., near the Iona Sewage Treatment Plant outfall (Rogers, 1979). The sewage plant effluent is chlorinated in late spring and summer to protect swimmers, but in the cold months chlorination is discontinued. Whether this practice contributes to increased levels of CPs in the effluent is not known.

Levels of PCP associated with the fish kill in 1972 at Little Campbell River (Table A4-4) were reported by Alderdice (1978). Water samples from the affected stream had concentrations of PCP from 0.07 to 53.75 ppm (Sect. 5.1.1). The intestinal tract from a dead fish (species not stated) from the affected stream contained 12.93 ppm of PCP. Whole samples of small fish yielded 16.3 ppm of PCP and two large cutthroat trout, Salmo clarki, had levels of 10.29 ppm of PCP.

A monitoring project in November 1978 for CPs in receiving waters for effluent from B.C. wood treatment plants in both fresh water and marine situations

included the analysis of fish, specifically sculpins (Sect. 5.1.1, 5.1.2, and 5.1.4.1) (Environment Canada, 1979). The prickly sculpin, Cottus asper, and the staghorn sculpin, Leptocottus armatus, from freshwater and marine sites, respectively, are not used directly by man but are important secondary consumers in aquatic food chains. Independant of the collection sites, either freshwater or marine, sculpin skeletal muscle burdens of CPs (ug/kg wet wt.) ranged from a trace to 84 ppb PCP (ave. 25 ppb) and from a trace to 100 ppb TTCP (ave. 30 ppb). No TCP was detected in any of the fish tissue samples. The concentrations of PCP and TTCP in the sculpin muscle tissue were very comparable with the level of these CPs detected in muscle tissue from crabs, clams, and crayfish (Sect. 5.1.4.1) (Table 25). In contrast with the levels of CPs in the muscle tissues of sculpins, levels of CPs in sculpin livers were considerably elevated (ie. from a trace to 2100 ppb PCP (µg/kg wet wt.) (ave. 454 ppb) and from 5 to 1600 ppb TTCP (ave. 275 ppb)) (Table 25). Based on average concentrations, the apparent differentials in concentrations of PCP and TTCP between sculpin muscle tissue and liver were in the order of 18x and 9x, respectively. Sculpin livers were identified as highly suitable organs for monitoring for CPs (Environment Canada, 1979).

The Westwater Research Center conducted a fish sampling program in 1972-73 to obtain information on organic contaminant levels in resident fish species in the lower Fraser River from Hope to the Estuary. At that time contaminants of interest included PCBs and the major agricultural pesticides. In 1978, new funding allowed further analyses to be undertaken, including analyses for CPs. The fish samples, frozen at the time of collection in 1972-73, were representative of five areas of the lower Fraser: the North Arm, South Arm, Upper Estuary, Chilliwack area and Hope area (Fig. 11). Residue levels of TTCP and PCP did not exceed 62 ppb and 125 ppb, respectively (Table 27). As noted by Garrett (1980) the CP compounds were identified with greater frequency and higher concentrations in fish from the industrially developed lower reaches of the Fraser River.

Europe

Landner et al (1977) provided field data to demonstrate that the same compounds that had concentrated in rainbow trout during experimental exposure in the laboratory (App. 8, Sect. 8.1) were also identified in perch, Perca fluviatilis, and northern pike, Esox lucius, from the vicinity of a Swedish mill producing full bleach kraft pulp (Table 28).

TABLE 27 LEVELS OF TETRACHLOROPHENOL (TTCP) AND PENTACHLOROPHENOL (PCP) (ppb wet weight) IN FISH COLLECTED IN 1972-73 FROM THE FRASER RIVER AND THE UPPER ESTUARY
(Adapted from Garrett, 1980)

Location	No. of Samples	Species	TTCP	PCP
Fraser River - North Arm Stns. 10, 11 & 15	2	Staghorn sculpin (Leptocottus armatus)	45.0+24.0 (28.0-62.0)	48.8+15.9 (37.5-60.0)
	2	Northern squawfish (Ptychocheilus oregonensis)	10.5+9.9 (ND-18.0)	10.8+13.1 (1.5-20.0)
	3	Dolly varden (Salcelinus malma)	ND (ND-ND)	38.0+16.5 (22.0-55.0)
	2	Largescale sucker (Catoftomus macrocheilus)	ND (ND-ND)	43.0+18.4 (30.0-56.0)
	2	Peamouth chub (Mylocheilus caurimus)	ND (ND-ND)	ND (ND-ND)
	2	White sturgeon (Acitenser transmontana)	ND (ND-ND)	ND (ND-ND)
	2	Cutthroat trout (Salmo clarki clarki)	ND (ND-ND)	ND (ND-ND)
	5	Black crappie (Pomoxis nigromaculatus)	ND (ND-ND)	2.7+0.5 (ND-TR)
Fraser River - South Arm Stns. 12, 13 & 14	5	Staghorn sculpin (Leptocottus armatus)	16.8+14.5 (TR-37.0)	70.9+34.9 (39.0-125.0)
	4	Northern squawfish (Ptychocheilus oregonensis)	ND (ND-ND)	22.8+6.8 (15.0-29.0)
	3	Dolly varden (Salcelinus malma)	ND (ND-ND)	31.7+6.7 (26.0-39.0)
	2	Largescale sucker (Catoftomus macrocheilus)	ND (ND-ND)	ND (ND-ND)
	2	Peamouth chub (Mylocheilus caurimus)	ND (ND-ND)	ND (ND-ND)
	2	White sturgeon (Acitenseb transmontana)	ND (ND-ND)	ND (ND-ND)
	2	Rainbow trout (Salmo gairdneri)	ND (ND-ND)	3.1+0.8 (ND-3.7)
Fraser River - Upper Estuary Stns. 16 & 17	2	Northern squawfish (Ptychocheilus oregonensis)	ND (ND-ND)	27.0+1.4 (26.0-28.0)
	2	Largescale sucker (Catoftomus macrocheilus)	ND (ND-ND)	3.7+1.8 (ND-TR)
	2	Peamouth chub (Mylocheilus caurimus)	ND (ND-ND)	5.5+4.2 (ND-8.5)
	3	White sturgeon (Acitenser transmontana)	ND (ND-ND)	ND (ND-ND)

TABLE 27 (Cont'd) LEVELS OF TETRACHLOROPHENOL (TTCP) AND PENTACHLOROPHENOL (PCP) (ppb wet weight) IN FISH COLLECTED IN 1972-73 FROM THE FRASER RIVER AND THE UPPER ESTUARY (Adapted from Garrett, 1980)

Location	No. of Samples	Species	TTCP	PCP
Fraser River - Chilliwack Area				
Stns. 18, 19 and 20	2	Peamouth chub (Mylocheilus caurimus)	ND (ND-ND)	ND (ND-ND)
	4	Cutthroat trout (Salmo clarki clarki)	ND (ND-ND)	ND (ND-ND)
	3	White sturgeon (Acitenser transmontana)	ND (ND-ND)	ND (ND-ND)
	1	Black crappie (Pomoxis nigromaculatus)	ND	ND
	2	Rainbow trout (Salmo gairdneri)	ND (ND-ND)	ND (ND-ND)
	1	Staghorn sculpin (Leptocottus armatus)	22.0	82.0
	2	Northern squawfish (Ptychocheilus oregonensis)	ND (ND-ND)	17.5+4.0 (14.0-21.0)
	3	Dolly varden (Salcelinus malma)	ND (ND-ND)	32.7+14.4 (20.5-48.5)
	2	Largescale sucker (Catoftomus macrocheilus)	ND (ND-ND)	2.7+0.5 (ND-TR)
Fraser River - Hope Area				
Stns. 21, 22 & 23	2	Northern squawfish (Ptychocheilus oregonensis)	ND (ND-ND)	14.5+2.1 (13.0-16.0)
Fraser River - Hope Area				
Stn. 23	1	Dolly varden (Salcelinus malma)	ND	33.0
	2	Largescale sucker (Catoftomus macrocheilus)	ND (ND-ND)	3.1+0.8 (ND-3.7)
	2	Peamouth chub (Mylocheilus caurimus)	ND (ND-ND)	ND (ND-ND)
	4	Cutthroat trout (Salmo clarki clarki)	ND (ND-ND)	ND (ND-ND)
	2	Rainbow trout (Salmo gairdneri)	ND (ND-ND)	ND (ND-ND)

TABLE 28 CHLOROPHENOLS IN LIVER FAT OF FISH CAUGHT IN THE VICINITY OF A PULP MILL PRODUCING FULL BLEACH SULPHATE PULP. (From Landner et al, 1977).

Species	Weight of fish (g)	Fat content (%)	Concentration (µg/g fat) of: 2.4.6-trichlorophenol	tri-chloro-guaiacol	tetra-chloro-guaiacol
Perch (Perca fluviatilis)	200	2.3	2.7	11.5	8.2
Northern pike: (Esox lucius)	370	10.1	0.4	2.0	0.5
" "	600	5.5	0.5	1.5	4.4

South America

Frogs and fishes which had been collected from NaPCP treated rice fields in Surinam (Sect. 5.1.4.1) were analyzed for PCP residues. Frogs, Pseudis paradoxa, which died following the NaPCP treatment, contained 8.1 ppm of PCP based on mean wet-weight of 6 composite samples. Three species of fish which were dead post-treatment contained mean wet-weight residues of 31.2, 41.6 and 59.4 ppm of PCP. The same species of live fish from unsprayed ditches contained mean wet-weight residues of 1.77, 8.76 and 13.4 ppm of PCP (Vermeer et al, 1974).

5.2 Residues in Terrestrial Systems

The information on CP residues in terrestrial systems is grouped under the general headings of soil, treated wood, plants, animals, humans, food and livestock feed. There is a general lack of Canadian CP residue data in terrestrial systems. To give the reader the proper perspective on the CP residues in terrestrial-aquatic systems, data from non-Canadian sources are included.

5.2.1 Soil

Canada

In Canada, PCP is the only CP which has a registered minor agricultural use as a herbicide, and then only in a mixture with the herbicides bromacil, or with 2,4-D present

as dimethylamine salt or isooctyl esters, and prometon. The low use of PCP as a soil herbicide may account in part, for the deficiency of soil residue data.

Recent analysis of soil samples, taken from a site formerly occupied by a pesticide plant in Richmond, B.C., indicated levels of TTCP of 2.0 ppm, TCP of 0.18 ppm, and low levels of PCP, at less than 0.05 ppm (Sect. 5.1.1, Sect. 5.1.2) (Garrett, 1980).

United States

Soil residue data were developed to determine if leaching or bleeding of CPs from treated wood can result in significant entry to the environment. Arsenault (1976) reported the collective results of analyses of soil samples taken adjacent to PCP treated poles from sites in Alabama, Georgia, South Carolina, Tennessee and Oregon. Soil types represented varied from clay to sand. Poles had been treated with PCP by either the Penta-Petroleum or Cellon process. Arsenault's (1976) summary of the investigations was as follows:

> "Results . . . of the analyses show that the PCP concentration in the one inch collar of soil immediately adjacent to the pole averaged 658 ppm, with one value as high as 9500 ppm. At a distance of 12 inches from the poles the soil had an average concentration of only 3.4 ppm with the maximum being only 40 ppm. This indicates either degradation of PCP by the soil or a lack of migration into the soil. However, at distances of 5 feet from the poles, the PCP concentration in the soil averaged 0.26 ppm, which is just about the same as the blank found in non-exposed soil samples (1 to 2 µg/sample or 0.2-0.4 ppm)."

In commenting on these results Fox (1978b) suggested that the blank of 0.26 ppm of PCP in unexposed soils seemed high, since detection limits are much lower. Further, this kind of background level would imply an incredibly high loading since PCP is not known to occur naturally. A possible cause for the high background level could be reagent contamination.

5.2.2 Treated Wood

Canada

In a joint undertaking during 1978-79, the Ontario Ministry of Agriculture and Food (O.M.A.F.) Veterinary Services Laboratory and the O.M.A.F. Provincial Pesticide Residue Testing Laboratory, Guelph, Ontario, collected and analyzed for PCP, 153

samples of wood shavings which were used as livestock litter on southern Ontario farms (Table 29). Over 90% of the samples came from poultry houses and the remainder from swine pens and other livestock areas.

To supplement the general survey information on PCP in wood shavings, additional samples were analyzed for other CPs and chlorinated anisoles. Although no TCP nor trichloroanisole were detected, TTCP and PCP and their corresponding chlorinated anisoles were detected and quantified (Table 30). Information was also obtained on changes in levels of CPs and chlorinated anisoles in shavings after 56 days use as litter in a poultry house actively used for broiler production (Table 30). A dramatic change in level of PCP, from 628 ppm to 96 ppm, was noted in one set of litter samples taken prior to and following the 56 days of use.

United Kingdom

Parr et al (1974) reported on a survey for presence and levels of CPs in wood shavings used in broiler house litter in the United Kingdom (U.K.). The shavings were derived from imported lumber, surface-treated prior to shipment with the sodium salts of 2,3,4,6-TTCP and/or PCP to prevent sap-stain (blue-stain). Treated lumber comprised over 90% of the lumber imported into the U.K. in 1972. As a result of planing the treated lumber most of the CPs removed from the lumber are concentrated in the shavings. Analyses of shavings from 32 broiler houses showed levels of 4 to 307 ppm (mean 53 ppm) of 2,3,4,6-TTCP and 1 to 83 ppm (mean 12 ppm) of PCP. Spent litter contained an average of 0.7 ppm TTCP and 0.3 ppm PCP. In only a few samples of fresh litter were chloroanisoles detected and then only in trace amounts. Spent litter contained a mean level of 0.5 ppm of tetrachloroanisole, and six out of 32 samples which contained pentachloroanisole had a mean level of 0.03 ppm. Spent litter had increasingly been used as a constituent of animal feed. The authors conservatively estimated that the 203 million kg of shavings used annually during broiler chicken and turkey production, could contain as much as 10,000 kg of 2,3,4,6-TTCP and PCP.

Sweden

Levin and Nilsson (1977) identified, by tlc and gc, concentrations of chlorinated contaminants present in wood-dust from the trimming-grading plant in a Swedish sawmill (Table 31). The wood had previously been treated with 2% Na-TTCP. Analyses of wood dust from several sawmills in Sweden indicated that the presence of the chlorinated contaminants was not an isolated problem.

TABLE 29 LEVELS OF PENTACHLOROPHENOL (PCP) IN WOOD SHAVINGS SAMPLES FROM SOUTHERN ONTARIO FARMS - 1978-79

PCP (µg/g)	No. of samples	% Total samples
< 0.1	18	11.8
0.1 - 1.0	41	26.8
1.1 - 10	57	37.2
11 - 50	26	17.0
51 - 100	7	4.6
101 - 140	4	2.6
Total	153	100

TABLE 30 LEVELS (µg/g) OF CHLOROPHENOLS AND CHLOROANISOLES IN WOOD SHAVINGS COLLECTED IN ONTARIO IN 1979

		Chlorophenols[1]		Chloroanisoles[1]	
Sample collection sites		Tetra	Penta	Tetra	Penta
I Farm survey samples		0.48	0.93	ND	ND
		2.60	2.30	ND	ND
		2.00	16.9	0.01	0.06
		12.9	37.5	0.20	0.30
II Broiler house litter samples					
A) Before use	(1)	1.5	8.5	0.02	0.04
	(2)	1.8	8.8	0.03	0.02
	(3)	70	628	0.08	0.11
B) After use (56 day period)	(1)	1.30	5.8	0.001	ND
	(2)	10.4	2.6	0.03	0.02
	(3)	21	96	0.84	0.25

ND = not detected
[1] No trichlorophenol or trichloroanisole detected
Data source: Provincial Pesticide Residue Testing Laboratory, O.M.A.F., Guelph, Ontario N1G 2W1

5.2.3 Plants. Available information on levels of CPs in plants in terrestrial situations is almost non-existant. In an investigation of a fish kill in British Columbia following the on-site PCP treatment of a power line pole, foliage on vegetation at the pole site had 2320 ppm of PCP two days after the application (Alderdice, 1978).

TABLE 31 CONCENTRATIONS* OF CHLORINATED CONTAMINANTS IN WOOD-DUST FROM THE TRIMMING-GRADING PLANT IN A SWEDISH SAWMILL. WOOD PREVIOUSLY TREATED WITH 2% Na-2,3,4,6-tetrachlorophenate** (Adapted from Levin and Nilsson, 1977)

Sample	2,3,4,6-tetra-chlorophenol (ppm)	Penta-chloro-phenol (ppm)	Chlorophenoxy-phenols (Cl_5,Cl_6, Cl_7,Cl_8) (ppm)	Chloro-dibenzo-furans (Cl_6,Cl_7) (ppm)	Chloro-dibenzo-dioxins (ppm)
1	300	100	40	6	<0.5
2	110	30	10	1	<0.5
3	100	100	10	3	<0.5
4	800	400	50	10	<0.5

*Concentrations represent recoveries of 70% or less.
**Composition: 10% 2,4,6-TCP, 70% 2,3,4,6-TTCP, 20% PCP, 1600 ppm chlorophenoxyphenols (Cl_5,Cl_6,Cl_7,Cl_8), 70 ppm chlorodibenzofurans (Cl_6, Cl_7), <1 ppm chlorodibenzodioxins.

5.2.4 Animals

Canada

Reports of CP residue levels in terrestrial vertebrates are scarce with only one from Canada. Purple martin fledglings from the central region of Alberta were analyzed for CPs by Alberta Agriculture Food Laboratory, Edmonton, Alberta. They detected 0.031 ppm PCP and 0.002 ppm of TTCP (Currie, 1978).

United States

An investigation into health problems in a Michigan dairy herd in 1977, led to the finding of PCP as a contaminant in bone marrow, fat, blood serum, and liver of the animals (Hoeting, 1977; Anonymous, 1977a). An additional seven herds had detectable levels of PCP in the blood of both cows and calves. One herd housed in a total-confinement barn, constructed in part of PCP-treated wood, had PCP levels in their blood of 270 to 570 ppb PCP (U.S. E.P.A. 1978b).

United Kingdom

Chickens that were exposed to CPs in broiler house litter contained up to 80 µg of 2,3,4,6-tetrachloroanisole and 0.02 µg of pentachloroanisole per gram of fresh tissue (Curtis et al, 1972).

South America

A major feature of the research project investigating the impact on fauna following the application of NaPCP to rice fields in Surinam (Sect. 5.1.4.1) was the determination of PCP residues in tissues from several species of birds (Table 32). The PCP residue levels in tissues from apparently healthy, sick or dead birds ably demonstrated the effect of a to ic material on the predator/prey relationships. As a result of this research an equally effective molluscicide, Bayluscide® was recommended to replace NaPCP (Vermeer et al, 1974).

5.2.5 Humans

Canada

In Canada, levels of CPs in humans have not been determined except for a relatively few cases where CPs were implicated in industrial over-exposure incidents. For example, in 1963, at a Winnipeg, Manitoba, window-sash factory, when employees were

TABLE 32 PENTACHLOROPHENOL (PCP) RESIDUES (MEAN WET-WEIGHT ppm) IN SELECTED SPECIES OF BIRDS COLLECTED NEAR WAGENINGEN, SURINAM, 1971. (Adapted from Vermeer et al, 1974)

Species	Predominant prey group	No. birds in sample pool	Tissues analyzed	No. tissue samples analyzed	% fat in tissues	PCP residues (ppm)	Notes: bird feeding/collection sites
Snail kites	Pomacea	5	brain	5	6.5	0.10	Collected on rice fields
(Rostrhamus	snails	5	liver	5	2.5	0.19	Collected on rice fields
sociabilis)		17	brain	17	4.83	11.25	Dead birds collected at roost site
		17	liver	17	4.29	45.56	Dead birds collected at roost site
		17	kidney	10	1.35	20.34	Dead birds collected at roost site
		3	brain	3	4.2	2.11	Birds shot returning to roost from rice fields
		3	liver	3	2.0	10.4	Birds shot returning to roost from rice fields
		3	kidney	3	1.1	16.6	Birds shot returning to roost from rice fields
		3	brain	3	6.0	0.04	Birds shot returning to roost from fresh water marsh
		3	liver	3	2.7	0.14	Birds shot returning to roost from fresh water marsh
		3	kidney	3	0.6	0.10	Birds shot returning to roost from fresh water marsh
Black vulture							
(Coragyps atratus)	rodents	5	brain	5	4.8	0.09	Collected on rice fields
		5	liver	5	3.1	0.06	Collected on rice fields
Common egret	fishes						
(Egretta alba)		10	brain	10	6.0	0.08	Collected on rice fields
		10	liver	10	2.8	0.14	Collected on rice fields
		1	brain	1	3.6	2.04	Sick bird, collected at roost site
		1	liver	1	0.8	10.0	Sick bird, collected at roost site
		1	kidney	1	1.6	5.87	Sick bird, collected at roost site
		1	brain	1	4.7	1.66	Dead bird, collected at roost site
		1	liver	1	0.4	5.56	Dead bird, collected at roost site
		1	kidney	1	0.8	3.16	Dead bird, collected at roost site
		9	brain	9	3.77	2.98	Dead or sick birds collected at rice fields
		9	liver	9	1.42	5.14	Dead or sick birds collected at rice fields
		9	kidney	7	0.89	1.35	Dead or sick birds collected at rice fields
Snowy egret	fishes &	10	brain	10	5.2	0.10	Collected on rice fields
(Egretta thula)	insects	10	liver	10	2.0	0.19	Collected on rice fields
		-	brain	-	3.2	0.48	Dead and sick birds in rice fields
		-	liver	-	1.2	1.79	Dead and sick birds in rice fields
Cattle egret	insects	10	brain	10	3.5	0.49	Collected on rice fields
(Ardeola ibis)		10	liver	10	3.2	0.07	Collected on rice fields
		-	brain	-	2.8	0.06	Collected on rice fields
		-	liver	-	1.6	0.10	Collected on rice fields

over-exposed to PCP, analysis for PCP in urine of workers was carried out, both prior-to and following precautionary measures (Table 33).

TABLE 33 PENTACHLOROPHENOL LEVELS (mg/L) IN URINE OF FACTORY WORKERS (Excerpted from Bergner et al, 1965).

	Pentachlorophenol levels (mg/L)		
Case no.	Date of sample	Sept. 4/63	Sept. 30/63	Oct. 15/63
2		10.8	17.5	1.8*
4		5.4		0.6**
5		2.4	10.0	0.1**

*Two days after removal from exposure
**After institution of proper precautions

United States

Bevenue and Beckman (1967) cited the report of Akisada (1965) who detected PCP and TTCP levels of 1.10 to 5.91 mg/L, and 0.07 to 0.37 mg/L, respectively, in urine of workers in a factory with air levels of PCP of 14.04 mg/m^3, and levels of TTCP of 3.54 mg/m^3; non-exposed individuals had 10 - 50 µg/L of PCP and 10 - 30 µg/L of TTCP.

Concurrent with the Bevenue and Beckman (1967) review of PCP, Bevenue et al (1967) reported the results of a survey of PCP content in human urine from 541 people residing in Hawaii. A total of 130 occupationally exposed pest control operators in 30 different commercial firms had mean concentrations of PCP in urine of 28 to 12,990 ppb (overall mean of 1802 ppb). The non-occupationally exposed group of 117 individuals had PCP levels of 14 to 186 ppb (overall mean of 40 ppb). A third group of 294 individuals composed of occupationally and non-occupationally exposed individuals had mean PCP levels of 465 ppb and 44 ppb, respectively.

Cranmer and Freal (1970) developed an analytical procedure for gc analysis of PCP in human urine. They observed that the mathematical means of PCP concentrations in human urine from six general population individuals, four replicates per sample, ranged from 2.2 to 10.8 ppb. During the same mini-survey, four individuals, including a carpenter, a boat-builder, and two spraymen, had a concentration range of PCP in urine of 24.1 to 265 ppb.

PCP levels in blood serum and urine of personnel in a year-round wood treatment plant in Idaho were determined by Wyllie et al (1975). Serum PCP levels ranged from 348.4 to 3,963.0 ppb for the exposed group and from 38.0 to 68.0 ppb for the control, a chemist. PCP levels in urine averaged 163.8 ppb (range 41.3 to 760.6 ppb) for the exposed group, and 3.4 ppb for the control group.

Dougherty and Piotrowska (1976) developed a procedure for screening by negative chemical ionization mass spectrometry for residues of toxic environmental contaminants within human urine. They found that urine samples from Florida State University dormitory residents and swim team members had concentrations of PCP of approximately 20 ppb, which was akin to the levels observed by Bevenue et al (1967), in non-occupationally exposed individuals in Hawaii.

Edgerton and Moseman (1979) developed a hydrolysis method for the determination of PCP in urine which gave yields as much as 17-fold higher than with previous methods for PCP determinations in urine, including the EPA Analytical Manual Method and the Cranmer and Freal (1970) method (App. 2, Sect. 2.1.3). They found PCP levels of 0.02 - 0.08 ppm in human urine samples from the general population, with levels in two exposed workers of 1.71 and 3.68 ppm, compared to 0.21 and 0.31 ppm, respectively, with the method of Cranmer and Freal (1970).

In preliminary sampling of human seminal fluid, Dougherty and Piotrowska (1976) observed PCP average concentration of 50 ppb (range 20 - 70 ppb) with every sample showing at least one other organic polychloride.

Shafik (1973) used ec-gc and ms to detect and quantify ethylated derivatives of PCP in human adipose tissue from the general population. The limit of detection for PCP in adipose tissue was 5 ppb, with average recoveries of 75%. The levels of PCP found in 18 subjects ranged from 12 - 52 ppb with an average content of 26.3 ppb. Shafik (1973) concluded that humans are continuously exposed to low levels of PCP from the environment and he also noted that "no-effect" levels had not been established for PCP in humans.

Japan

Ohe (1979) reported levels of PCP in 25 samples of human adipose tissue from 13 males, 9 females, and 3 unknowns, none of whom were known to have been occupationally exposed to CPs. Analysis was done with ec-gc, and gc-ms to detect, quantify, and confirm PCP acetate derivatives in the adipose tissue. The limit of detectability for PCP in the adipose tissue was 5 ppb. Levels of PCP of less than twice

background were designated as trace amounts. The analytical method used by Ohe (1979) allowed recoveries in the range of 85 - 98% with a sensitivity of 1 ppm on samples of fortified butter fat. PCP content in fat extracted from human adipose tissue, from the 25 subjects in hospitals at Kyoto and Osaka in 1974, ranged from ND to 0.57 ppm, with a mean value of 0.14 ppm and a calculated standard deviation of 0.04 ppm. The PCP determinations utilized the acetyl derivatives. The sensitivity of the gc detection to PCP acetate was approximately 2.5 pg. The reported levels were slightly higher than those of Shafik (1973). Ohe (1979) stated that although the levels were not toxicologically significant, there was no information available on the source and routes of the PCP residues.

5.2.6 Food

Canada

Published information on levels of CPs in Canadian foods is scarce. A report by Swackhammer (1965) on pesticide residues in restaurant meals in Canada did not include information on CPs. There was no indication that the residue analysis included a search for these compounds. Neither did Smith (1971) mention CPs in her report on pesticide residues in the total diet in Canada.

In contrast to the United States where annual survey data on Pesticides on a regional and national basis are readily available, such information is not made readily available to the general public in Canada. The Alberta Department of Agriculture has sampled for, and detected, PCP and TTCP in agricultural produce (Table 34) (Currie, 1978; 1979). Although the samples are not necessarily representative of the normal food supply, the results of the analyses do indicate that the presence of CPs in agricultural produce can, in some cases, be a consequence of cross-contamination between CP treated wood and agricultural produce exposed to it.

The general plan of operation for a pesticides-in-food survey is to obtain "grab" samples of raw foods for chemical analysis to determine possible occurrence of pesticides, including CPs. Product sampling occurs at time of delivery to the market, prior to processing, when sources of the food would still be identifiable. If abnormally high levels of pesticides are detected then the sources of the contaminants can be located and remedial action can be taken.

TABLE 34 SUMMARY OF SAMPLES ANALYZED FOR PENTACHLOROPHENOL (PCP) AND TETRACHLOROPHENOL (TTCP) BY THE ALBERTA DEPARTMENT OF AGRICULTURE, FOOD LABORATORY, EDMONTON, ALBERTA, FOR THE PERIOD 1-75 TO 12-78

Lab. File No.	Sample Type	PCP (ppm)	TTCP (ppm)	Region of Alberta
5-319	(a)Hide Scraping	1938 Fat Basis	Not determined	Central
6-657	(a)Carrots	0.013	0.005	Central
6-658	(a)Potatoes	0.058	0.015	Central
7-069	Potatoes	Trace	ND	Central
7-071	Potatoes	Trace	ND	Central
7-082	Potatoes	Trace	ND	South
7-093	Turnips	Trace	ND	Central
7-132	Carrots	0.004	ND	Central
7-300	Potatoes	Trace	Trace	Central
7-301	Potatoes	0.001	Trace	Central
7-302	Potatoes	0.003	Trace	Central
7-303	Potatoes	0.001	Trace	Central
7-304	Potatoes	Trace	Trace	Central
7-368	(a)Bee Crates	0.47	0.020	Central
7-625	Potatoes	0.028	0.002	Central
7-698	Potatoes	0.001	Trace	Central
7-699	Potatoes	0.002	Trace	Central
7-701	Potatoes	Trace	Trace	Central
7-702	Potatoes	0.001	Trace	Central
7-703	Potatoes	0.002	Trace	Central
7-704	Potatoes	Trace	Trace	Central
7-705	Potatoes	Trace	Trace	Central
7-706	Potatoes	0.005	Trace	Central
7-707	Potatoes	0.001	Trace	Central
7-708	Potatoes	0.002	Trace	Central
7-709	Potatoes	0.002	Trace	Central
7-710	Potatoes	0.003	Trace	Central
7-711	Potatoes	0.001	Trace	Central
7-712	Potatoes	Trace	Trace	Central
7-713	Potatoes	0.002	Trace	Central
7-833	Raw Milk	0.002	ND	Central
7-834	Raw Milk	0.005	ND	Central
7-835	Raw Milk	0.002	ND	Central
7-845	Raw Milk	0.002	ND	Central
7-847	Potatoes	0.009	0.001	North
7-848	Carrots	0.008	0.002	North
7-849	Turnips	0.003	Trace	North
7-850	Raw Milk	0.001	ND	Central
7-851	Raw Milk	0.001	Trace	Central
7-866	Potatoes	0.003	0.001	Central
7-867	Cabbage	0.001	ND	Central
7-868	Beets	0.004	0.001	Central
7-877	Potato	0.005	Trace	Central
7-878	Potato	Trace	ND	Central

TABLE 34 (Cont'd) SUMMARY OF SAMPLES ANALYZED FOR PENTACHLOROPHENOL (PCP) AND TETRACHLOROPHENOL (TTCP) BY THE ALBERTA DEPARTMENT OF AGRICULTURE, FOOD LABORATORY, EDMONTON, ALBERTA, FOR THE PERIOD 1-75 TO 12-78

Lab. File No.	Sample Type	PCP (ppm)	TTCP (ppm)	Region of Alberta
7-879	Potato	Trace	Trace	Central
7-880	Potato	0.007	Trace	Central
7-881	Potato	Trace	ND	Central
7-882	Potato	0.007	Trace	Central
7-883	Potato	Trace	Trace	Central
7-884	Potato	0.006	Trace	Central
7-885	Potato	0.004	Trace	Central
7-886	Potato	0.009	Trace	Central
7-887	Potato	0.005	Trace	Central
7-888	Potato	0.009	Trace	Central
7-889	Potato	0.003	Trace	Central
7-890	Potato	0.008	Trace	Central
7-891	Potato	0.005	Trace	Central
7-892	Potato	0.279	0.035	Central
8-096	(a)(b)Potato	2.71	0.472	South
8-097	(a)(c)Potato	0.073	0.022	South
8-175	Potatoes	0.002	0.001	Central
8-176	Potatoes	0.001	ND	Central
8-177	Potatoes	0.002	Trace	Central
8-178	Potatoes	0.035	0.007	Central
8-179	Potatoes	0.002	ND	Central
8-180	Potatoes	0.002	Trace	Central
8-181	Potatoes	0.043	0.045	Central
8-182	Potatoes	0.003	Trace	Central
8-183	Potatoes	0.003	Trace	Central
8-184	Potatoes	0.001	ND	Central
8-185	Potatoes	0.001	Trace	South
8-186	Potatoes	0.001	Trace	South
8-187	Potatoes	0.001	Trace	South
8-188	Potatoes	0.003	Trace	South
8-205	(d)Potato Pulp	0.005	0.014	South
8-206	(d)Potato Peel	12.5	2.16	South
8-207	(e)Potato Pulp	0.006	0.001	South
8-208	(e)Potato Peel	0.157	0.150	South
8-750	Carrots	Trace	ND	South
8-751	Potatoes	Trace	ND	South
8-795	Potatoes	ND	ND	South
8-796	Carrots	ND	ND	South

Note
(a) biases samples.
(b) Sample 8-096 was taken from next to support post in wooden bin. The wood had been treated with a PCP formulation in 1964.
(c) Sample 8-097 came from the same bin as sample 8-096 but from somewhere near the center of the pile of potatoes.
(d) Samples 8-205 and 8-206 are subsamples of sample 8-096
(e) Samples 8-207 and 8-208 are subsamples of sample 8-181
ND = not detected; Trace = <0.001 ppm

In 1977, 45 composite samples of milk collected from 8 individual bulk milk transporters picking up milk in southern Ontario were analyzed for PCP, 2,3,4,6-TTCP; 2,4,5-, and 2,4,6-TCP (Frank et al, 1978). These CPs were not detected in whole milk at detection levels of 0.1 ppb for PCP and 1 ppb for the tri- and tetra-CPs.

United States

In a review on pesticide residues in American food, Duggan and Duggan (1973) in the introduction to a section on data source, noted that:

> "The total amount of data on pesticide residues in food is so great that even bibliographies of the papers containing it are unmanageable in length. Carefully chosen descriptors and a computerised search will still result in a mixture of three types of data with consequent difficulties of comparison. These categories are research data, surveillance data, and survey data."

The most accessible United States data fall into the survey, or monitoring, category. The data on pesticide residues in total diet samples have been published on an annual basis in the Pesticide Monitoring Journal. In the residue reports compiled for the late 60's and early 70's (Duggan et al (1967), Martin and Duggan (1968), Corneliussen (1969), Corneliussen (1970), Manske and Corneliussen (1974), Johnson and Manske (1976), and Manske and Johnson (1977), PCP was identified as one of the pesticides found infrequently. From 1965 to 1969 PCP residues were annually identified in trace to low amounts (0.004 - 0.310 ppm) in dairy products - meat, fish, and poultry - and in grain and cereal. In the 1970's only a few samples with trace amounts of PCP were noted, in those food classes. With much lower incidence and in only trace amounts, PCP was also identified during the regional surveys from 1965 to 1974 in the other classes of foods: potatoes, leafy vegetables, legume vegetables, root vegetables, garden fruits, fruits, oils, fats shortenings, sugar and adjuncts, and beverages. Duggan and Duggan (1973) showed the incidence (in %) of PCP residues in total diet in the U.S. from 1965 - 1970 as non-detectable (N.D.) to 3.3%, with a daily intake range of N.D. - 0.006 mg.

5.2.7 Livestock Feed. The circumstances described below which led to residues of PCP in a shipment of feed grains is an illustration of the problems which can arise from shipment of PCP in non-dedicated equipment or in railway cars which have not been adequately cleaned or decontaminated. In November 1977, a box-car was used for a shipment of PCP from the production plant in Alberta to a pole-treatment plant. The box-car was then used for movement of feed oats from northern Alberta to Thunder Bay,

Ontario. Following unloading of the PCP, the car was loaded with feed grain for movement to eastern Ontario. The feed grain became contaminated with PCP (average levels of 2000 ppm of PCP were detected in sweepings from the car) and was eventually used in cattle rations. The contaminated feed led to feed refusal by the livestock. Although residues of dioxins were also detected in the sweepings, the levels of hexa-, hepta- and octa-dioxin were not confirmed. No dioxins were detected in milk from cattle fed the PCP contaminated feed (Johnston, 1977).

5.3 Levels in the Atmosphere

Only a limited amount of data are available on CP levels in air. Wylie et al (1975) determined levels of PCP in the air at a year-round wood treatment plant in Idaho in 1972. Average air levels of PCP ranged from a low of 263 ng/m^3 in April to 1887.9 ng/m^3 in May. The highest air level recorded was 15,275.1 ng/m^3 in the pressure treatment room in February (Sect. 5.2.5, App. 3, Sect. 3.1.1.4).

6 RESIDUES OF POLYCHLORINATED DIBENZO-p-DIOXINS AND CHLORODIBENZOFURANS IN THE ENVIRONMENT

Improvements in analytical techniques and use of gc-ms equipment have been paralleled by lower detection limits (ppt) for PCDDs and PCDFs which occur in micro amounts (ppt) when present in environmental samples (App. 2, Sect. 2.2). This chapter includes the small amount of recently published residue data for these toxic impurities which were associated with CPs.

6.1 Treated Wood

Levin et al (1976) investigated the presence and levels of PCDDs and PCDFs in sawdust at Swedish sawmills which used CPs for wood treatment. Their results suggested that the impurities, such as PCDFs in the CP formulations, were subject to a concentration effect in the sawdust, in comparison to the CPs themselves, particularly in the sludge in the dipping tanks. For example, the content of HCDF and HpCDF in the commercial Na-2,3,4,6-TTCP, which is generally used in dipping tanks in Sweden, ranged from 70 - 150 ppm, while the total chlorophenoxyphenol level ranged from 2,000 to 5,000 ppm. The main products in the Na-2,3,4,6-TTCP were 50% TTCP, 10% PCP, and 5% TCP. Analysis of a sample of sludge from a dipping tank where a 2.5% solution of TTCP was used yielded the following levels of chlorinated contaminants: 200 ppm of chlorophenoxyphenols, and 700 ppm of CDFs. The multiplication factor for the CDF content in the sawdust sludge from the dipping tank compared to the starting material was 3X to 10X.

A study conducted at Oregon State University by Lamberton et al (1979) also demonstrated that PCDDs will accumulate both in the recirculating PCP solution used in the Boulton process of wood treatment and in the sludge in the bottom of the recirculating tank. Following analyses of samples from two local sawmills near Corvallis, Oregon, Lamberton et al (1979) stated that when normalized against the PCP content, the OCDD level was 34% higher in the recirculating PCP solution than in the fresh 5% PCP solution. The OCDD content of the sludge was 90% higher relative to the fresh solution and 42% higher when compared to the recirculating PCP solution. Similarly the HpCDD levels were 18% and 86% higher in the recirculating PCP solution and in the sludge, respectively, when compared to the fresh PCP solution. It was not possible to determine whether the increase in dioxin levels was due to 1) conversion of appropriate predioxins during the wood treatment process, 2) selective deposition of PCP in the wood leaving a

dioxin enriched solution, 3) low solubility of the PCDDs in the petroleum distillate used as the carrier, or 4) to some other factor.

During the course of an investigation into a PCP related health problem in a Michigan dairy herd in 1977, samples of structural penta-treated lumber from the dairy barn were analyzed for dioxins. The wood samples contained 6 to 15 ppm octa-, and 6 to 7 ppm hepta-CDD on a wood weight basis (Michigan Dept. Agric., 1977).

6.2 Fly Ash, Flue Gas, and Air-borne Particulates

Olie et al (1977) identified qualitatively, but not quantitatively, 5 CDDs and 5 CDFs in fly ash and 5 CDDs in flue gas from a municipal incinerator in the Netherlands. They stated that the amounts of these products entering the atmosphere were probably quite small.

Buser et al (1978) reported that in previous investigations (Buser and Bosshardt, 1978) they found a total of 0.2 ppm PCDDs and 0.1 ppm PCDFs in fly ash from a municipal incinerator in Switzerland and 0.6 ppm PCDDs and 0.3 ppm PCDFs in the fly ash from an industrial heating facility, also in Switzerland. They observed that OCDD was the major dioxin component in the fly ash from the municipal incinerator with a gas temperature at the sampling point of 260°C; PnCDD and HCDD were the major dioxins in the fly ash from the industrial heating plant stack. The flue gas temperature at the latter sampling point was 200°C. By using high resolution gc more than 30 individual PCDDs were identified in fly ash from the two sources.

Following the disclosure by Dow Chemical, U.S.A., that trace levels of PCDDs had been detected in fish taken from the Tittabawassee River near Midland, MI, (Anonymous, 1978) (Sect. 6.5.2), Michigan Division, Dow Chemical Co., undertook a major project to identify potential sources of the PCDDs. The project report by Dow's Chlorinated Dioxin Task Force (Dow Chemical, U.S.A., 1978) included the results of their survey for PCDDs in environmental samples. Samples included air and water borne particulates, soil, and food; all from various sources.

The Task Force (Dow Chemical, U.S.A., 1978) acknowledged that there were several weaknesses in the project and resulting report, and that these were due primarily to 1) limited sampling, 2) data not amenable to statistical analysis, 3) lack of validation of newly developed analytical methodology, 4) the small number of specifically trained technologists and scientists, 5) the necessity to depend on highly sophisticated instruments, 6) the large number of variables, and 7) the inherent difficulties of working with levels of PCDDs that were close to the limit of detection. The report advanced Dow's

viewpoint that PCDDs were perhaps ubiquitous in the environment and that the presence of PCDDs was due to the existence of a natural phenomenon termed by Dow as trace chemistries of fire, or in other words, PCDD synthesis has occurred in most combustion processes.

The Dow generated data (Dow Chemical, U.S.A., 1978) on environmental levels of PCDDs is extensively quoted in this and the following Sections, 6.3, 6.4, and 6.6. This is done to illustrate a) the varied environmental sources of samples from which Dow scientists identified PCDDs and also to b) provide evidence on the relationship between degree of chlorination of the PCDDs and the levels of the PCDDs in environmental samples, ie. levels of PCDDs increased from ND to trace levels of TCDDs to the comparatively higher levels of OCDDs, with the intermediate levels exhibited in the HCDDs and HpCDDs, although general levels were dependent on the source of the samples.

Dow's analyses of samples of air-borne particulate matter from industrial and domestic combustion processes showed the presence of PCDDs in emissions from fossil fueled powerhouses (Table 35), incinerators (Tables 36 and 37), mufflers from automobiles and diesel trucks (Table 38), and soot from fireplaces (Table 39).

TABLE 35 CHLORINATED DIOXINS IN PARTICULATES FROM THE DOW CHEMICAL CO., MIDLAND, MI, POWERHOUSE STACK (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, ng/g (ppb)				
	TCDD[A]				
	Other Isomers	2,3,7,8-	HCDD[B]	HpCDD[B]	OCDD[B]
Powerhouse, oil and coal-fired	38(20)	ND (10)	2	4	24

[A]Capillary column gc-ms [B]Analysis by electron capture gas chromatography

(Note: In all Tables excerpted from the Dow Task Force Report the assignment of TCDD levels under the specific 2,3,7,8-TCDD heading was arbitrary, except in those cases where capillary chromatography was used. The 2,3,7,8-TCDD column can include 2,3,7,8-TCDD plus 2 to 16 other isomers depending on analytical

TABLE 36 CHLORINATED DIOXIN CONTENT OF PARTICULATE MATTER FROM THE STATIONARY TAR BURNER AT THE DOW CHEMICAL CO., MIDLAND, MI (Dow Chemical, U.S.A., 1978)

Sample Identification*	Feed	Apparent Dioxins, ng/g (ppb)				
		TCDD				
		Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
R_1F_1	gas and tars	ND (1.0)	ND (1.3)	20	90	330
R_1F_2	gas and tars	ND (1.7)	ND (2.6)	7	125	440
R_2F_1	gas and tars	ND (1.2)	ND (2.0)	6	60	190
R_3F_1	gas and tars	ND (1.2)	ND (3.0)	4	160	320
R_4F_1	gas and tars	ND (0.7)	ND (1.5)	1	27	250

*R_1F_1 means Run 1 Filter 1; R_1F_2 means Run 1 Filter 2, etc.

methodology (Sect. 3.1). Signals less than 10 times noise will have the detection limit reported in parenthesis. "ND" means not detected at 2.5 times noise.

Eiceman et al (1979) identified by gc and gc-ms PCDFs and PCDDs in fly ash samples collected from municipal incinerators in Ontario, Japan, and the Netherlands. In the two municipal incinerators sampled in Ontario, estimated concentrations of TCDD in fly ash samples ranged from 0.4 to 4.0 ng/g.

6.3 Soil and Dust

Canada

No information was located in the literature on presence or persistence of PCDDs or PCDFs in Canadian soil.

United States

Arsenault (1976) reported on the presence of OCDD, relative to PCP, in soil at the base of utility poles treated with a petroleum oil-PCP solution. He concluded from his results (Table 40) that OCDD degraded at a slower rate than PCP and that there was little movement of OCDD away from the base of the treated poles.

Kimbrough et al (1977) described the epidemiological and laboratory investigation of a poisoning outbreak in Missouri traced to presence of 2,3,7,8-TCDD, 2,4,5-TCP,

TABLE 37 CHLORINATED DIOXIN CONTENT OF PARTICULATE MATTER FROM THE ROTARY KILN INCINERATOR, DOW CHEMICAL, MIDLAND, MI (Dow Chemical, U.S.A., 1978)

Sample Identification*	Fuel		Apparent Dioxins, ng/g (ppb)				
			TCDD				
	Kiln	Secondary	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
Without Supplemental Fuel							
R_1F_1	tars, solid waste, and gas	tars	1,800	2,800**	13,000	110,000	180,000
R_1F_2	"		5,000	8,200**	65,000	510,000	810,000
R_2F_1	"		3,300	110	1,300	2,000	3,000
R_3F_1	"		12,000	ND(260)	5,600	37,000	59,000
With Supplemental Fuel							
R_3	tars, solid waste, and gas	oil and gas	ND(8.0)	ND(2.0)	1.4	13.0	30.0
R_4	"		ND(7.0)	ND(5.0)	ND(1.0)	4.0	9.0
R_5	"		ND(2.0)	ND(2.0)	ND(0.5)	6.0	15.0
R_6	"	tars and gas	ND(2.0)	ND(4.0)	5.0	27.0	170.0
R_7	"		ND(2.0)	ND(2.0)	4.0	110.0	950.0

* R_1F_1 means Run 1 Filter 1; R_1F_2 means Run 1 Filter 2, etc.

** The high results reported for the 2,3,7,8- isomer are probably due to analysis by the non-specific gc-ms packed column method. Later results were by use of a capillary column which is specific for 2,3,7,8-. The TCDD results from Run 1 are not comparable to those from Run 2 and Run 3.

TABLE 38 CHLORINATED DIOXIN CONTENT OF PARTICULATE MATTER IN MUFFLERS FROM AUTOMOBILES AND DIESEL TRUCKS (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, pg/g(ppt)							
	TCDD		HCDD		HpCDD		OCDD	
Sample Source	Other Isomers	2,3,7,8-	GC-MS	GC-EC	GC-MS	GC-EC	GC-MS	GC-EC
Auto Mufflers								
No. 1 non-catalytic	4.0(2.0)	ND(2.0)	ND(14)	N.A.	ND(6)	3(0.4)	16(8)	10(0.6)
No. 5 catalytic carbon	ND(1.0)	ND(3.0)	ND(10)	2.0(0.4)	14(8)	10(0.4)	68	72(1.0)
No. 5 catalytic rust	4.0	0.4(0.1)	-	0.7(0.1)	+	3(0.1)	+	28(0.4)
No. 2 catalytic	0.1(0.1)	ND(0.2)	-	0.5(0.1)	+	2(0.2)	+	8(0.5)
Diesel Mufflers								
No. 7	ND(7.0)	ND(3.0)	ND(25)	4.0(2.0)	110(30)	35(2.0)	280	205(3.0)
No. 6	20	3.0(1.0)	20(15)	37(1.0)	100(15)	49(1.0)	260	190(3.0)

\+ Means the positive result was confirmed by gc-ms.
\- Means that gc-ms was unable to confirm the positive result.
N.A. Means not analyzed.
Limits of detection are given for all gc-ec results

TABLE 39 CHLORINATED DIOXIN CONTENT OF SOOT FROM FIREPLACES AND HOUSE DUST, MIDLAND, MI (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, ng/g(ppb)				
	TCDD				
Sample Source	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
Fireplace A	0.27	0.1(0.04)	3.4	16	25
Fireplace B	ND(0.04)	ND(0.04)	0.23*	0.67*	0.89*
Electrostatic Precipitator	0.40(0.40)	0.6(0.2)	34	430	1300

*Data obtained by gc-ec analysis, all others by gc-ms.

TABLE 40 PENTACHLOROPHENOL (PCP) AND OCTA- CHLORODIBENZO-p-DIOXIN (OCDD) IN THE SOIL AT THE BASE OF UTILITY POLES TREATED WITH PETROLEUM OIL - PCP SOLUTION. (Arsenault, 1976)

Distance from pole (inches)	PCP in soil (ppm)	OCDD in soil (ppm)	Ratio (pts OCDD/1000 pts PCP)
1	322	9.6	30
12	1.6	0.13	81

and PCBs in three riding arena soils which had been sprayed with salvage oil contaminated with toxic industrial waste products (App. 3, Sect. 3.3). Concentration of the 2,4,5-TCP was about 5600 ppm in one arena soil and the concentration of the associated 2,3,7,8-TCDD was approximately 32 ppm (Table 41). In addition to the disposal of the contaminated oil in arenas, a farm road was also treated with the oil. The concentration of 2,3,7,8-TCDD in the farm road soil, three years after treatment, was 0.61 ppm (Table 41).

TABLE 41 CHLORINATED COMPOUNDS IN SAMPLES OF SOIL AND OIL ASSOCIATED WITH DISPOSAL OF SALVAGE OIL, MISSOURI, U.S.A. (Kimbrough et al, 1977)

Sample origin and type	Date Collected	Concentrations 2,4,5-TCP	2,3,7,8-TCDD(ppm)	PCB(ppm)
Arena A soil	8-71	0.56 - 0.65%	31.8 - 33.0	1350 - 1590
	[a]8-74	Trace	None	None
Arena C soil	8-74	11.5 ppm	[b]0.22 - 0.44	15
from dump	8-74	1.8 ppm	0.49	25
sites	8-74	14.8 - 19.2 ppm	[b]0.63 - 0.85	20
	8-74	1.5 ppm	0.38	10
Farm road soil	9-74	32.6 ppm	0.61	None
Chemical waste oil from plant	9-74	30.4 - 34.3%	306 - 356	None

[a]Collected after arena had been excavated twice

[b]Column fractionated by method of Baughman and Meselson (1973). Method of Firestone et al (1972) used on all other TCDD samples.

As part of an overall assessment of possible sources of PCDDs in the Midland, MI, area, Dow Chemical, U.S.A. (1978) reported levels of CDDs in soil and dust samples from industrial and domestic sources in urban and rural areas (Sect. 6.2). Data on CDD content in soil samples from both within and outside the Dow Chemical plant area are included in Table 42. CDDs were detected in dust samples from a Dow Chemical research building (Table 43), from urban areas in Michigan (Table 44), and in both soil and dust samples from rural, urban, and major metropolitan areas (Table 45). As noted in Dow's report (Dow Chemical, U.S.A., 1978) TCDD content (content used in the inclusive sense of possibly 16 isomers) in three of the soil samples from urban and metropolitan areas of Michigan were confirmed by high resolution mass spectrometry (Table 46).

(Although the Dow data on CDD content in dust and soil samples are reported in this section, the source of the CDDs was air-borne particulate matter).

The U.S.E.P.A. reported that soil at the site of an o-CP spill from a railroad tank car at Sturgeon, MO, in January 1979, contained 2,3,7,8-TCDD at a level less than 0.01 ppb (Anonymous, 1979c). Samples of o-CP from the shipment contained trace amounts of TCDD, ie an average of 37 ppb TCDD.

Europe

Bonaccorsi et al (1978) reported that samples of soil, collected in 1976 from the Seveso area of Italy following the explosion at the chemical plant which produced TCP (Peterson, 1978), were analyzed for TCDD, with no distinction as to isomer. The soil samples contained up to 5477 µg TCDD/m^2 (Table 51) (App. 3, Sect. 3.3).

6.4 Sediment, Water, and Water-borne Particulates

Canada

No published data were found on levels of PCDDs or PCDFs in sediment and water from the Canadian environment.

United States

As noted in App. 7, Sect. 7.3.2, silt in an aquatic system in a herbicide test area in Florida had a level of 10 - 35 ppt of undifferentiated isomers of TCDD. The silt had originated from eroded soil which had 10 - 710 ppt of TCDD (Young et al, 1976).

Kearney et al (1973) noted the low solubility of TCDD in water (0.6 µg/L) as did Arsenault (1976) for OCDD (also see App. 2, Sect. 7.3.1). The latter stated that:

TABLE 42 CHLORINATED DIOXIN CONTENT OF SOIL SAMPLES FROM MIDLAND, MI (Dow Chemical, U.S.A., 1978)

	Apparent Dioxin Concentration ng/g (ppb)				
	TCDD				
Sample Number	Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
1A	17	16	280	3200	20500
1B	9	6	40	470	2500
2A	18	100	120	650	6300
2B	18	16	280	240	11700
5B	0.8	0.3	7	70	490

TABLE 43 CHLORINATED DIOXIN CONTENT OF DUST SAMPLES FROM A DOW CHEMICAL CO. RESEARCH BUILDING (Dow Chemical, U.S.A., 1978)

	Apparent Dioxin Concentration, ng/g (ppb)				
	TCDD				
Sample Source	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
1st Floor, Front	0.5	1.0	18(5)	240	960
1st Floor, Rear	2.3	2.3	28	520	3800
2nd Floor, Center	1.3	2.6	11(4)	140	650
2nd Floor, Rear	N.A.	0.7	9(7)	250	2600
2nd Floor, Rear (2 weeks after cleaning)	1.5	1.2	20(7)	320	2000
Air Intake	2.3	2.3	35(9)	1200	7500

"N.A." means not analyzed

TABLE 44 CHLORINATED DIOXIN CONTENT OF DUST SAMPLES FROM MIDLAND, MI, AND A METROPOLITAN AREA (Dow Chemical, U.S.A., 1978)

Sample Source	Apparent Dioxin Concentration, ng/g (ppb)			
	TCDD	HCDD	HpCDD	OCDD
Midland, Sample 1	0.03(0.02)	0.2	2.3	19
Midland, Sample 2	0.04(0.02)	0.4	3.9	31
Metro, Motorcycle Shop, 3300' Metro Burner	ND(0.02)	0.09	0.8	3.5
Metro Schoolyard, 800' Metro Burner	ND(0.02)	0.1	0.3	0.4
Metro Soil & Ashes 500' Metro Burner	ND(0.02)	ND(0.02)	ND(0.02)	ND(0.1)
Metro River Shoreline	ND(0.02)	0.3	1.0	3.8

"The solubility of OCDD in water is extremely small due to the hydrophobic nature of the molecule. A typical effluent water from a PCP-oil treating process was analyzed and contained 44 mg/L of penta (44 ppm) and 0.003 µg/L (3 ppt) of OCDD. This extremely low ratio of OCDD to 1000 parts of PCP (0.00007) is indicative of the low solubility of OCDD in water and its tendency to either stay in the sludge or in the oil. In fact the fairly high persistence in soil may be due to its low water solubility and, therefore, its unavailability to bacteria. The low water solubility also indicates that OCDD cannot leach into the ground waters and will not be an environmental pollutant."

Shadoff et al (1977) searched for 2,3,7,8-TCDD from two aquatic environments exposed annually to 2,4,5-T herbicide. Mud and water samples were obtained from a reservoir for recycled irrigation water for rice near Grady, Arkansas, and from an impoundment of the North Concho River, near San Angelo, Texas. The irrigation water cycle had been in use at Grady, Arkansas, for 18 years up to the time of sampling. With an average detection level of less than 10 ppt with gc-ms equipment, no TCDD was detected from the mud and water samples from these two areas.

The Dow Chemical, U.S.A. (1978) report on levels of CDDs in environmental samples, mainly from the Midland, MI, area (see Sect. 6.2, 6.3), also included data on levels of CDDs in their Michigan Division plant's aqueous streams. CDD levels were measured in cooling tower residues (Table 47), particulates from the rotary kiln

TABLE 45 CHLORINATED DIOXIN CONTENT OF SOIL AND DUST FROM RURAL, URBAN, AND MAJOR METROPOLITAN AREAS (Dow Chemical, U.S.A., 1978)

Sample Source	Apparent Dioxin Concentration, ng/g (ppb)			
	TCDD	HCDD	HpCDD	OCDD
Soil				
Rural 1	ND(0.005)	ND(0.02)	ND(0.005)	ND(0.02)
Rural 2	ND(0.03)	ND(0.02)	ND(0.02)	0.1(0.05)
Rural 3	ND(0.005)	ND(0.02)	ND(0.005)	ND(0.01)
Urban No. 1, Downtown, 600' ENE of Powerhouse	ND(0.01)	1.2	1.6	2.0
Urban No. 2, Downtown, 900' ENE of Powerhouse	ND(0.01)	ND(0.04)	0.23	0.96
Urban No. 3, Downtown, 1500' NNE of Powerhouse	ND(0.007)	0.03(0.03)	0.30	2.0
Urban No. 4, 600' NE of Powerhouse	ND(0.005)	ND(0.05)	ND(0.03)	0.05(0.03)
Urban No. 5, 300' NNE of Powerhouse	ND(0.009)	ND(0.04)	0.035(0.02)	0.20
Major Metro No. 1, 100' NE of Incinerator	ND(0.02)	ND(0.03)	0.14	0.41
Major Metro No. 2, 200' NE of Incinerator	ND(0.01)	0.03(0.03)	0.24	1.0(0.03)
Major Metro No. 3, 400' NE of Incinerator	0.03	0.31	3.3	22.0
Major Metro No. 4, 1000' NE of Incinerator	ND(0.02)	0.12(0.04)	1.4	8.5
Major Metro No. 5, 100' NE of Incinerator	0.006(0.003)	0.14	0.85	3.2
Major Metro No. 6, 200' NE of Incinerator	0.005(0.005)	0.04(0.03)	0.36	1.4
Major Metro No. 7, 400' NE of Incinerator	0.005(0.005)	0.09(0.05)	0.96	6.0
Major Metro No. 8, 1000' NE of Incinerator	ND(0.006)	0.02(0.02)	0.10	0.35
Rural No. 4	ND(0.003)	ND(0.03)	0.03(0.02)	0.10
Rural No. 5	ND(0.005)	ND(0.05)	0.05(0.03)	0.17
Rural No. 6	ND(0.005)	ND(0.02)	0.02(0.02)	0.16
Rural No. 7	ND(0.007)	ND(0.005)	ND(0.03)	ND(0.03)
Rural No. 8	ND(0.007)	ND(0.03)	0.03(0.02)	0.11(0.04)
Dust				
Major Metro Parking Lot >1000' NE of Incinerator	ND(0.02)	ND(0.08)	0.64	2.6
Major Metro Cocktail Lounge >1000' NE of Incinerator	0.04(0.04)	0.34(0.20)	3.2	8.2

TABLE 46 HIGH RESOLUTION CONFIRMATION OF THE TCDD CONTENT OF SELECTED SAMPLES OF SOIL FROM URBAN AND MAJOR METROPOLITAN AREAS (see Table 44) (Dow Chemical, U.S.A., 1978)

Sample Source	Apparent TCDD, ng/g (ppb) Low Res ms	High Res ms
Urban No. 1	ND(0.01)	ND(0.02)
Major Metro No. 3	0.03	0.03
Major Metro No. 7	0.005(0.005)	0.007(0.004)

TABLE 47 CHLORINATED DIOXIN CONCENTRATION OF COOLING TOWER RESIDUES FROM THE DOW CHEMICAL CO., MIDLAND, MI (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, ng/g (ppb)			
Location	TCDD	HCDD	HpCDD	OCDD
Northwest	ND(0.05)	ND(1)	25	119
East	ND(0.05)	ND(1)	12	56
Central No. 1	1.6(0.5)	10	20	107
Central No. 2	6.0	N.A.	N.A.	N.A.
Central No. 2 Wash Water	ND(0.06)	N.A.	N.A.	N.A.

incinerator scrubber waters (Table 48), and the scrubber water after filtration (Table 49). Sewer waters, prior to waste treatment and from various streams within the Dow Michigan Division plant, were also sampled and analyzed for CDDs (Table 50). The report (Dow Chemical, U.S.A., 1978) noted in Table 50, that although the OCDD levels were high enough to be considered positive, those for TCDD were very close to the limit of detection, and their value therefore was uncertain.

6.5 Animals

6.5.1 Invertebrates. Prawn (Peneidae) collected in the fall of 1970 in South Vietnam near areas heavily exposed to 2,4,5-T contained a mean level of 18 ppt of 2,3,7,8-TCDD (Baughman and Meselson, 1973). Similar type samples of River Prawn (Palaemonidae) had mean levels of 42 ppt of TCDD.

TABLE 48 CHLORINATED DIOXINS IN PARTICULATES FILTERED FROM ROTARY KILN INCINERATOR SCRUBBER WATER AT THE DOW CHEMICAL CO., MIDLAND, MI (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, ng/g (ppb)				
	TCDD				
Sample Identification	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
Without Supplemental Fuel	300	2200*	3400	26,000	42,000
With Supplemental Fuel	14	32*	200	970	1,200

*The analytical method did not separate 2,3,7,8- from 11 other isomers.

TABLE 49 CHLORINATED DIOXIN CONTENT OF FILTERED SCRUBBER WATER FROM ROTARY KILN INCINERATOR AT THE DOW CHEMICAL CO., MIDLAND, MI (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, ng/g (ppb)				
	TCDD				
Sample	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
Without Supplemental Fuel	0.0018(0.001)	0.001*(0.0006)	0.005	0.024	0.026

*The analytical method did not separate 2,3,7,8- from 11 other isomers.

Insect larvae, snails, diving beetles, and crayfish collected from streams draining a herbicide test area in Florida which had been subjected to massive applications of 2,4,5-T, did not contain any TCDD (Young et al, 1975).

No additional references could be located which cited environmental levels of TCDD in invertebrate fauna.

Data have been developed on levels of TCDD in invertebrate organisms exposed to TCDD in aquatic model ecosystems (App. 8, Sect. 8.2).

TABLE 50 ANALYSES OF SEWER WATERS BEFORE WASTE TREAMENT, DOW CHEMICAL CO., MIDLAND, MI (Dow Chemical, U.S.A., 1978)

Sample Number	Apparent TCDD pg/g (ppt)	Apparent OCDD pg/g (ppt)*
1 (a)	2(2)	60;20
(b)	ND(2)	22;160
(c)	ND(2)	400
2 (a)	4(1)	140
(b)	4(1)	150;140;160
(c)	ND(2)	800;1200;1100;1400
3 (a)	1(0.4)	18;20
(b)	ND(1)	6;3
(c)	ND(1)	ND(2)
4 (a)	2(1)	11
(b)	3	5
(c)	ND(0.5)	48
5 (a)	1(0.5)	180
(b)	3(2)	600;1000;1500
(c)	1(0.5)	1100
6 (a)	ND(3)	ND(5)
(b)	ND(2)	ND(5)
7 (a)	ND(1)	100,50
(b)	ND(10)	600
8 (a)	ND(10)	6
(b)	ND(25)	15
9 (a)	ND(5)	ND(10)
10 (a)	ND(50)	15
(b)	ND(25)	80

* Replicates were analyzed using different clean-up techniques

6.5.2 Vertebrates

6.5.2.1 Aquatic

Canada

During the course of a survey for PCDDs and PCDFs in aquatic vertebrates from the Bay of Fundy/Gulf of Maine area, Zitko (1972) analyzed a limited number of samples which included muscle and liver of white shark (Carcharodon carcharias), commercial herring oil, and groundfish herring fishmeal. No residues of CDDs or CDFs were found, with detection limits of 0.04 µg/g -TCDD, 0.02 µg/g - HCDD, and 0.01 µg/g -OCDD, 0.02 µg/g - CDF, and 0.01 µg/g - OCDF. This particular investigation with the negative findings was also reported by Zitko et al (1972).

United States

Young et al (1975) summarized a study on the ecological impact of repetitive aerial applications of herbicides on the ecosystem of a test area at Eglin United States Air Force Base, (U.S.A.F.B.), Florida, (also referred to in the paragraph under invertebrates, Sect. 6.5.1). The study reported on the detection of TCDD in fish as follows:

> "Concentrations of TCDD (12 ppt) were found in only two species of fish from the stream, Hotropis hypselopterus, sailfin shiner, and Gambusia affinis, mosquitofish. The sample of mosquitofish consisted of bodies with heads and tails removed. Two samples of sailfin shiners were analyzed; one containing viscera only and the other bodies less heads, viscera, and caudal fins. Only the viscera contained TCDD. Samples of skin, muscle, gonads, and gut were obtained from Lepomis punctatus, spotted sunfish, from the test grid pond. Levels of TCDD in those body parts were 4, 4, 18, and 65 ppt, respectively."

Also, in the United States, Shadoff et al (1977) conducted a study to determine whether 2,3,7,8-TCDD was accumulating in environments exposed to approved agricultural uses of 2,4,5-T herbicide on range land and rice fields (Sect. 6.4). They analyzed samples of catfish and bass from a pond in the rice growing area of Arkansas, and catfish and walleyed pike from a reservoir in Texas in a watershed exposed to 2,4,5-T for control of mesquite and brush. No TCDD was detected by a gc-ms procedure with a detection limit which averaged less than 10 ppt.

The New York State Department of Health, in a news release dated April 24, 1979, stated that a dioxin had been detected in two fish taken from Lake Ontario. A brown trout caught off Rochester had 6.5 ppt dioxin and a small-mouth bass taken from Oswego River harbour in the southeastern end of the lake contained 4.6 ppt of dioxin. The limit of detection was 0.83 ppt. Neither the New York State Department of Health news release nor the follow-up reports carried in the The Globe and Mail, Toronto, April 26 and 27, 1979, identified the dioxin isomers (Anonymous 1979a, 1979b). Parrott (1979), in a statement to the Ontario Legislature noted that the New York State Health Department officials recognized that the sample was too small to be conclusive.

A news report in Chemical Week, July 12, 1978 (Anonymous, 1978) noted that Dow Chemical Co., Midland, Michigan, had reported to the Michigan Department of Natural Resources that trace levels (0.01 - 0.02 ppb) of 2,3,7,8-TCDD had been detected in fish from the Tittabawassee River downstream from their plant. The source of the dioxins, whether from the Dow industrial complex at Midland, or otherwise, has not been fully resolved Smith, 1978; Rawls, 1979) (Sect. 6.2, 6.4).

South-east Asia

A limited number of samples of carp and catfish from two rivers in the interior of Vietnam and croakers from the sea coast were collected in 1970 from areas heavily exposed to the herbicide 2,4,5-T. Mean levels of TCDD detected 2 1/2 years later in these samples ranged from 49 to 801 ppt. The authors stated that the results suggested ". . . . that TCDD may have accumulated to biologically significant levels in the food chains in some areas of South Vietnam exposed to herbicide spraying." However, in the review by Shadoff et al (1977) they noted that Baughman (1974) had examined later samples taken in 1973 and could not demonstrate the presence of TCDD at detection limits of 20 to 150 ppt.

6.5.2.2 Terrestrial

Canada

In addition to the aquatic samples analyzed by Zitko (1972) for detection of PCDDs and PCDFs, a few samples of eggs of double-crested cormorants (Phalacrocorax auritus), and herring gulls (Larus argentatus) were analyzed. No residues of CDDs or CDFs were found, with the same detection limits as noted in the reference to Zitko's work under aquatic vertebrates, above.

Bowes et al (1973) reported that no CDFs or CDDs had been detected in herring gull eggs obtained in the spring of 1972 from Scotch Bonnet Island in Lake Ontario. The eggs were from colonies where virtually no young had hatched in 1972.

United States

No dioxins were detected in tissue from 19 bald eagle carcasses collected between 1966 and 1971 from 15 states in the continental U.S. and Alaska (Woolson et al, 1973). The minimum detection limit was 50 ppb.

From the intensive study on environmental impact of herbicides, particularly 2,4,5-T, conducted at the aerial application test site at Eglin U.S.A.F.B., Florida, Young et al (1975) reported levels of TCDD in beach mice, Peromyscus polionotus. The mice were collected in 1974 from areas which had significant levels (10 - 710 ppt) of TCDD in the soil. The mice had accumulated 540 - 1300 ppt of TCDD in their liver, while their pelts were contaminated with 130 - 140 ppt of TCDD.

In this same test area with the high levels of TCDD in the soil, reptiles had significant levels of TCDD in their visceral mass (360 ppt) and trunks (370 ppt) (Young et al, 1975).

In 1977 the Michigan State Department of Agriculture completed a survey for polybrominated biphenyls (PBBs) and other toxic contaminants in 1100 cattle herds. Cattle from nine of these herds, which were further identified as dairy herds, had detectable levels of PCP in tissue, from 2 ppb to 12 ppm. Dioxins were detected in PCP contaminated cattle from two of these nine herds (Conklin and Fox, 1978).

A news report (Anonymous, 1977d) stated that the U.S.E.P.A. had reported that OCDD had been found at levels of 16 ppm and 50 ppm in two liver samples from Michigan cows contaminated with PCP.

In one dairy herd in which PCP residues had been identified, octa-, hepta-, and hexa-dioxins were detected in the ppb and ppt range in fat and liver tissue, respectively (Conklin and Fo , 1978; Hoeting, 1977). Further to this a report in the Federal Register (U.S.E.P.A. 1978b stated:

> "As a result of this incident, the Animal and Plant Health Inspection Service of the Department of Agriculture (USDA) instituted a nationwide survey of beef fat and liver for the presence of the hexa- and octachlorodioxins found in PCP. In the first group of 238 beef samples collected in seventeen States, 70 (29.4%) showed positive levels using low resolution mass spectrometry. Of these 70

samples, 4 had been confirmed using high resolution mass spectrometry. Detection levels were in fractional nanograms/gram for both he·a- and octa-chlorodiben o-p-dioxins. The determination of the significance of these residues awaits the completion of the survey and its subsequent statistical and methodological analysis."

Shadoff and Hummel (1978) stated that the levels of 2,3,7,8-TTCD detected in the small percentage of the beef fat samples collected in the U.S.E.P.A. study from the U.S. Midwest were in the range of 20 - 60 ppt.

Europe

Bonaccorsi et al (1978) extracted information from a government report on the Seveso incident (Sect. 6.3, App. 3, Sect. 3.3) and tabulated the levels of TCDD found in rabbit liver from three contaminated zones and the surrounding areas. The soil concentrations of TCDD used to define the three zones and the amounts of TCDD in the liver tissue of rabbits from these zones are shown in Table 51.

TABLE 51 TETRACHLORODIBENZO-p-DIOXIN (TCDD) LEVELS (ng/g) FOUND IN RABBIT LIVER FROM CONTAMINATED ZONES AND SURROUNDING AREAS, SEVESO, ITALY, 1976. (Bonaccorsi et al, 1978).

Zone	TCDD levels (ug/m^2) in soil	ng TCDD/g liver			
		Range	Median + SE (standard error)	No. of samples	% Positive[a]
A	n.v. - 5477	3.7 - 633	56.3 + 15.7	67	97
B	n.v. - 43.8	7.0 - 383	53.0 ± 31.5	19	84
R	n.v. - 5	0.27 - 460	5.6 ± 7.9	137	81
S		0.32 - 55	4.0 ± 7.8	86	13

a TCDD levels higher than 0.25 ng/g
R zone of Risk (zone outside of main area of fallout)
S surrounding areas
n.v. 0.75 $\mu g/m^2$

Recently, Fanelli et al (1980) reported levels of 2,3,7,8-TCDD found in animals from a contaminated zone at Seveso. The animals were collected 2 years after the incident from a 6000 m^2 area where levels of 2,3,7,8-TCDD in the top 7 cm of soil varied from 0.010 to 12 ppb. The mean value of 23 determinations was 3.5 ppb. Fanelli et

al (1980) stated that, "All field mice were found positive for TCDD with whole body concentrations ranging from 0.070 to 49 ppb (mean value 4.5 ppb; median value 1.2 ppb)."

6.5.3 Humans. No published information was located on presence of PCDDs and PCDFs in humans in Canada.

In the U.S., the E.P.A. reported dioxin blood levels of 7 and 31 ppt in samples from workers exposed to o-CP at a railroad tank car spill site in Sturgeon, Mo (Sect. 6.3) (Anonymous, 1979e). The E.P.A. noted that although the dioxins were not identified as to isomers, this was the first time blood levels of dioxins had been found.

As reported by Rappe et al (1979c) liver samples from two deceased Yusho patients in Japan were analyzed for PCDFs as was a sample of PCB contaminated rice oil. Using recently available standards, 31 PCDFs were identified in the Yusho rice oil and 14 of these PCDFs were in the liver samples. Concentrations of PCDFs in the liver samples varied from a trace to high. The samples had been taken about one year after initial exposure of the patients to the PCB contaminated rice oil.

6.6 Food

Canada

In Canada, there are no published documents with information on presence or levels of PCDDs and PCDFs in food for human consumption, or in feed for livestock. In 1978 there were only two to three laboratories in Canada which had the capabilities, both in instrumentation and developed methodology, to analyze for CDDs and CDFs at the detection levels (ppt) required (App. 2, Sect. 2.2).

The March 23, 1977, edition of Pesticide and Toxic Chemical News (Anonymous, 1977b) noted that Agriculture Canada had announced they had cleared milk for human consumption which had been previously quarantined because of possible PCP and CDD contamination. The milk had been extensively tested and no CDDs were found. Agriculture Canada had tested the milk after finding that cattle feed had been contaminated with PCP in shipment (Sect. 5.2.7).

United States

Reports carried in Pesticide and Toxic Chemical News (Anonymous, 1977a, 1977b, and 1977c) presented information on PCP and dioxin contamination of milk in Michigan dairy herds. Following quarantine and continued monitoring for the contaminants in the milk of affected herds, milk from six of seven herds was eventually

released for consumption when it was proved to be free from contamination. A sample of milk from the seventh herd had .09 ppm PCP and trace amounts of dioxin.

The possible transfer of TCDD from 2,4,5-T to cows milk was investigated by Mahle et al (1977). They found that surveillance samples of milk from Oklahoma, Arkansas and Missouri, collected from cows grazing on pasture or rangeland treated with normal applications of 2,4,5-T, did not contain TCDD when analyzed by gc-ms at a detection limit of 1 ppt.

Dow's Task Force Report (Dow Chemical, U.S.A., 1978) on possible sources for CDDs included information on analyses for CDDs in samples of charcoal grilled steaks (Table 52) and cigarette smoke (Table 53).

TABLE 52 CHLORINATED DIOXIN CONTENT OF EXTRACTS FROM CHARCOAL GRILLED STEAKS (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, pg/g(ppt)							
	TCDD							
Sample	Other Isomers	2,3,7,8-	HCDD GC-MS	EC	HpCDD GC-MS	EC	OCDD GC-MS	EC
Blank	ND(18)	ND(14)	ND(15)	ND(1)	ND(10)	4(1)	ND(20)	6(2)
Medium-Rare	ND(16)	ND(5)	ND(15)	ND(1)	ND(19)	3(1)	ND(20)	5(2)
Well-Done	ND(28)	ND(7)	ND(15)	ND(1)	ND(11)	6(1)	ND(22)	12(2)
Over-Done	ND(21)	ND(5)	ND(17)	ND(1)	ND(16)	7(1)	29(29)	16(2)

TABLE 53 CHLORINATED DIOXIN CONTENT OF THE PARTICLES IN CIGARETTE SMOKE (Dow Chemical, U.S.A., 1978)

	Apparent Dioxins, picogram/cigarette				
	TCDD				
Location of Purchase and Test	Other Isomers	2,3,7,8-	HCDD	HpCDD	OCDD
Urban 1	ND(10)	ND(10)	8.0	8.5	50
Urban 2	ND(7)	ND(5)	4.2	9.0	18

At the conclusion of their report (Dow Chemical, U.S.A., 1978), and as noted in Sect. 6.2, Dow's Chlorinated Dioxin Task Force proposed that CDDs might be ubiquitous in all combustion processes and that they may have been with us since the advent of fire. This proposal has not been universally accepted (Anonymous, 1979f). As noted by Rawls (1979), some government officials have stated that the source of the CDDs may be the organochlorine precursors found at the Dow plant. Further, in a Pesticide and Toxic Chemical News report dated February 21, 1979, (Anonymous, 1979d) the U.S.E.P.A. is quoted as saying that "Controlled combustion studies are needed to evaluate Dow's hypothesis that PCDD synthesis occurs in most combustion processes as well as to indicate the scope of any future monitoring effort."

Europe

Following the explosion at the chemical plant which produced 2,4,5-TCP at Seveso, Italy, in 1976 (Sect. 6.3, and 6.5.2, and App. 3, Sect. 3.3), samples of milk were taken from cows fed TCDD contaminated fodder. Analysis was delayed until the sophisticated procedures for the analysis were available. Results of the analysis as reported by Bonaccorsi et al (1978) indicated residues of up to 7 µg TCDD/L of milk.

7 CURRENT CANADIAN RESEARCH

Projects, with reference to chlorinated phenols, currently underway or projected to have "start-ups" before the end of 1979, including those projects for which progress reports or final reports were unavailable, and some additional proposed projects for the 1980-81 federal fiscal year, include the following:

1) A two-part study on the fate of PCP in aquatic systems: a) Fate of PCP in a model ecosystem. Identify PCP accumulation in biota, PCP degradation products and pathways arising from biological, chemical, or photochemical processes. (App. 8, Sect. 8.1). b) Fate of PCP in water, sediment, and biota of the Bay of Quinte, Lake Ontario (Sect. 5.1.1).
2) Determination of dioxin levels in products and environmental samples in the vicinity of two wood preserving operations using pentachlorophenol.
3) Investigation of the effects upon the eastern oyster Crassostrea virginica of chronic exposure to sub-acute levels of pentachlorophenol and byproducts.
4) Détermination des sources de BPC et de chlorophénols dans des effluents de certains secteurs industriels au Québec.
5) CEPEX controlled experimental study of the behaviour, pathways, residence time, and toxicity of pentachlorophenol in the marine environment. (The project to be undertaken in British Columbia.)
6) Short-term sublethal toxicity tests to assess safe levels of environmental contaminants. The test organism is fingerling rainbow trout, Salmo gairdneri. One of the substances in the test program is PCP. (B.C. Research).
7) Projects related to CPs in livestock litter in Ontario (Sect. 5.2.2), include:
   a) Effect of PCP on immunosuppression in chickens.
   b) Analysis of tissues taken from chickens raised on CP contaminated litter (Table 30).
   c) Analysis of chickens fed PCP to study immunosuppression.
   d) Survey of randomly collected poultry meats in Ontario.
8) In British Columbia, a project for development of a code of good practice for operating chlorophenol wood protection facilities and for storing and transporting chlorophenol fungicides.

9) The environmental effects of chlorophenols and other contaminants on stream biology. (Nat. Water Res. Inst.)

10) Current uses and health effects of pentachloro- and tetrachlorophenols. (Health and Welfare Canada)

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APPENDIX 1

APPENDIX 1

1 CHEMISTRY OF THE CHLOROPHENOLS, CHLORODIBENZO -p-DIOXINS, CHLORODIBENZOFURANS, AND OTHER IMPURITIES

The information in this section covers the synthesis of CPs, a brief summary statement as to their chemical reactions, and data covering their chemical and physical properties. Similar data, although limited, are given for the impurities commonly found in the chlorophenols.

1.1 Chlorophenols

1.1.1 Synthesis of Chlorophenols. The commercial processes for the production of CPs were presented in Section 2.1 along with the general reaction chemistry (Fig. 1). Further details on the reactions in the chlorination of phenols are shown in Fig. A1-1, adapted from Firestone (1977).

1.1.2 Chemical and Physical Properties. The chemical and physical properties of the CPs are closely correlated with their behaviour, biological activity, and persistence in the environment. The major physical properties for the CPs are presented in Table A1-1.

The biological activity of the CPs increases with the degree of chlorine substitution (Blackman et al, 1955b). With the exception of o-CP, all the CPs are solid at room temperature. As the degree of chlorination increases so does the melting point. (Prior to the advent of gc analysis, melting points were often used to determine purity of compounds). Similarly, as the number of chlorine substitutions increases, the solubility decreases, as do the pK values. Although PCP is only slightly soluble in water, the sodium salt of PCP is very soluble in water, i.e. 33% w/w at 25°C (Table A1-2). To obtain the same degree of solubility for PCP, organic solvents are required, although there was limited solubility of PCP in CCl_4 and in paraffinic petroleum oils (Table A1-3) (Firestone, 1977). The volatility of pure PCP in steam was 0.167 g/100 g of steam at 100°C. (Monsanto Europe S.A., 1976).

1.1.3 Chemical Reactions. The following information on chemical reactions of CPs has been abstracted from Doedens (1967).

Generally the CPs react very much the same as phenol itself. The methyl, ethyl, propyl, and butyl ethers of all of the CPs are readily available for the reaction of the sodium chlorophenoxides with the corresponding alkyl halides. Of commercial

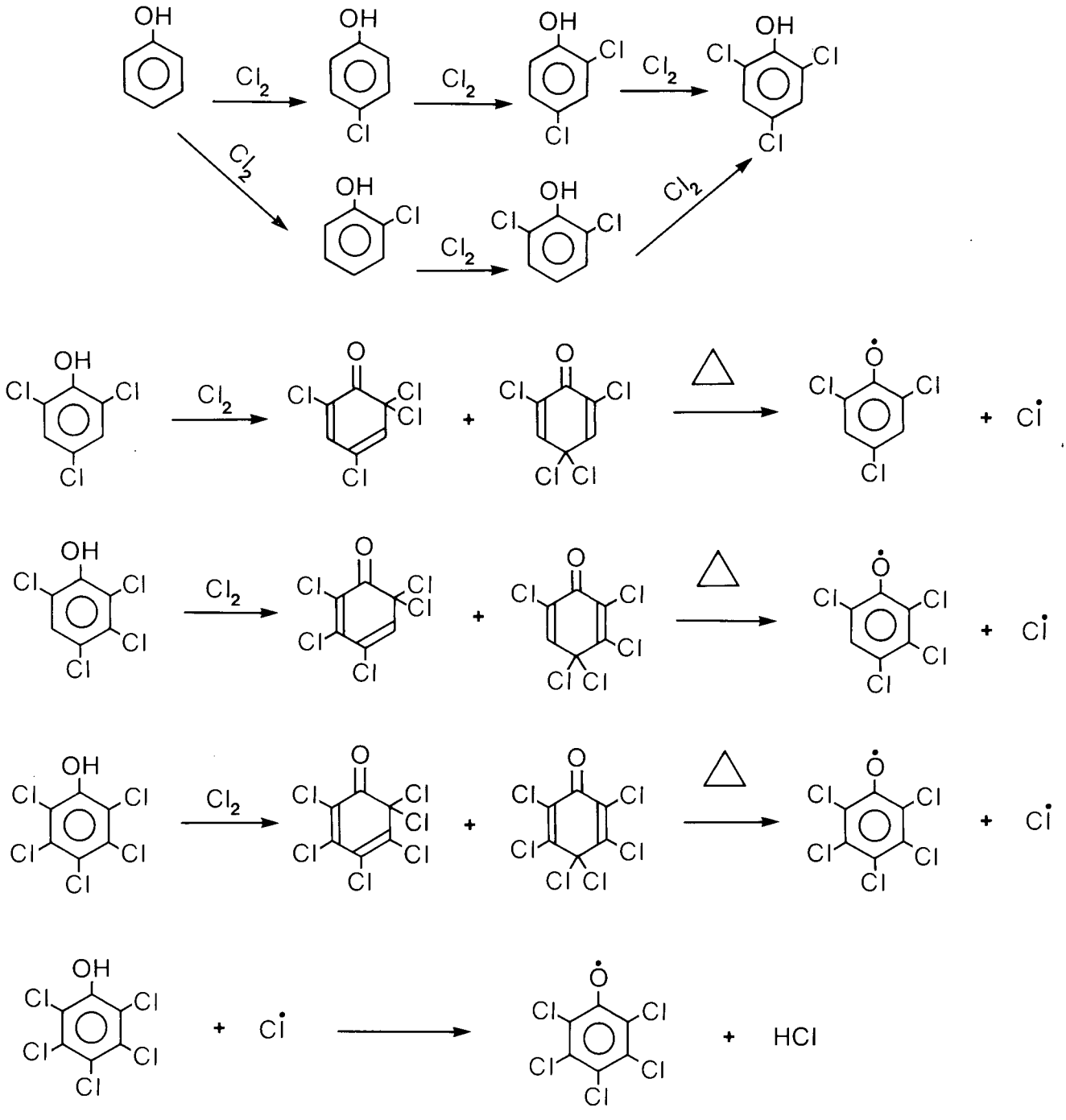

FIGURE A1-1 REACTIONS IN CHLORINATION OF PHENOL
(Firestone, 1977)

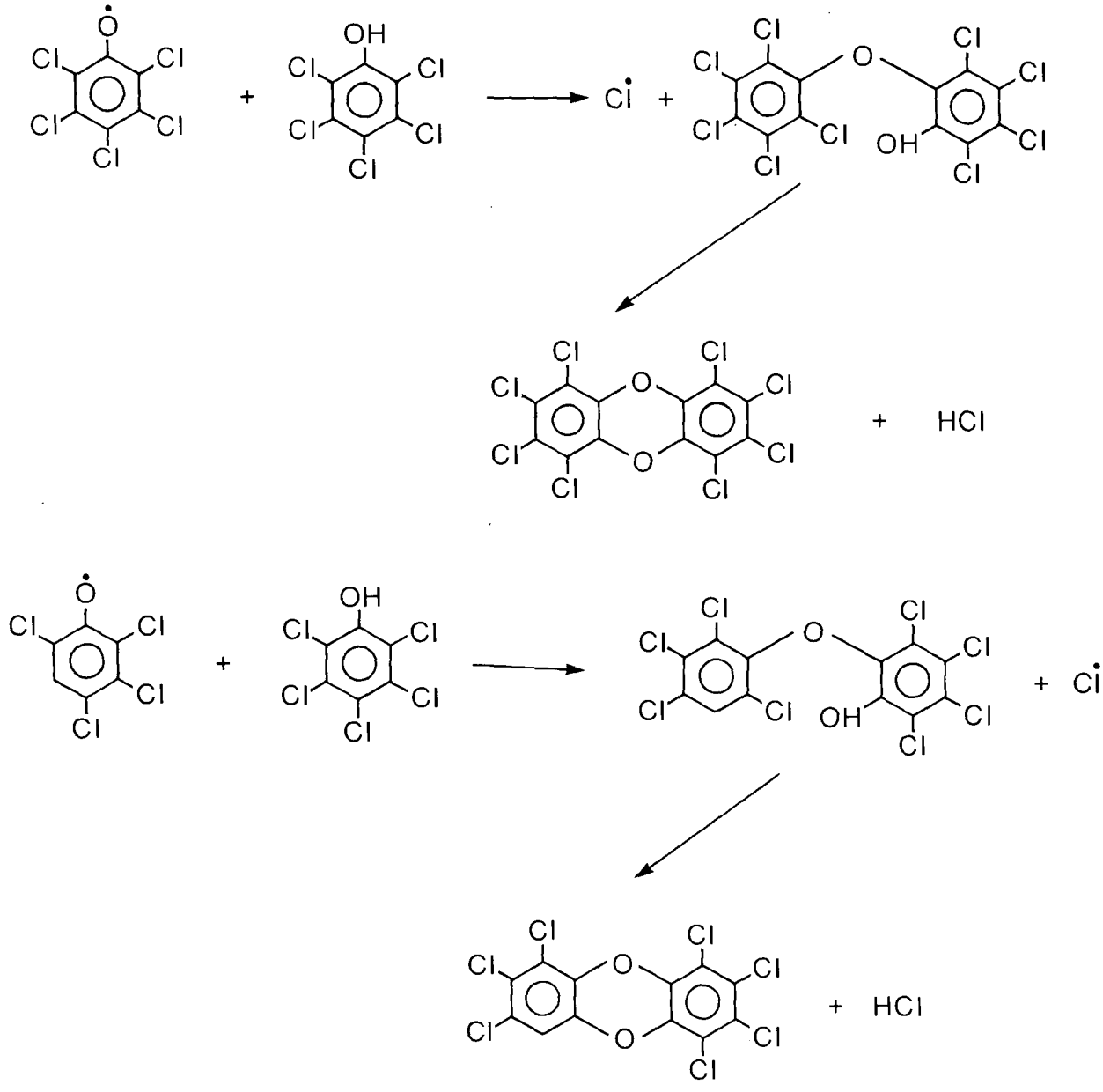

FIGURE A1-1 (Cont'd)

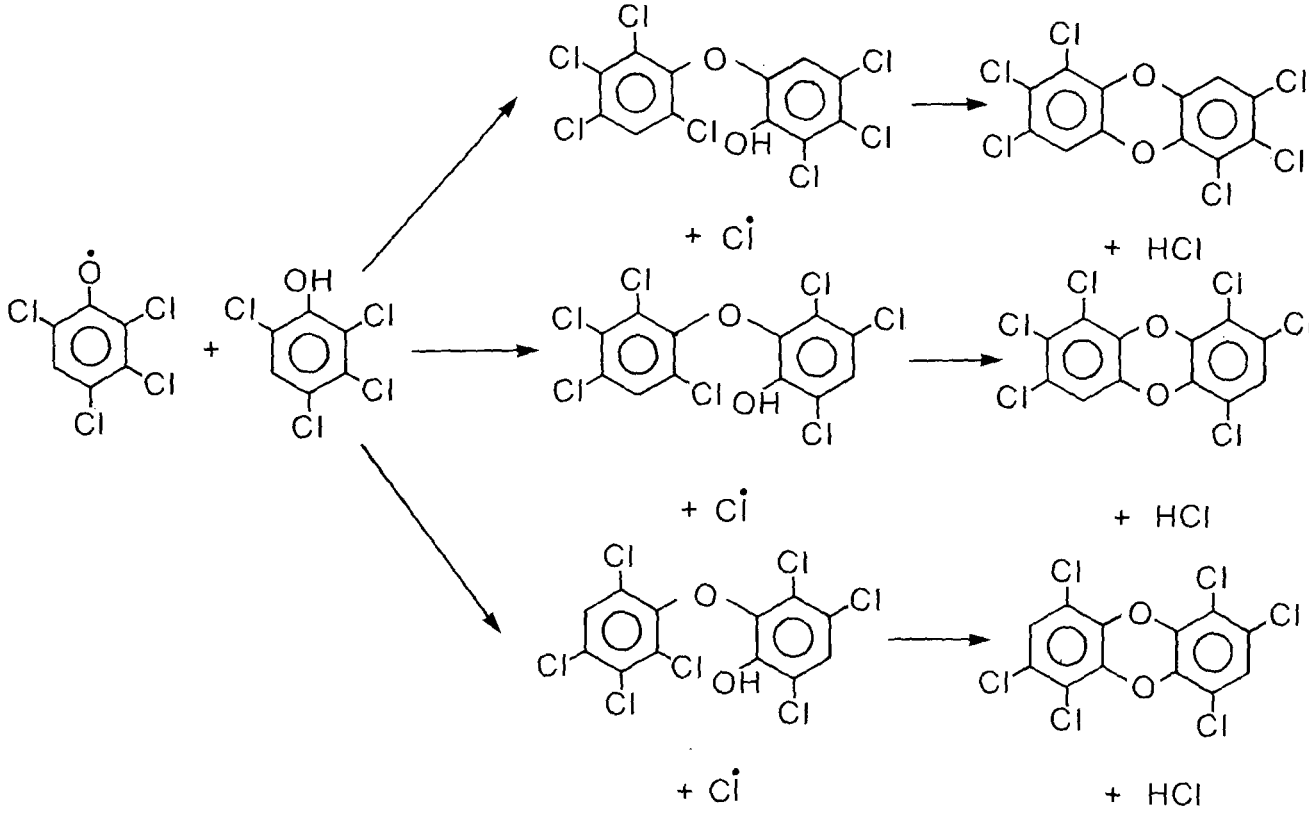

FIGURE A1-1 (Cont'd)

TABLE A1-1 PHYSICAL PROPERTIES OF CHLOROPHENOLS

CAS No.	Compound	Commercial utility[b]	Formula	Molecular Weight[a]	Boiling point[a] (760 mm or as stated), °C	Melting point[a] (°C)	Dissociation constant[b] at 25 °C, Ka
95578	2-CP	limited	C_6H_5ClO	128.56	174.9	9.0	3.2×10^{-9}
108430	3-CP	limited	"	"	214	33	1.4×10^{-9}
106489	4-CP	yes	"	"	219.75	43.2-43.7	6.6×10^{-10}
576249	2,3-DCP	No	"	"	206[b]	57-59	3.6×10^{-7}
120832	2,4-DCP	Yes	$C_6H_4Cl_2O$	163.00	210	45	2.1×10^{-8}
583788	2,5-DCP	No	"	"	211 (744)	59	4.5×10^{-7}
87650	2,6-DCP	No	"	"	219-220 (740)	68-69	1.6×10^{-7}
95772	3,4-DCP	No	"	"	253.5 (767)	68	4.1×10^{-8}
591355	3,5-DCP	No	"	"	233 (757)	68	1.2×10^{-7}
15950660	2,3,4-TCP	No	$C_6H_3Cl_3O$	197.45	sublimes	83.5	2.2×10^{-8}
933788	2,3,5-TCP	No	"	"	248.5-249.5 (250)	62	4.3×10^{-8}
933755	2,3,6-TCP	No	"	"	272[b]	58	7.4×10^{-8}
95954	2,4,5-TCP	Yes	"	"	sublimes (275[b])	68-70.5	3.7×10^{-8}
88062	2,4,6-TCP	Yes	"	"	246	69.5	3.8×10^{-8}
609198	3,4,5-TCP	No	"	"	271-7 (746)	101	1.8×10^{-8}
4901513	2,3,4,5-TTCP	No	$C_6H_2Cl_4O$	231.98	sublimes	116-117	1.1×10^{-7}
58902	2,3,4,6-TTCP	Yes	"	"	150 (15)	70	4.2×10^{-6}
935955	2,3,5,6-TTCP	No	"	"	-	115	3.3×10^{-6}
87865	PCP	Yes	C_6HCl_5O	266.34	309-310 (754)	191	1.2×10^{-5}
131522	NaPCP	Yes	C_6Cl_5ONa	288.36			

[a] Weast, R.C. 1974
[b] Doedens, J.D. 1967
[c] Pearce, P.J., and R.J.J.Simpkins. 1968
[d] Farquharson, M.E., et al 1958
[e] Blackman, G.E., et al 1955b.
[f] Density is relative to water, the superscript indicates the temperature of the liquid and the subscript the temperature of the water to which the the density is referred.
[g] Sax, N.I. 1975.
[h] Arsenault, R.D. 1976.
[i] Dobbs, A.J., and C. Grant. 1980

TABLE A1-1 PHYSICAL PROPERTIES OF CHLOROPHENOLS (Cont'd)

Compound	pK[c,e]	pK[d]	Water solubility[e] (pH 5.1, 25°C) (moles/L)	Density[a,f]	Vapor Pressure[g] @ °C	Flash Pt.[g] °C	Appearance
2-CP	8.48	8.65		$1.2634^{20}/4$	1mm @ 12.1 C	63.9	Light amber liquid
3-CP	9.08	9.12	2.1×10^{-1}	1.268^{25}	1mm @ 44.2 C		Crystals
4-CP	9.42	9.37		$1.2651^{30}/4$	1mm @ 49.8 C	121.1	Needle-like, white to straw colored crystals
2,3-DCP	7.70						
2,4-DCP	7.85	7.85	3.8×10^{-2}		1 mm @ 76.5 C	62	Colorless needles, or yellow solid
2,5-DCP	7.51						
2,6-DCP	6.79	6.91					
3,4-DCP	8.59						
3,5-DCP	8.19						
2,3,6-TCP		5.98					
2,4,5-TCP	7.0	7.07	4.8×10^{-3}		1mm @ 72 C		Colorless needles, or grey flakes
2,4,6-TCP	6.1	6.62	2.2×10^{-3}		1mm @ 53.0 C	113.9	Colorless crystals
3,4,5-TCP		7.83					
2,3,4,6-TTCP			7.9×10^{-4}		1mm @ 100.0 C		Light brown mass
2,3,5,6-TTCP	5.3						
PCP	4.8	5.00	3.6×10^{5}	$1.978^{22}/4$	3.2×10^{-4}mm @ 30C[h] 40mm @ 211.2 C 5.0×10^{-6}mm @ 19°C[i]		Dark colored flakes and sublimed needle crystals

[a] Weast, R.C. 1974
[b] Doedens, J.D. 1967
[c] Pearce, P.J., and R.J.J.Simpkins. 1968
[d] Farquharson, M.E., et al 1958
[e] Blackman, G.E., et al 1955b.
[f] Density is relative to water, the superscript indicates the temperature of the liquid and the subscript the temperature of the water to which the the density is referred.
[g] Sax, N.I. 1975.
[h] Arsenault, R.D. 1976.
[i] Dobbs, A.J., and C. Grant. 1980

TABLE A1-2 SOLUBILITY OF PENTACHLOROPHENOL (PCP) AND SODIUM PENTACHLOROPHENATE (NaPCP) IN WATER (Monsanto Europe SA, 1976; Dow Chemical Co., 1976; Firestone, 1977)

Temp. °C	Solubility PCP (pure) (ppm)	NaPCP (Commercial) (g/100 g)
0	5	
5		19
15	12	30
25		33
30	20	
50	35	
60		37
70		39

TABLE A1-3 SOLUBILITY OF PENTACHLOROPHENOL (PCP) IN VARIOUS ORGANIC SOLVENTS (Monsanto Europe S.A., 1976)

Solvent	% Pentachlorophenol on Weight of Solution at Indicated Temperature (°C)			
	0	20	30	60
Methanol	40.5	57.0	65.5	77.5
Diacetone alcohol	39.5	56.5	62.5	73.5
Ethanol (100%)	46.0	53.0	56.5	67.0
Diethyl ether	-	52.9	60.3	-
Ethanol (95%)	39.0	47.5	52.0	65.5
Pine Oil	24.5	32.0	35.5	46.5
Diethylene glycol	-	27.5	37.5	-
Cellosolve	8.0	27.0	37.5	-
Acetone	-	21.5	33.4	-
Xylene	8.5	14.0	17.5	34.0
Dioxan	-	11.5	16.0	37.5
Toluene	6.0	11.5	15.0	31.0
Benzene	-	11.0	14.0	31.5
Orthodichlorobenzene	5.5	8.5	11.5	26.0
Ethyl benzene	-	8.5	11.5	25.0
Dipentene	-	8.4	10.3	-
Ethylene glycol	-	6.0	11.5	38.5
2-chloro-o-phenylphenol	-	6.0	9.1	-
Turpentine	-	3.0	4.4	-
Carbon disulphide	-	3.0	4.3	-
Carbon tetrachloride	-	2.0	3.1	-

importance is the reaction of the sodium chlorophenoxides with -halo aliphatic acids (e.g. the reaction of 2,4-dichlorophenoxide with chloracetic acid leads to 2,4-dichlorophenoxy acetic acid (2,4-D)). Other reactions listed for the CPs by Doedens (1967) include:

1) formation of sulfonates by the reaction of sodium salts of CP with aromatic sulfonyl chlorides;
2) substitution reactions (e.g. nitration, alkylation, and acetylation);
3) condensation of the lower substituted CPs with formaldehyde to form phenolic resins;
4) formation of the mono-, di-, and triphosphate esters by the reaction of CPs with phosphorus oxychloride;
5) formation of CP salts through reaction with amines.

1.2 Chlorodibenzo-p-dioxins, Chlorodibenzofurans, and Other Impurities

1.2.1 Formation of Chlorodibenzo-p-dioxins and Chlorodibenzofurans during Commercial Synthesis of Chlorophenols. The information that follows in this Section was taken directly from Firestone (1977) and was reported again by the U.S.E.P.A. Environmental Health Advisory Committee (United States, Environ. Prot. Agency, 1978a).

Formation of Chlorodioxins and Chlorofurans during Commercial Synthesis of PCP

> "Chlorodioxins may be prepared in condensation reactions from ortho-substituted chlorophenoxy radicals (Kulka, 1961) or anions (Pohland and Yang, 1972). According to Vogel (Monsanto Industrial Chemicals Co., St. Louis, MO, private communication, 1977), dioxin formation occurs during commercial synthesis of PCP via a series of reactions involving phenoxy radicals. Phenoxy radicals are produced from decomposition of polychlorocyclohexadienone produced by overchlorination of tri-, tetra-, or pentachlorophenol. The phenoxy radical (an electrophile) attacks electronegative sites (ortho or para positions) on a polychlorophenol molecule to form phenoxyphenols which undergo further reaction to form chlorodioxins.
>
> "The decomposition of tri-, tetra-, or pentachlorophenol can also be catalyzed by chlorine (the chlorine radical is the initiator). The tetrachlorophenol present in commercial reaction mixtures (very little trichlorophenol is present) serves as a substrate for chlorine radicals, limiting the chain reaction with PCP molecules, which accelerates PCP decomposition. The chlorination is

normally stopped when 3-7% tetrachlorophenol remains. Further chlorination results in increased decomposition (see reactions in Figure A1-1).

"Rearrangement via a spirocyclic anion (Smiles rearrangement) can yield additional isomers (Gray et al., 1975). Highly alkaline conditions are required for efficient operation of the Smiles rearrangement since the reaction involves rapid equilibration of the anion forms of a phenoxyphenol through a spirocyclic intermediate. This is illustrated by the formation of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexachlorodioxin from 2,3,4,6-tetrachlorophenol. (Figure A1-2).

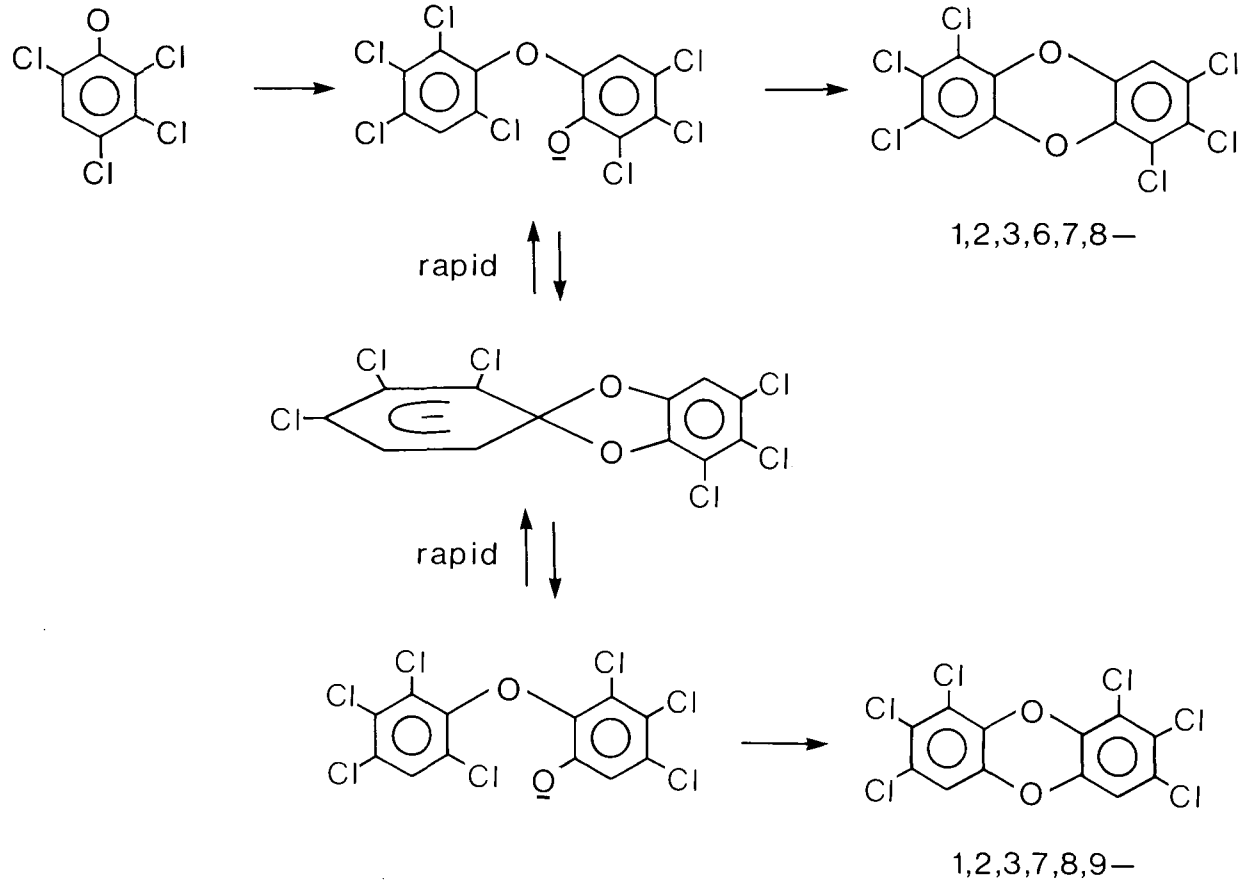

FIGURE A1-2 FORMATION OF 1,2,3,6,7,8- and 1,2,3,7,8,9- HEXACHLORODIOXIN from 2,3,4,6-TETRACHLOROPHENOL (Firestone, 1977)

"In the manufacture of PCP, a considerable amount of hydrochloric acid is present, so that this type of rearrangement is unlikely. Dioxin congeners formed by normal and spirocyclic rearrangement vs. the levels of individual dioxin found in PCP (Vogel, private communication, 1977) are shown in Table A1-4. The hexachlorodioxins found agree with those predicted to form without Smiles rearrangement.

TABLE A1-4 DIOXIN CONGENERS IN COMMERCIAL PCP (Firestone, 1977)

No Smiles rearrangement	With Smiles rearrangement	Relative % isomers found	PCP, ppm[a]
1,3,6,8 (100%)	1,3,6,8 (25%) 1,3,7,9 (75%)	none	ND
1,2,4,7,9 (75%)	1,2,4,7,9 (31.25%)		
1,2,3,7,9 (25%)	1,2,3,7,9 (25%) 1,2,4,6,8 (43.75%)	none	ND
1,2,3,6,8,9 (50%) 1,2,3,6,7,8 (25%)	1,2,3,6,7,9 (31.25%) 1,2,3,6,8,9 (18.75%)	40-50	
1,2,4,6,7,9 (25%)	1,2,4,6,7,9 (12.5%) 1,2,4,6,8,9 (12.5%) 1,2,3,7,8,9 (18.75%) 1,2,3,6,7,8 (6.25%)	20-40 trace 20-40	ca 15
1,2,3,4,6,7,9 (75%)	1,2,3,4,6,7,9 (75%)	ca 60	ca 200
1,2,3,4,6,7,8 (25%)	1,2,3,4,6,7,8 (25%)	ca 40	
1,2,3,4,6,7,8,9 (100%)	1,2,3,4,6,7,8,9 (100%)	100	ca 1000

[a]PCP producers' composite sample.

"Little information is available on the formation of dibenzofurans during PCP production. Formation of dibenzofurans can be explained by the production of polychlorodiphenyl ether intermediates (Kulka, 1961; Plimmer, 1973; Arsenault, 1976) which can lose chlorine to yield dibenzofuran." (Figure A1-3). "Cleavage of the polychlorodiphenyl ether in the presence of hydrochloric acid yields PCP and hexachlorobenzene." (Firestone, 1977).

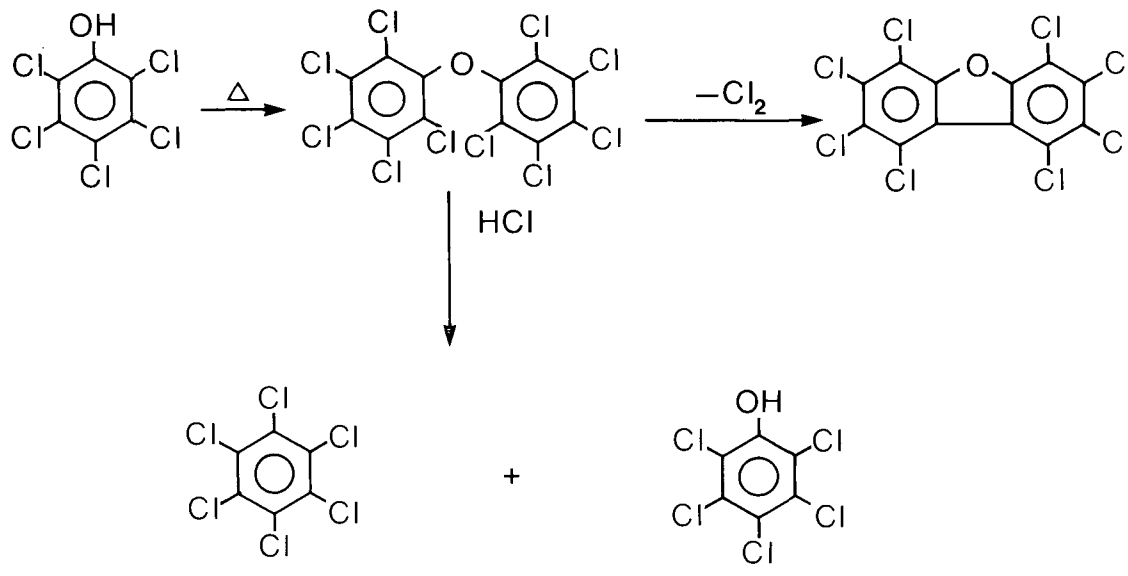

FIGURE A1-3 SUGGESTED REACTIONS FOR FORMATION OF DIBENZOFURANS, PENTACHLOROPHENOL, AND HEXACHLOROBENZENE FROM POLYCHLOROBIPHENYL ETHER INTERMEDIATES (Firestone, 1977)

"Various free radical reactions might also yield a number of biphenyl compounds." (Figure A1-4).

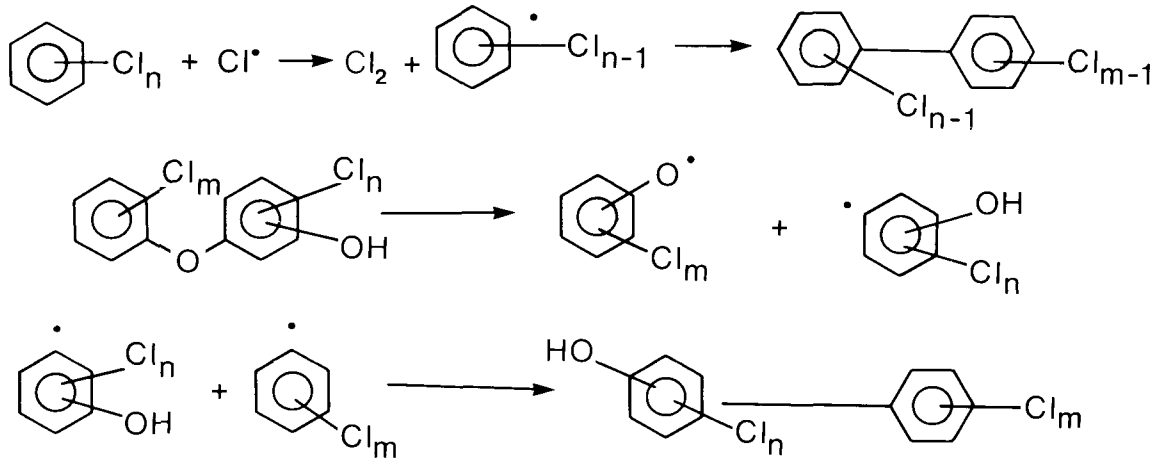

FIGURE A1-4 SUGGESTED REACTIONS FOR FORMATION OF BIPHENYL COMPOUNDS (Firestone, 1977)

Another possible route for the formation of PCDFs from CPs is through an intermediate compound, o-dihydroxy-PCB (Rappe et al, 1978a).

> "MS data obtained from analysis of contaminants in PCPs (Firestone et al., 1972) suggested that polychlorohydroxybiphenyls were present in these products." (Firestone, 1977).

1.2.2 Preparation of Chlorodibenzo-p-dioxins and Chlorodibenzofurans in the Laboratory. Studies on the toxicology of the PCDDs have required gram quantities of pure material. Aniline (1973) reported on the preparation of several PCDDs as follows:

> "Chlorinated dibenzo-p-dioxins were prepared on the gram scale for use as toxicological standards. 2,7-Dichlorodibenzo-p-dioxin was prepared by catalytic condensation of potassium 2-bromo-4-chlorophenate in 70% yield. Thermal condensation of the potassium salt of 2,4,4'-trichloro-2'-hydroxydiphenyl ether gave a mixture of the 2,8- and 2,7-dichlorodibenzo-p-dioxins which were separated by fractional recrystallization. 2,3,7,8-Tetrachlorodibenzo-p-dioxin of 99.9+% purity was prepared by catalytic condensation of potassium 2,4,5-trichlorophenate. An isomeric mixture of hexachlorodibenzo-p-dioxins was prepared by pyrolytic condensation of sodium 2,3,4,6-tetrachlorophenate. Chlorination of pentachlorophenol (containing <0.07% tetrachlorophenol) in trichlorobenzene gave octachlorodibenzo-p-dioxin in 80% yield contaminated by 5-15% heptachlorodibenzo-p-dioxin. Oxidative methods were used to produce octachlorodibenzo-p-dioxin at 99.9% purity."

Kende and DeCamp (1975) demonstrated Smiles rearrangement in the synthesis of a PCDD. The two compounds, 1,2,3,7,8,9-HCDD and 1,2,3,6,7,8-HCDD, which had been directly implicated with chick edema disease were synthesized using, as key intermediates, functionalized hexachlorodiphenyl ethers.

Grey et al (1976) prepared a number of TriCDDs through HpCDDs by the condensation of chloro- substituted catechols with chloro- substituted o-chloronitrobenzenes.

Nilsson et al (1978) listed the synthetic routes most frequently used for the synthesis of PCDDs and PCDFs as follows:

1. Methods for the synthesis of PCDDs
   a) Chlorination of dioxins
   b) From chlorocatechols and chloronitrobenzenes

c) Pyrolysis of chlorophenate

d) From chlorinated 2-phenoxyphenols

2. Methods for the synthesis of PCDFs

a) Chlorination of dibenzofuran

b) Diazotisation of o-NH_2-chlorodiphenyl ether

c) From o,o'-diphenyldiols

d) From polychlorinated diphenylethers

Recently, Nestrick et al (1979) reported the synthesis and identification of the 22 TCDD isomers. They reported that by controlled flow pyrolysis conditions, various combinations of DCP, TCP, and TTCP were reacted to form TCDDs. The reaction products were separated by high performance liquid chromatography (HPLC) and were characterized by gc-ms.

1.2.3 Chemical and Physical Properties. Properties of various CDDs, are given in Table A1-5, as excerpted from Firestone (1977). The solubility of several CDs in various solvents are presented in Table A1-6 (Firestone, 1977).

The chemical and physical properties of the CDFs (Table A1-7) are even less well known than those of the CDDs. The evidence to date indicates that the PCDDs and PCDFs, which are two series of tricyclic aromatic compounds, exhibit similar physical, chemical, and biological properties (Rappe et al, 1979).

The U.S. E.P.A. Environmental Health Advisory Committee (1978a) had estimated vapor density and rate of evaporation for the CDDs (Table A1-8) and CDFs (Table A1-9). They suggested that the estimated rate of volatilization (Q) may be overestimated when dealing with a compound such as PCP which has a high adsorptive force. They also calculated the rate of evaporation for PCP from a solid surface, rather than its own surface, would be 1.7×10^{-10} $g/cm^2/s$. By comparison, the rate of volatilization of the CDDs was very low, with only a slightly higher rate for the CDFs.

Regarding the chemical and physical properties of the impurities other than the CDDs and CDFs in CPs, Firestone (1977) stated that:

> "Data on the physical and chemical properties of chlorophenoxy phenols are limited. Sundstrom and Hutzinger (1976) prepared several chlorodiphenyl ethers and also cited a number of these compounds prepared via various routes by other workers".

The melting points of some of the chlorodiphenyl ethers are given in Table A1-10.

TABLE A1-5 PROPERTIES OF VARIOUS CHLORODIOXINS (Firestone, 1977)

Chlorodioxin	Mol. wt	MP, °C	"p" value[a]	Est. vapor pressure[b]	Molar refraction[c]	UV max ($CHCl_3$), nm
2,7-Cl_2	253.08	--	0.76	6.0×10^{-6}	--	302
2,3,7-Cl_3	287.53	162	0.86	3.6×10^{-6}	--	305
2,3,7,8-Cl_4	321.87	306	0.51	1.7×10^{-6}	71.4	310
1,2,4,7,8-Cl_5	356.42	206	--	--	72.6	307
1,2,3,7,8-Cl_5	356.42	241	--	--	--	308
1,2,4,6,7,9-Cl_6	390.86	240	0.94[d]	6.6×10^{-7}	--	310
1,2,3,6,8,9-Cl_6	390.86	--	--	--	--	81.1
1,2,3,6,7,8-Cl_6	390.86	285	--	--	--	317
1,2,3,7,8,9-Cl_6	390.86	243	--	--	--	317
1,2,3,4,6,7,9-Cl_7	425.31	--	0.90	3.0×10^{-7}	85.9	--
1,2,3,4,6,7,8-Cl_7	425.31	--	0.90	--	--	--
1,2,3,4,6,7,8,9-Cl_8	459.75	331	0.90	1.8×10^{-7}	90.7	318

[a] "p" value determined for dioxin between hexane and acetonitrile (Beroza and Bowman, 1965).

[b] Vapor pressure estimated from GLC data of Woolson et al. (1972).

[c] Estimated by summing atomic refractions.

[d] "p" value determined for mixture of hexachlorodioxin isomers.

TABLE A1-6 SOLUBILITY (mg/L) OF SEVERAL CHLORODIOXINS IN VARIOUS SOLVENTS[a] (Firestone, 1977)

Solvent	2,3,7,8-TCDD	HCDD[b]	OCDD
Acetone	90	--	5
Anisole	--	2600	1700
Benzene	470	1600	1000
Chloroform	550	--	560
Methanol	10	--	--
Toluene	--	1800	1600
o-Xylene	--	--	3600
Water	0.0002	--	--

[a]Firestone (unpublished data, 1976) observed that 1,2,3,6,7,8-HCDD is considerably less soluble in organic solvents than other HCDD isomers. The solubility of the 1,2,3,6,7,8-isomer in isooctane is about 20 mg/L.

[b]Dow standard 82-A, a mixture of 71% 1,2,3,6,7,8-HCDD and 29% 1,2,3,6,7,9-HCDD and 1,2,3,6,8,9-HCDD.

TABLE A1-7 PROPERTIES OF CHLORINATED DIBENZOFURANS (Firestone, 1977)

Compound	Mol. wt	MP, °C	Vapor pressure[a] (est.) 25°C	Molar refraction[b]	UV max ($CHCl_3$), nm
Dichloro	209.1			60.2	
2,4			7.3×10^{-6}		
3,7			7.0×10^{-6}		
2,8		185[c]	6.8×10^{-6}		
Trichloro	243.5			65.0	
2,4,6		116-117[d]	4.0×10^{-6}		
2,3,8		189-191[d]	3.7×10^{-6}		256,302,313
2,4,7					
2,4,8					
Tetrachloro	278.1			69.8	
1,4,6,8			2.5×10^{-6}		
2,4,6,8		198-200[d]	2.5×10^{-6}		257,294,310,323
2,3,6,8			2.2×10^{-6}		
2,4,6,7			2.1×10^{-6}		
1,2,7,8			2.0×10^{-6}		
2,3,7,8		227-228[d]	2.0×10^{-6}		259,309,316
2,3,6,7			1.9×10^{-6}		
3,4,6,7			1.8×10^{-6}		
Pentachloro	312.6			74.6	
1,3,4,7,8			1.3×10^{-6}		263,272,297,320
1,2,4,7,8		234-235[d]	1.3×10^{-6}		256,266,297
1,2,3,6,7			1.1×10^{-6}		
2,3,4,7,8			1.1×10^{-6}		
Heptachloro	381.6			84.2	
			4.4×10^{-7}		
			3.6×10^{-7}		
			3.0×10^{-7}		
Octachloro	416.1		1.9×10^{-7}	89.0	

[a]From GLC data supplied by D.W. Phillipson, FDA (1977).

[b]Calculated from table of atomic refraction.

[c]Gilman et al. (1934).

[d]Gray et al. (1976b).

TABLE A1-8 ESTIMATED VAPOR DENSITY[a] AND RATE OF EVAPORATION[b] OF CHLORODIOXINS (U.S., E.P.A. 1978a)

Compound	Vapor Density (g/cm^3)	Q ($g/cm^2/s$)
2,7-di	8.2×10^{-11}	6.1×10^{-12}
2,3,7,8-tetra	2.9×10^{-11}	2.0×10^{-12}
Penta	1.7×10^{-11}	1.1×10^{-12}
Hexa	1.4×10^{-11}	8.4×10^{-13}
Hepta	6.9×10^{-12}	4.0×10^{-13}
Octa	4.5×10^{-12}	2.5×10^{-13}

(a) Above own surface, (b) from absorbing surface.

TABLE A1-9 ESTIMATED VAPOR DENSITY[a] AND RATE OF EVAPORATION[b] OF CHLORINATED DIBENZOFURANS (U.S., E.P.A. 1978a)

Compound	Vapor Density (g/cm^3)	Q ($g/cm^2/s$)
2,4-di	8.2×10^{-11}	6.8×10^{-12}
2,4,6-tri	5.2×10^{-11}	4.0×10^{-12}
2,3,7,8-tetra	3.0×10^{-11}	2.1×10^{-12}
1,4,6,8-tetra	3.7×10^{-11}	2.7×10^{-12}
2,3,4,7,8-penta	1.9×10^{-11}	1.3×10^{-12}
1,3,4,7,8-penta	2.2×10^{-11}	1.5×10^{-12}
Octa	4.3×10^{-12}	2.5×10^{-13}

(a) Above own surface, (b) from adsorbing surface.

TABLE A1-10 MELTING POINTS OF CHLORODIPHENYL ETHERS
(Firestone, 1977)

Diphenyl ether	MP, °C
2,4,4'-	51-52
2,3',4,4'-	70
2,3',4,4'-	oil
3,3',4,4'-	oil
2,3,4,5,6-	132-133
2,2'4,4',5-	oil
2,3',4',5'-	65-67
2,2',4,4',5-	oil
2,3',4,4',6-	36
2,2',3,4,5,6,6'-	147-148
Deca-	224-225

APPENDIX 2

APPENDIX 2

2 RESIDUE ANALYSIS FOR CHLOROPHENOLS, CHLORODIBENZO -p-DIOXINS AND CHLORODIBENZOFURANS

This section on residue analysis of CPs, CDDs, and CDFs in environmental samples will not be an in-depth review but will only include methods currently in vogue or as used in Canadian laboratories. In some cases suggested modifications have been noted.

2.1 Chlorophenols

2.1.1 Water. Fox (1978) commented on the analytical methods for determination of PCP as follows:

> "Most published analytical methods for the determination of PCP in trace quantities from environmental samples rely on the electron capture GC analysis of derivatization PCP. The most popular and easiest to make derivative is the methyl ether produced by reaction of the environmental extract with diazomethane. The acetate derivative produced by the aqueous acetylation method of Chau & Coburn (1974) offers substantial advantages however. Most environmental samples contain a variety of non-phenolic organochlorines which may seriously interfere with the GC determination of chlorophenols. Due to the aqueous base extraction step of the acetylation method, these compounds are eliminated before the GC step."
>
> (Note: A description of the method in which CPs are methylated using ethereal diazomethane is detailed in the Environmental Laboratory Manual compiled and published in January 1979 by Environment Canada, EPS Laboratory Services Regional Laboratory in West Vancouver, B.C. The manual notes that the detectable concentration of PCP, 2,3,4,5-, 2,3,4,6-, and 2,3,5,6-TTCP, utilizing ec-gc was 0.01 µg/L (ppb) in natural and waste waters).

The Chau and Coburn (1974) procedure Fox (1978) referred to allowed detection of as little as 0.01 ppb PCP in one litre water. The procedure was summarized by Chau and Coburn (1974):

> "PCP is extracted from the preserved sample with benzene and from the benzene into a potassium carbonate solution. The addition of acetic anhydride

to the aqueous solution produces the acetate derivative of PCP, which is extracted in hexane and analyzed by electron capture gas-liquid chromatography."

Fox (1978) suggested two modifications to the Chau and Coburn (1974) method:

1) Alkaline storage at pH 12 (in 1 L amber bottles) to increase solubility, which in turn decreases adsorption onto surfaces, followed by acidification to pH 1 - 2 immediately prior to analysis.
2) Replace benzene with toluene as the extraction solvent, because of the suspected role of benzene as a carcinogen. Extraction efficiencies of the two solvents are approximately equal.

In a paper read at a symposium held in May 1978 at the Canada Center for Inland Waters, Burlington, Ontario, Sirons and Paik (1978) indicated the usefulness of Amberlite polymeric adsorbents for the extraction of CPs from water samples, irrespective of size of sample. Their system included extraction followed by elution with 0.1 N NaOH in methanol (1:4) mixture plus extraction with chloroform. The $CHCl_3$ extract was evaporated, the CPs were dissolved in iso-octane (containing small amounts of methanol) and quantified using gc equipped with electron capture (polychlorinated) or conductivity (mono and dichloro phenols) detection systems. The detection limit was well below the 0.1 ppb range for the tri-, tetra- and penta-CPs and was dependent on the impurities of the samples. The authors stated the methodology could be extended to include any substrate.

Krijgsman and van de Kamp (1977) explained that in an unmodified gc analysis the separation and quantitation of phenols was difficult because of their properties of high polarity and low vapor pressure. Unless precautions were taken asymetrical peaks were obtained. Derivitization to less polar compounds overcame the tailing effect. Krijgsman and van de Kamp (1977) found that determination of CPs could be accomplished in a single step by use of capillary gas chromatography. Using the method of Chau and Coburn (1974) the chlorophenols were acetylated prior to injection on to a capillary column coated with SE-30. The column was connected to an electron-capture detector, which allowed determination in the nanogram and sub-nanogram ranges. In their study the limit of detection of pentachlorophenol acetate (PCP-acetate) was about 1 pg. They also confirmed that recovery of the extraction and acetylation step of CPs was 80 - 100%. Krijgsman and van de Kamp (1977) commented that:

"Several aspects make the developed method attractive for the determination of chlorophenols in environmental samples: (1) selectivity of the extraction; (2) high separation power of the capillary column; and (3) specificity and sensitivity of the electron-capture detector."

Faas and Moore (1979) reported on methods used to measure PCP in samples from the estuarine environment (App. 2, Sect. 2.1.3). They stated that sea water concentrations of PCP as low as 0.002 ppb could be detected by formation of the amyl diazohydrocarbon derivative; furthermore, they noted that formation of the amyl derivatives of PCP and several related compounds gave GLC separation not possible with the methyl or ethyl derivatives.

2.1.2 Soil. As noted in App. 2, Sect. 2.1.1 the analytical method used for determination of CPs in water will also apply to other substrates such as soil if appropriate extraction and clean-up techniques are used (Sirons and Paik, 1978; Krijgsman and van de Kamp, 1977).

2.1.3 Biological Samples. Rudling (1970) developed the method for analysis of PCP in fish tissue by derivitization of the PCP to PCP-acetate; this method served as the basis for the procedure of Chau and Coburn (1974). Rudling (1970) extracted PCP from an acidified sample with n-hexane and then re-extracted it into a borax solution, which was then acetylated by extracting with n-hexane containing acetic acid anhydride and pyridine. The resulting PCP-acetate was then analyzed by gc with an electron capture detector. Confirmation of PCP in the samples was by glc-ms. Recoveries exceeded 80% from PCP contaminated fish tissue samples of 1 g size. Using this method, Rudling (1970) detected in authentic samples of perch, Perca fluviatilis, 0.15 mg PCP/kg fresh tissue.

Hoben et al (1976d) described a method for determination of PCP in rat plasma, urine, tissue, and in air-aerosol samples. PCP was isolated through extraction from the samples with benzene or hexane after acidification, derivitization, and its subsequent purification through florosil columns. Recoveries were greater than 91%. The lower limit of detectability was 20 ppb. They stated their procedures were particularly suited for exposure experiments where different types of samples with a large variety of PCP concentrations would be encountered.

Farrington and Munday (1976) developed a method for determination of trace amounts (ppb) of CPs in chicken flesh by electron-capture (ec) gas-liquid chromatography (glc). In a summary they stated that solutions of TCPs, TTCPs and PCP were made to

react with 2,4-dinitro-1-fluorobenzene in the presence of pyridine as a catalyst. They noted that the CP derivatives were all resolvable from each other and from co-extractive interferents. Recoveries with spiked samples ranged from 85 - 92%. Levels of 2,3,4,6-TTCP found in chicken flesh ranged from 0.002 to 0.003 mg/kg and levels of PCP from 0.005 to 0.012 mg/kg.

Faas and Moore (1979) described a method for measuring PCP in samples from the estuarine environment. GLC was used to determine PCP residues in marine biota tissues as low as 0.01 ppm by formation of the ethyl diazohydrocarbon derivative, followed by Florosil cleanup.

An analytical method recently described by Lamparski et al (1978) to determine PCP in bovine milk consisted of sulfuric acid digestion, silica gel column chromatography, methylation, alumina column chromatography, and detection with ec-gc. Recoveries using this method were 80%.

Edgerton and Mòseman (1979) developed a gc method for more reliable determination of PCP in urine. By using a hydrolysis procedure, yields as much as 17-fold higher for biologically incorporated PCP were obtained when compared to other current methods. Edgerton and Moseman (1979) briefly summarized the system as follows:

> "After hydrolysis and extraction the sample was reacted with diazomethane to produce the methyl ether of PCP prior to analysis by electron-capture gas chromatography. An acid alumina column clean-up system was developed to remove interferences from the sample extracts and allow detectability of 1 ppb PCP. Average recoveries of greater than 90% were obtained from urine fortified with known amounts of PCP".

A companion, quantitative method was described by Edgerton et al (1979) for determination of CP metabolites of PCP in urine. The method permits a level of 1 ppb as a minimum detection for each metabolite.

2.2 Chlorodibenzo-p-dioxins and Chlorodibenzofurans

In November 1979, there were six Canadian laboratories with identified capabilities for analysis for CDDs and CDFs. Plant Products Division, Agriculture Canada, Ottawa, had an ongoing program for detection and quantification of impurities, including CDDs, in formulated CPs presently registered for use in Canada. Preliminary investigations to determine presence and levels of these compounds in foods had been undertaken by a laboratory of Health and Welfare Canada, Ottawa. Wellington Science

Associates, Inc., Rockwood, Ontario, recently determined levels of CDDs in power transformer fluids.

Other laboratories with CDD analysis capabilities are the Fisheries Research Board Laboratory at St. Andrews Biological Station, St. Andrews, N.B., the Chemical Division Laboratory of the Air Pollution Control Directorate, Ottawa, Ontario, and a laboratory under the direction of Dr. F.W. Karasek at the University of Waterloo, Waterloo, Ont.

The analytical methods used by these laboratories for determination of the CDDs and CDFs were adapted from one of the standard methods reviewed by Firestone (1977), from which the following was extracted.

> "Until recently, EC-GLC has been mainly used for chlorodioxin analysis. But because of the presence of other components, MS has come into use for specific detection and confirmation of chlorodioxins as well as chlorofurans."
>
> "Villanueva et al., (1975) compared 4 methods (Firestone et al., 1972; Jensen and Renberg, 1972; Crummett and Stehl, 1973; Rappe and Nilsson, 1972) for chlorodioxins in PCP. The Crummett and Stehl and Jensen and Renberg methods, which involve extraction of non-acidic material in PCP with ether-hexane (1+1), gave the best recoveries, while the Crummett and Stehl method employing ion exchange to remove the acidic components was simpler than the Jensen and Renberg procedure.
>
> "Crummett and Stehl (1973) employed GLC-MS for the determination of hexa- and octachlorodioxin in PCP. Buser (1975) developed a specific method for analysis of chlorodioxins and chlorofurans in PCP and other chlorophenols. Phenolic compounds were extracted with alkali and the neutral material was chromatographed on a basic alumina mini-column to eliminate polychlorinated benzenes and polychlorodiphenyl ethers. The fraction containing chlorodioxins and chlorofurans was then subjected to mass fragmentographic (GLC-MS) analysis at selected m/e values.
>
> "Buser and Bosshardt (1976) employed the GLC-MS procedure to examine a number of commercial PCP and PCP-Na samples. Chlorodioxins and chlorofurans were detected by mass fragmentography and their presence was confirmed by complete MS analysis. Hexachlorodioxins were recovered from PCP and PCP-Na samples with 80-95% efficiency (0.1-30 ppm) and octachlorodioxins with 95% efficiency (10-30 ppm)."

Firestone (1978) noted that:

"Baughman and Meselson (1973 a,b) described a sensitive procedure for-measuring TCDD in tissue samples, which involved extensive clean-up and analysis by high-resolution MS, using a multi-channel analyser to average successive scans. ^{37}Cl-TCDD was added to each sample before clean-up as a carrier in order to calculate absolute recovery of TCDD. The procedure was used to detect TCDD in biological samples at levels approaching 1 ng/kg."

During the past decade improvements have been made both in methods of analysis for TCDD and also in the quality of 2,4,5-TCP produced, as demonstrated by Firestone (1978) (Table A2-1).

TABLE A2-1 IMPROVEMENTS TO PRODUCT AND ANALYTICAL METHODS FOR 2,4,5-TRICHLOROPHENOL (TCP) AND TETRACHLORODIBENZO-p-DIOXIN (TCDD), RESPECTIVELY. (Firestone, 1978)

			TCDD conc. (ppm)	
Product	Year	Analytical method	Limit of detection	Product specification
2,4,5-TCP	1964	glc	1.0	<1.0
	1970	glc	0.5	<0.5
	1971	glc-ms	0.05	<0.1
	1976	glc-ms	0.0001	0.01[a]

[a]Maximum concentration present in Dow product. (glc = gas-liquid chromatography; ms = mass spectrometry).

Firestone (1978) has noted further developments in analytical techniques for detecting trace amounts of TCDD:

"Buser (1977) recently demonstrated that high-resolution GLC using capillary columns combined with MS is a powerful tool for detecting trace amounts of TCDD in environmental samples. On the other hand, Hunt et al. (1975) developed a low-resolution negative-ion chemical ionization MS technique which appears to afford greater sensitivity and specificity than that obtained

with MS instruments operated in the electron-impact mode. TCDD yields a negative-ion chemical ionization spectrum when oxygen is used as the reagent gas, containing the molecular ion at m/e 320 and a characteristic isotope cluster at m/e 176 ($M^- + O_2 - C_6H_2Cl_2O_2$) which carries more than 80% of the sample ion current. This technique may prove to be useful for convenient, specific detection of TCDD at levels of 10^{-12} - 10^{-13} g."

Shadoff and Hummel (1978) reported their latest refined technique and methodology to detect low levels (ppt) of 2,3,7,8-TTCD in environmental samples. They stated that:

"Using both gas chromatography low resolution mass spectrometry and gas chromatography high resolution mass spectrometry, 2,3,7,8-tetrachlorodibenzo-p-dioxin at the part per trillion level may be determined in preconcentrated extracts of bovine fat, liver and milk; human milk; rats; rice; grass; soil and water."

They also defined and made a clear distinction between detection limit and spectrometer sensitivity as follows:

"It should be noted that there is a clear distinction between detection limit as described here and the ultimate limit of the spectrometer to detect TCDD, hereafter referred to as sensitivity. The sensitivity of the spectrometer may be obtained by analyzing standard solutions. The data, free from interferences, do define the lowest detection limit possible. However, most of the data obtained during these studies of biological extracts yielded interferences of varying severity from other substances depending on the efficacy of the clean-up procedure and the nature of the sample. The detection limit is the minimum amount of TCDD which may be observed in the presence of the sample matrix, and usually varies from sample to sample."

McKinney (1978) also emphasized the need for continued development of new analytical methods for detection of trace amounts of TCDD.

"Elaborate methods for the clean-up and analysis of environmental samples allow the monitoring of part per trillion (ppt 10^{-12}) levels of TCDD. Special mass spectral techniques have generally been used to achieve the desired high sensitivity and specificity. Application of these methods to environmental

samples has, however, been complicated by time, cost and a number of effects of sample matrix and size that are involved in running the samples and interpreting the data. Promising new approaches that are being developed include negative chemical ionization mass spectrometry, bioassays and radioimmunoassays. Other techniques are of confirmatory nature.

The sparse, available analytical data provide little evidence that TCDD is accumulating in the environment as a result of normal domestic use of products such as the chlorinated phenoxy acid herbicides; however larger numbers of samples must be analysed with even more specific methods before this can be established."

Lamparski et al (1979) developed an analytical procedure which allowed determination of approximately 10 to 100 ppt (10^{-12} g/g) concentrations of 2,3,7,8-TCDD in fish. Their multistep clean-up procedure not only was very effective at removing halogenated aromatic compounds, such as PCBs, PBBs, and DDE, which act as interferences in analyses for TCDD, but also allowed high recovery and good precision for TCDD. Lamparski et al (1979) stated in their summary that:

"The technique involves digestion and extraction of the matrix followed by a series of adsorbent, and chemically-modified adsorbent, liquid column chromatographic clean-up steps. A final "residue polishing" step via elevated temperature reversed-phase high performance liquid chromatography is applied prior to detection by multiple ion mode gas chromatography-mass spectrometry. Using ^{13}C-labeled TCDD as an internal standard and carrier, the procedure has been validated for rainbow trout from approximately 10 to 100 ppt TCDD. Relative to this range, TCDD recovery is 75% $\pm$ 25%, and the precision of a single determination at the 95% confidence level (2σ) is $\pm$ 20% relative at 50 ppt TCDD concentration."

The usefulness of negative chemical ionization mass spectrometry as an analytical tool was pointed out by Dougherty and Hett (1978).

"Negative Chemical Ionization (NCI) mass spectrometry is unusually sensitive to non-specific toxic substances and is generally much less sensitive to biomolecules. These features make NCI mass spectra particularly attractive for screening environmental substrates for environmental contamination."

They also remarked on the levels of environmental contamination by toxic substances that should be of concern, toxicologically speaking, citing TCDD as an example:

> "Parts per trillion concentrations of toxic substances may be important if the molecules are exceptionally toxic. One class of molecules that falls into this category are the polychlorodioxins. Tetrachlorodioxin is lethal at parts per billion concentrations and this suggests that parts per trillion concentrations should be of concern for long term contamination. For most molecules, however, parts per billion concentrations are the lowest limits that one should concern oneself with in terms of long term toxicology. This means that the screening techniques that one should develop should be sensitive to nanogram quantities of toxic materials in the presence of orders of magnitude larger quantities of biomolecules."

As reported in Pesticide and Toxic Chemicals News of January 17, 1979, (Anonymous, 1979a) five laboratories had reported to the U.S.E.P.A. in January 1979 on their ability to analyze for TCDD in the ppt range; but they all noted continuing problems that a) made the techniques difficult and b) which also ruled out consideration of the techniques as presented as "state-of-the-art" methods. Representatives for the laboratories noted several factors which influenced the accuracy of analysis for TCDD at the ppt level (1 pg/g) as follows: sample size, sample extraction and clean-up, interference from other compounds, interference from equipment related sources, and precision of techniques.

Haas and Friesen (1979) recently reviewed past and current qualitative and quantitative methods for PCDD analyses. In the conclusion to their report they stated that:

> "In summary, the most sensitive and specific analysis for PyCDD in biologic matrices would involve an efficient sample clean-up with PyCDD determination on a large double-focusing mass spectrometer operated at a resolution of ca. 3000 to separate any extraneous hydrocarbon signal, with sample introduction by means of a glass-capillary-column-equipped gas chromatograph. By careful attention to detail and the use of suitable carrier compounds to compensate for sample losses, detection limits of ca. 10^{-13} g PyCDD/g sample should be possible from one g samples. To the best of my knowledge, no single laboratory has all these techniques available at the present time."

APPENDIX 3

APPENDIX 3

3 TOXICOLOGY OF CHLOROPHENOLS AND THEIR IMPURITIES IN TERRESTRIAL SYSTEMS

Information on the toxicological impact of CPs and their impurities on organisms in terrestrial and aquatic systems is presented in Appendices 3 and 4, respectively. Information derived from the literature was further separated as to its generation, either from laboratory or from field studies.

Because of the omnipresence of the toxic impurities in the CPs, a discussion of the toxicology of the CPs requires recognition of the toxicological effects from the impurities; where possible this has been done in both this and the aquatic section.

Kimbrough and Linder (1978) conducted an 8-month feeding study with Sherman rats to compare the effect of technical and purified PCP on the rat liver. Dietary concentrations of 0, 20, 100, and 500 ppm of technical grade PCP were fed to the male and female rats. A comparable feeding regimen used purified PCP. Without going into detail, the results obtained from the study suggested to the authors that most of the toxicity associated with feeding technical grade PCP to rats, at the dietary concentrations indicated, stems from toxic contaminants rather than from PCP. It is worth emphasizing this problem by noting the statement of Kimbrough and Linder (1978) in their discussion, that:

> "It is difficult to determine in retrospect which of the toxic effects reported in the literature are truly caused by pentachlorophenol and which are due to toxic contaminants. Our results suggest that the contaminants cause most of the alterations reported in rat livers."

The acute and chronic toxicity data for the CPs and their impurities are presented in tabular form. The pathological, physiological, biochemical, and other effects of the CPs and their impurities are discussed under appropriate headings (e.g. carcinogenicity, teratogenicity, and immunosuppression).

3.1 Laboratory Toxicology

3.1.1 Toxicology of Chlorophenols

3.1.1.1 Acute toxicity.

Plants

During one period in the development of commercial uses for CPs their herbicidal properties were investigated by various researchers. Blackman et al (1955a) assessed the biological activity of the CPs by determining the concentration required in either nutrient solution or agar medium which a) brought about a standard level of chlorosis in the duckweed, Lemna minor , an aquatic plant (App. 4, Sect. 4.1.1), or b) halved the radial growth of the mold, Trichoderma viride. In the later case they demonstrated that under controlled environment and pH, as the number of chlorine atoms substituted in the phenol ring increased, the biological activity was progressively augmented (Table A3-1).

TABLE A3-1 CONCENTRATIONS OF CHLOROPHENOLS, IN AGAR, REQUIRED FOR 50% INHIBITION OF RADIAL GROWTH OF THE MOLD, TRICHODERMA VIRIDE. (Adapted from Blackman et al, 1955a)

Compound	Conc. (moles/L)	Relative Conc. (ppm)[1]
4-chlorophenol	3.7×10^{-4}	47.6
2,4-dichlorophenol	5.3×10^{-5}	8.6
2,4,6-trichlorophenol	3.5×10^{-5}	6.9
2,3,4,6-tetrachlorophenol	3.4×10^{-6}	.8
2,3,4,5,6-pentachlorophenol	1.2×10^{-6}	.3

[1]No allowance made in calculations for Spec. Grav. of the agar.

During a herbicide screening program in Hawaii, Sund and Nomura (1963) noted the activity of PCP against germinating radish and sudan grass seeds, examples of dicotyledonous and monocotyledonous plants respectively. Intrigued by the PCP's

exceptional inhibiting effect compared to other standard herbicides, Sund and Nomura (1963) tested the phytotoxicity of other CP derivatives (Table A3-2).

TABLE A3-2 CONCENTRATIONS OF CHLOROPHENOLS, IN AQUEOUS SOLUTION, REQUIRED TO INDUCE 50% INHIBITION OF SEED GERMINATION IN RADISH RAPHANUS SATIVUS, AND SUDAN GRASS, SORGHUM SUDANENSE. (Adapted from Sund and Nomura, 1963)

Compound	R. Sativus		S. Sudanense	
	Conc. (moles/L)	(ppm)[1]	Conc. (moles/L)	(ppm)[1]
4-chlorophenol	8.53×10^{-4}	109.7	1.40×10^{-3}	180.0
2-chlorophenol	6.90×10^{-4}	88.7	2.17×10^{-3}	279.0
3-chlorophenol	4.81×10^{-4}	61.8	1.05×10^{-4}	13.5
2,4-dichlorophenol	3.01×10^{-4}	49.1	6.13×10^{-4}	100.0
2,5-dichlorophenol	3.01×10^{-4}	49.1	1.56×10^{-4}	25.4
2,4,6-trichlorophenol	2.28×10^{-4}	45.0	5.58×10^{-4}	110.2
2,4,5-trichlorophenol	1.62×10^{-4}	32.0	1.73×10^{-4}	34.2
2,3,4,6-tetrachlorophenol	6.90×10^{-5}	16.0	2.89×10^{-4}	67.0
pentachlorophenol	2.70×10^{-5}	7.2	2.03×10^{-5}	5.4

[1]Relative concentrations in ppm

Animals

The introduction and widespread use of PCP and NaPCP in the 1930's as preservatives suggested to several researchers the need for toxicity studies relevant to the possible occupational hazards of these materials. Deichmann et al (1942) reviewed the early reports on toxicity of PCP and summarized the acute toxic effects of PCP as follows:

> "Pentachlorophenol or its sodium salt, when absorbed in sufficient quantity, produced in all species of animals studied (dogs, rabbits, rats, guinea pigs), an acute toxic state characterized by increased blood pressure, hyperpyrexia (104-114 F.), hyperglycemia and glycosuria, hyperperistalsis, an increased and later a diminished urinary output, and rapidly developing motor weakness. In addition to these signs and symptoms, dying animals showed complete collapse

and asphyxial convulsive movements. Rigor mortis was immediate and profound. The post-mortem evidences of injury were not specific and consisted largely of extensive damage to the vascular system, with heart failure and involvement of the parenchymatous organs. Pentachlorophenol applied cutaneously caused a more or less pronounced edema of the skin, which in about a week became dry and wrinkled. Slight cracks developed and hair was lost completely from the treated areas, but the hair follicles and the deeper structures of the skin apparently suffered no permanent injury."

Toxicity data for o-CP to TTCP in terrestrial mammals are given in Table A3-3.

Farquharson et al (1958) investigated in rats, the acute toxicity and related biological effects of phenol and 11 CPs following interperitoneal injection. With an increase in chlorination, there was a corresponding increase in toxicity to the rats, as evidenced by an LD_{50} of 430 mg/kg for 2,4-DCP (Table A3-3) compared to an LD_{50} of 56 mg/kg for PCP (Table A3-4).

Characteristic signs of CP poisoning in rats included a) convulsions, which were usually more pronounced following injection of the lower CPs (PCP is non-convulsant) (App. 3, Sect. 3.1.1.3) b) production of hypotonia, which was sometimes overshadowed by the convulsions, c) a change in body temperature (i.e. an increase in temperature following injection of tetra-and penta-CPs), d) change in respiration rate, and e) acute rigor mortis. Other effects noted by Farquharson et al (1958), which were not consistently present, included chromodacryorrhoea, lachrymation, salivation and diarrhea.

Harrison (1959) reported that post mortem examination of acutely poisoned sheep that had received lethal doses of PCP (Table A3-4) showed a generalized congestion. The lymphatic nodes were enlarged and oedematous haemorrhages had occurred in the epicardium and along the aorta. Lungs showed isolated areas of collapse and generalized congestion, with some animals showing mild congestion in the stomach, intestines, liver and kidneys.

Walters (1952) stated that a 23 kg lamb given a sub-lethal dose by gavage of 23 g of PCP solution (approx. 4 mg PCP/100 g-bw). (The solution contained 5.12% PCP, 5% diacetone alcohol, and 90% mineral spirits; and weighed 0.7 kg/L). The lamb, examined histologically 24 h later, only showed some evidence of diffuse degenerative changes in the hepatic cells. No PCP was found in chemical analyses of the kidney, liver,

TABLE A3-3 ACUTE TOXICITY OF LOWER CHLORINATED PHENOLS IN TERRESTRIAL MAMMALS

CPs	Species	Route of Sex	LD_{50} mL/kg-bw or Admin.	mg/kg-bw	Notes	Ref.
2-chlorophenol (o-chlorophenol)						
	Rat		O	0.67 mL	50% mix. in olive oil	Deichmann and Mergard (1948)
		male	SC	0.95 mL		Deichmann and Mergard (1948)
		male	IP	230 mg		Farquharson et al (1958)
3-chlorophenol (m-chlorophenol)						
	Rat	male	IP	355 mg		Farquharson et al (1958)
			O	0.56 mL	25% mix. in olive oil	Deichmann and Mergard (1948)
			SC	1.39 mL	25% mix. in olive oil	Deichmann and Mergard (1948)
4-chlorophenol (p-chlorophenol)						
	Rat		O	660 mg	25% sol'n in olive oil	Deichmann and Mergard (1948)
			O	500 mg		Gurova (1964)
			SC	1030 mg	50% sol'n in olive oil	Deichmann and Mergard (1948)
			IP	281 mg		Farquharson et al (1958)
			D	1500 mg		Gurova (1964)
2,4-dichlorophenol						
	Rat		O	580 mg		United States Dept. H.E.W. (1976)
			IP	430 mg		Farquharson et al (1958)
			SC	1720 mg	20% sol'n in fuel oil	Deichmann and Mergard (1948)
		male	O	3670 mg		Kobayashi et al (1972)
		female	O	4500 mg		Kobayashi et al (1972)
	Mice	male	O	1630 mg		Kobayashi et al (1972)
		female	O	1630 mg		Kobayashi et al (1972)
2,6-dichlorophenol						
	Rat	male	IP	390 mg		Farquharson et al (1958)
2,3,6-trichlorophenol						
	Rat	male	IP	308 mg		Farquharson et al (1958)

O = oral
IP = interperitoneal
SC = subcutaneous
D = dermal

TABLE A3-3 ACUTE TOXICITY OF LOWER CHLORINATED PHENOLS IN TERRESTRIAL MAMMALS (Cont'd)

CPs	Species	Route of Sex	LD_{50} mL/kg-bw or Admin.	mg/kg-bw	Notes	Ref.
2,4,5-trichlorophenol						
	Rat		O	820 mg	20% sol'n in fuel oil	Deichmann and Mergard (1948)
			SC	2260 mg	20% sol'n in fuel oil	Deichmann and Mergard (1948)
		male	IP	355 mg		Farquharson et al (1958)
		male	O	2830 mg		Dow Chemical Co. (1976a)
		female	O	2460 mg		
		male	O	2960 mg		McCollister et al (1961)
2,4,6-trichlorophenol						
	Rat	male	IP	276 mg		Farquharson et al (1958)
3,4,5-trichlorophenol						
	Rat	male	IP	372 mg		Farquharson et al (1958)
2,3,4,6-tetrachlorophenol						
	Rat	male	IP	130 mg		Farquharson et al (1958)
Na-tetrachlorophenate						
	Rabbit		O	529 mg		Kehoe et al (1939)

O = oral
IP = interperitoneal
SC = subcutaneous

and muscles. The author stated that since the lamb was killed 24 h after drenching, only a part of the PCP may have been absorbed. In the same study, Walters (1952) stated that swine dosed by gavage at approximately 80 mg/kg - bw had large concentrations of PCP in the urine and feces at 24 h and 48 h following exposure, thus indicating much of the PCP was excreted. For example, 48 h samples of urine and feces had 21.2 and 3.06 ppm of PCP, respectively. Retention of PCP in the blood was 40 times greater than that in the control animals. Histological examination indicated PCP had affected the kidney and liver and to some extent the spleen.

Knudsen et al (1974) briefly reviewed the acute toxicity of PCP in mammals. Acute toxicity data quoted by them, supplemented with information from other sources is presented in Table A3-4. In summary, these authors stated that the acute symptoms of PCP intoxication were vomiting, hyperpyrexia, elevated blood pressure, increased respiration rate and amplitude, tachycardia and hyperglycaemia. Later frequent defecation, weakened eye reflex and developing motor weakness were seen.

To further improve and explain the acute toxicity data for PCP, Hoben et al (1976b) investigated the inhalation toxicity of PCP in Sprague-Dawley rats. The amount of PCP in aerosol inhaled was based on the assumption that a rat whose weight was 220 g would inhale 80 mL/min. The dosage of PCP received varied with the exposure times of 28 to 44 minutes. They reported the LD_{50} for inhaled aerosol of NaPCP in rats as 11.7 mg/kg-bw (Table A3-4) and, as they pointed out, it is much lower than the ingested dose of 210.6 mg/kg-bw (Deichmann et al, 1942) and the intraperitoneal dose of 34 mg/kg-bw determined in their laboratory.

3.1.1.2 Chronic toxicity

2,4-dichlorophenol

Kobayashi et al (1972) reported on a 6 months chronic oral toxicity study in ICR mice freely fed 2,4-DCP mixed food. They found that at levels up to 230 mg/kg/day, the minimum poisonous level and also the maximum dose level, there were no adverse changes in behaviour, growth rate, and blood and serum parameters. Histopathologically, only slight unfavorable changes in the liver were noted, such as, small round cell infiltration, and the swelling and unequal size of hepatic cells.

TABLE A3-4 ACUTE TOXICITY OF PENTACHLOROPHENOL (PCP) AND SODIUM PENTACHLOROPHENATE (NaPCP) IN TERRESTRIAL MAMMALS

Toxicant	Species	Sex	Route	LD_{50} (or as noted) mg/kg-bw	Notes (Concentration and solvents; time till death)	Ref.
Pentachlorophenol	Rat		O	27	0.5% in stanolex fuel oil; TTD, 3-19 h	Deichmann et al (1942)
			O	78	1% in olive oil; TTD, 3-11 h	Deichmann et al (1942)
		male	O	146		Gaines (1969)
		male	O	205	commercial grade PCP	Schwetz et al (1974b)
		female	O	175		Gaines (1969)
		female	O	135	commercial grade PCP	Schwetz et al (1974b)
		male	D	320		Gaines (1969)
		female	D	330		Gaines (1969)
		male	IP	56		Farquharson et al (1958)
			SC	90		Deichmann and Mergard (1948)
	Mice		O	120-140		Knudson et al (1974)
			SC	MLD 56		Davis et al (1959)
	Rabbit		O	LD 70-90	5% in stanolex fuel oil; TTD, 2-5 h	Deichmann et al (1942)
			O	LD 100-130	11% in olive oil; TTD, 10-16 h	Deichmann et al (1942)
			D	LD 60-70	5% in stanolex fuel oil, No. 1; TTD 1.5-4 h	Deichmann et al (1942)
			D	LD 90-100	5% in stanolex furnace oil; TTD 1.5-3 h	Deichmann et al (1942)
			D	LD 110-120	5% in Shell dione oil; TTD 5-6.5 h	Deichmann et al (1942)
			D	LD 130-170	5% in Shell fuel oil No. 3; TTD 6 h	Deichmann et al (1942)
			D	LD 40-50	1.8% in pine oil; TTD 9-22 h	Deichmann et al (1942)
			SC	LD 70-85	5% in olive oil; TTD, 3-6 h	Deichmann et al (1942)
			D	LD 350	11% in olive oil	Kehoe et al (1939)
	Guinea pig		O	100		Knudson et al (1974)
	Dog		O	150-200		Knudson et al (1974)
	Sheep		O	120		Harrison (1959)
	Calf		O	140		Harrison (1959)
Na-pentachlorophenate	Rat	male	INH	12	aerosol	Hoben et al (1976b)
			O	210	2% aqueous; TTD, 2-13 h	Deichmann et al (1942)
			SC	66	2% aqueous; TTD, 2-8 h	Deichmann et al (1942)
		male	IP	34		Hoben et al (1976b)
	Rabbit		O	LD 250-300	5% aqueous; TTD, 3-6 h	Deichman et al (1942)
			D	LD 250	10% aqueous; TTD 3-8 h	Deichmann et al (1942)
			SC	LD 100	10% aqueous; TTD, 7 h	Deichmann et al (1942)
			IV	LD 22-23	2% aqueous; TTD, 1.5-4 h	Deichmann et al (1942)
	Guinea pig		O	80-160		Dow Chemical Co. (1976b)

O = Oral
IP = interperitoneal
SC = subscutaneous
D = dermal
INH = inhalation
IV = intravenous
MLD = minimum lethal dose
LD = lethal dose, single administration. (Deichmann et al, 1942)
TTD = time till death (Deichmann et al, 1942)

2,4,5-trichlorophenol

Rabbits

McCollister et al (1961) summarized a chronic oral toxicity study of 2,4,5-TCP in rabbits by noting that repeated oral feeding by intubation, 20 doses of 0.5 g/kg in 28 days, produced very slight pathologic changes in liver and kidney.

Rats

McCollister et al (1961) in a companion study to the one in rabbits noted there was no evidence of adverse effects in rats, fed 18 doses of 0.3 g/kg of 2,4,5-TCP in 24 days, and at the 1.0 g/kg dosage the only significant effect was a slight increase in average weight of the kidneys. No pathological changes were found upon microscopic examination. In a 98 day rat feeding study, no adverse effects were seen in rats at a level of 0.1 g/kg/day of 2,4,5-TCP. Rats maintained at the 1.0 or 0.3 g/kg/day level showed diuria, but only mild reversible pathologic changes were seen in the liver and kidneys. Results of the rabbit and rat chronic feeding studies suggested to McCollister et al (1961) that there was little health hazard from ingestion of 2,4,5-TCP incidental to ordinary industry handling or commercial use.

Pentachlorophenol

Rats

In 1974 Knudsen et al reported on a 12 week dietary study of PCP in 64 Wistar rats. This study gives an indication of the results which might be expected in a longer term chronic toxicity study. (An analysis of the PCP used in the study showed levels of 200 ppm OCDD and 82 ppm pre-OCDD). The treatments, PCP at dietary levels of 0, 25, 50, and 200 ppm for 12 weeks, had no effect on food intake and behaviour. They noted that liver weight was increased at the 50 and 200 ppm dose levels accompanied by an increased activity of microsomal liver enzyme. Red blood cell counts fluctuated, first showing a higher count than normal at 6 weeks, then a lower count at 11 weeks. There was a dose-related decrease of calcium deposits in the kidney. The no-toxic effect level for all criteria was 25 ppm of PCP, which was confirmed in a 90 day feeding study in rats.

A series of investigations by a research team led by H.J. Hoben, University of Hawaii, dealt with the inhalation toxicity of NaPCP in rats. After developing the methodology (Hoben et al 1976a, 1976d) they dealt with the problem of distribution and excretion of inhaled PCP in rats (Hoben et al, 1976c), plus a follow-up study on protein

binding of PCP (Hoben et al, 1976e). In summary, in the rat repeated respiratory exposures to PCP did not result in an increase in the body burden of this compound as would be suspected from the 24 hour half-life determined from a single inhalation exposure. Removal of PCP from the body was not readily explainable from the data. It was suggested that since storage appeared unlikely, then an alternative explanation might be increased metabolism. Data supported the proposal that differences in binding of PCP between human plasma and rat plasma contribute to the longer retention and higher blood values observed in the human data of Casarett et al (1969) vs rat data from Hoben et al (1976b).

Sheep

Harrison (1959) reported from short term (19 days) chronic toxicity tests in sheep that the toxic cumulative effects of PCP as reflected in loss of body weight and general condition, could be caused by a daily intake of 27.8 to 55.6 mg of PCP/kg of body weight. The sheep had received daily doses of the PCP for 19 days by gavage of an aqueous suspension of PCP treated Douglas fir ground-wood.

Rabbits

Kehoe et al (1939) noted in an investigation of PCP chronic toxicity to rabbits that, following cutaneous treatment, no chronic systemic diseases were associated with the repetition of sublethal doses of PCP. Any observable local damage to the skin by application of the PCP was fully reversible. Preliminary studies had indicated that treatment of rabbits with 10 mL of 1% solution of PCP in mineral oil, for 21 consecutive days, caused no ill effects.

Deichmann et al (1942) noted in a NaPCP chronic toxicity study that rabbits, subjected to sublethal doses (i.e. 40 mg PCP/kg administered cutaneously for 100 days, or 3 mg PCP/kg administered orally for 90 days), had a gradual loss of weight but no significant changes in the hemoglobin content of the blood. The numbers of erythrocytes and of various types of leukocytes remained within normal limits. Rectal temperatures and blood sugar values rose markedly after the administration of single sublethal doses but curiously failed to show such elevations when the doses were repeated.

Cattle

In a program to determine possible mammalian toxicity of potential molluscicides to be used in control of the snail intermediate host of human schistosomes, Herdt et

al (1951) fed, in drinking water, 7.6 mg/kg/day of NaPCP and CuPCP to young bulls for a period of 5 weeks. No significant deviations were found in pulse, respiration rate, temperature, urinalyses, or in blood counts. Post mortem examinations revealed no toxic manifestations.

Harrison (1959) concluded from 11 day toxicity studies of PCP in calves that 35 to 50 mg/kg - bw, daily, would eventually cause death.

Pigs

Greichus et al (1979) initiated a series of studies on diagnosis and physiologic effects of PCP on young pigs. Following a pilot study which indicated acute toxicosis in young pigs receiving 30 mg PCP/kg/day for 7 days, 6 week old pigs were treated with purified PCP at 5, 10, and 15 mg/kg/day for 30 days. No overt signs of toxicity were evident. However, clinical pathological signs in treated pigs at the 10 and 15 mg/kg level included significantly larger livers, lower white blood cell counts, and higher blood urea nitrogen than the controls. Greichus et al (1979) further stated that levels of PCP in the blood, muscles, kidneys, and livers appeared to plateau and did not increase as treatment levels increased from the 5 to the 15 mg PCP/kg/day level.

3.1.1.3 Pathological and physiological effects

Rats

Farquharson et al (1958) during investigations on the biological action of a series of CPs, noted that the higher CPs produced a contracture of the isolated rat phrenic nerve diaphragm and a stimulation of in-vitro oxygen uptake of rat brain homogenate. Following correlation of these actions with the dissociation constant for the CPs, the authors suggested that the higher CPs interfered with oxidative phosphorylation, and that this property could be attributed to the chlorophenate ion. They also suggested that as convulsions are undoubtedly the most characteristic effect of CPs with pK values of 8.65 or higher (Table A1-1), that this reaction could be attributed to the undissociated molecules. Farquharson et al (1958) noted that although convulsions are not typical of CPs with lower pK values, they are produced by 2,6-DCP, 2,4,6-TCP, and sometimes by 2,3,6-TCP, which are all substituted in the di-ortho-position. They suggested that "It seems probable that the anion produced by ionization of 2:6-dichlorophenol cannot react, presumably with some basic centre in the receptor system, by virtue of the steric or electronic interference by the ortho chlorine atoms."

Powell et al (1973) implanted capsules with camphorated p-CP (CPC) into connective tissue in the backs of rats to evaluate tissue reaction to the drug. CPC is a widely used and accepted drug to medicate root canals for inhibition of tissue growth and for sterilization of pulpless teeth. The tests revealed moderate inflammation of tissue at 3 - 7 days following implantation, and recovery to normal in 14 - 30 days.

Humans

Kovsh et al (1970) reported that following DCP poisoning the existence of liver trouble and gall-excretion irregularities was observed in 7 patients. Diagnosis was carried out using clinical, X-ray, and biochemical tests. He noted that "The most convincing dyscholia features are bilirubin content changes and albumin proportions in the gall."

3.1.1.4 Teratogenicity, carcinogenicity, and cytogenicity

Teratogenicity

2,3,4,6-TTCP

Schwetz et al (1974a) evaluated the effect of 2,3,4,6-TTCP, both commercial and purified grade, on rat embryonal and fetal development. This was a companion study to the one on PCP. Dose levels up to and including the maximum tolerated dose of 30 mg/kg/day, were administered to pregnant Sprague-Dawley rats on days 6 through 15 of gestation. The researchers stated that the only fetal anomaly associated with the administration of 2,3,4,6-TTCP was delayed ossification of the skull bones - evidence of fetotoxicity, not teratogenicity. The authors observed that: "This anomaly occurs in all control populations in our laboratory and reflects either a mild, nonspecific toxicity or stress without definitive consequence." The no effect dose level of these materials for embryotoxicity was 10 mg TTCP/kg/day administered on days 6 through 15 of gestation. Schwetz et al (1974a) stated: "the only fetotoxic effect observed at 10 mg/kg, subcutaneous edema, was not observed at 30 mg/kg."

PCP

Schwetz et al (1974b) evaluated the effect of both purified and commercial grade PCP on rat embryonal and fetal development. Doses up to and including a maximum tolerated dose of 50 mg/kg/day were administered po (per os) to pregnant Sprague-Dawley rats on days 6 through 15, 8 through 11, and 12 through 15 of gestation. The authors stated that: "Following the administration of PCP, signs of embryotoxicity and fetotoxicity, such as resorptions, subcutaneous edema, dilated ureters and anomalies

of the skull, ribs, vertebrae and sternebrae were observed at an incidence which increased with increasing the dose. Purified PCP, with its low nonphenolic content, was slightly more toxic than the commercial grade of PCP containing a much higher level of nonphenolics." (Levels of the nonphenolics present in CPs used in biological research are infrequently reported.) It was further reported that: "The developing rat embryo is most susceptible to the toxic effects of a given dose of PCP during the period of early organogenesis. The no-effect dose level was 5 mg of the commercial grade of PCP/kg/day." In their summary, Schwetz et al (1974b) noted that relative to the maximum tolerated dose for the maternal animal, PCP had a greater effect on embryonal and fetal development than did TTCP as reported by Schwetz et al (1974a). While neither compound was teratogenic, PCP was highly embryolethal and embryotoxic. TTCP was not embryolethal and caused only a minimal degree of embryotoxicity.

Similar results to those of Schwetz et al (1974b) were reported by Hinkle (1973) who orally administered 1.25 - 20 mg PCP/kg/day to Golden Syrian hamsters from day 5 to 10 of gestation. Fetal deaths and/or resorptions were observed in 3 of 6 test groups. In samples of maternal blood and fat, and entire fetuses, concentrations of PCP in maternal fat persisted in measureable amounts up to 120 hours following the last oral dose and exceeded the maternal blood and fetal concentrations at that time.

Larsen et al (1975), after a preliminary set of experiments utilizing Charles River CD strain pregnant rats, stated that PCP may be slightly teratogenic, but considering the negligible amount of PCP that crossed the placental barrier, the teratogenic effect may be an indirect one resulting from toxicity to the maternal rat. They suggested their results should be verified in in-depth studies with rats and other species of animals. Courtney et al (1976) determined that PCP was not teratogenic in CD rats. PCP was administered by oral intubation at various times during gestation. The animals were sacrificed 1-2 days before parturition, and the fetuses examined for malformations. The malformations observed, which were very small and not statistically significant, included: a) enlarged cerebral ventricles, b) umbilical hernias, and c) slightly enlarged renal pelves.

Schwetz et al (1978) in a two year reproduction study on PCP in Sprague-Dawley rats fed PCP, with a low content of nonphenolic impurities, at dose levels of 0, 3, and 30 mg/kg/day for 62 days prior to mating, as well as during 15 days of mating, and subsequently throughout gestation and lactation. The researchers stated in their abstract that: "Except for a significant decrease in neonatal survival and growth among litters of

females ingesting 30 mg PCP/kg/day, measures of reproductive capacity were unaffected at both dose levels of PCP". Further conclusions from this study were that ingestion of 3 mg PCP/kg/day had no effect on reproduction or neonatal growth, survival or development, and that ingestion of this amount of PCP by females and 10 mg PCP/kg/day or less by males was not associated with significant toxicologic effects.

Carcinogenicity

Boutwell and Bosch (1959) examined the role of phenol and its derivatives in promoting the formation of both papillomas and carcinomas on the skin of adult, albino mice. Included in the large number of compounds tested were several CPs. The phenols, the CPs and other related compounds tested, which were dissolved in benzene, acetone, or dioxane, were applied repeatedly to the skin of the back of mice either alone or following a single application of dimethylbenzanthracane (DMBA). Papillomas appeared rapidly and in large numbers after treatment with DMBA followed by repeated applications of a 10% or higher solution of phenols, carcinomas appeared more slowly. The authors noted that phenol alone was capable of eliciting tumors. The response noted in the experiment (i.e. the appearance of papillomas and epithelial carcinomas) was dependant on the quantity of the compound applied, the susceptibility of the mice used, and the structure of the phenolic compound. As a demonstration of the structural relationships, PCP treated mice did not develop abnormalities, nor did 2,4,6-TCP treated mice. However, 2,4,5-TCP treatments induced large numbers of papillomas. Treatments with 2,4-DCP produced both papillomas and carcinomas. Response to the 2-chlorophenol treatment included formation of either papillomas alone, or both papillomas and carcinomas. The test with the 3-chlorophenol produced large numbers of papillomas but no carcinomas. In summary, the results would suggest a direct relationship between the presence of tumorigenic lesions and the isomeric structure of the chlorophenol in question, setting aside the possible effect from CP impurities.

Innes et al (1969) listed PCP as being non-tumorigenic in mice. The regimen consisted of a daily dose of 46.4 mg PCP (Dowicide 7)/kg body weight administered by gavage to the mice for 3 wk, for the period from 1 wk to 4 wk of age. After 28 days from birth, the mice received 130 ppm PCP in the diet, given ad libitum, until necropsy and diagnosis was undertaken at 18 months of age.

Schwetz et al (1978), reporting on a 2-yr toxicity study on PCP in rats, stated that PCP was not carcinogenic when administered to rats in their diet on a chronic

basis at dose levels sufficiently high to cause mild signs of toxicity (1, 3, 10, or 30 mg/kg/day).

Räsänen et al (1977) considered there was little likelihood of the following CPs being either carcinogenic or mutagenic, following negative Ames tests results:

2,3-dichlorophenol
2,4-dichlorophenol
2,5-dichlorophenol
2,6-dichlorophenol
3,4-dichlorophenol
3,5-dichlorophenol
2,3,5-trichlorophenol
2,3,6-trichlorophenol
2,4,5-trichlorophenol
2,4,6-trichlorophenol
2,3,4,6-tetrachlorophenol

An Ames test is only one of several test methods to determine mutagenicity and as an isolated test method should not be relied upon as conclusive evidence. The Ames test frequently fails to identify mutagenic substances that are organochlorine based.

The U.S. National Cancer Institute (1979) reported that under conditions of a 2-yr feeding study, 2,4,6-TCP was carcinogenic in male F344 rats, inducing lymphomas or leukemias, but did not cause cancer in female rats. In a corresponding 2-yr feeding study of 2,4,6-TCP in B6C3F1 mice, hepatocellular carcinomas or adenomas were induced in both sexes of mice.

The mutagenic-cytotoxic potential of PCP was recently reviewed by the U.S. E.P.A. Environmental Health Advisory Committee (1978) as follows:

> "PCP has not shown mutagenic activity in the Ames test (Andersen et al, 1972), the host-mediated assay (Buselmaier et al, 1973) or the sex-linked lethal test on drosophila (Vogel and Chandler, 1974)."

The Aug. 1, 1979, issue of Toxic Materials News (p. 243-4) noted that the U.S. National Cancer Institute's Carcinogenesis Testing Program included 2,4,-DCP, PCP, and pentachloroanisole, in a list of 106 chemicals slated for lifetime carcinogenicity bioassays. The chemicals were to be prescreened in bacteria.

Cytogenicity in plants

Amer and Ali (1968) investigated the cytological effects of p-CP (@ 250 mg/L), 2,4-DCP (@ 62.5 mg/L), and PCP (@ 174, 87, and 43.5 mg/L), on mitosis in lateral roots of seedling Vicia faba. All three chlorophenols affected a considerable decrease in the mitotic index when compared with the controls. All induced anaphase bridges, lagging chromosomes, "prophase-metaphase", and cytomixis. Disturbed meta- and ana-telophase comprised the most dominant type of anomalies. The authors noted that critical comparisons were not possible because the concentration of chemicals could not be employed on an equi-molar basis due to differential solubility in water and differential toxicity in the plants.

Cytogenicity in humans

Wyllie et al (1975) determined levels of PCP in the air and in serum and urine in personnel in a year-round wood treatment plant in Idaho in 1972, relative to incidence of chromosomal aberrations (breaks and gaps). They found no statistically significant differences between exposed and control groups in aberrations, although the sizes of the study groups were too small, six and four, respectively, to permit significant generalizations. (Sect. 5.2.5, Sect. 5.3).

3.1.2 Toxicology of Polychlorinated Dibenzo-p-dioxins and Chlorodibenzofurans

Delvaux et al (1975) placed the early studies on CPs and PCDDs in a historical perspective when they extensively reviewed the chemistry and toxicology of PCDDs, with particular reference to chloracne and chick edema.

Crow (1978), in discussing toxicity of chloracnegens, which included PCDDs and PCDFs, stated some of the elements in a chemical's structure which apparently determine its effective toxicity, as follows:

> "Certain basic elements of their chemical structure determine the toxicity of chloracneigens. The crudest of all factors is the degree of chlorination. It appears that a chlorine content somewhere between maximum and minimum produces the greatest toxicity. For example, the dioxins can have from one to eight chlorine atoms. Less than four, or more than six, chlorine atoms in a dioxin molecule greatly reduce toxicity: between four and six produce highly toxic compounds. However, the most important chemical property

determining toxicity is isomerism, that is, not just the number of halogen atoms but their exact position in the molecule.

"Lateral symmetry in the molecule, the chlorination of two adjacent carbon atoms, the presence of available hydrogen, and the absence of steric hindrance (where the spatial arrangement of a molecule "hides" reactive atoms, preventing certain reactions) all enhance toxicity. Recent work indicates that all chloracneigenic compounds are either exactly or approximately isosteric (that is, have the same distributions of electrons over the outside of the molecule - . . .). The absence of free hydrogen, lateral asymmetry, chlorination next to the diphenyl bridge (causing steric hindrance) and the absence of adjacent chlorines all result in the relative non-toxicity of the compounds on the right of Figure 3." (Fig. A3-1).

Moore (1978) summarized the information on the toxicity of TCDDs by stating that:

"The toxic effects of TCDD are produced with extremely low doses (ng $\cdot kg^{-1}$). Toxicity studies in laboratory animals are characterized by progressive wasting, weight loss and a hypoplastic effect on lymphoid tissues. The liver is a target organ, but the degree of effect varies between species. One of the earliest effects consistently produced in man is chloracne; integumentary effects are also produced in nonhuman primates. TCDD has been classified as a teratogen, but the dominant prenatal effect is fetal toxicity or death.

TCDD is reported to have an extended half-life (24 to 31 days) and is found primarily in liver and fat. Significant species variability occurs with regard to liver storage. Existing reports on the mutagenicity of TCDD are not definitive."

Poland et al (1979) summarized the structure activity relationships for the halogenated dibenzo-p-dioxins and dibenzofurans as follows,

". . . those congeners which induce AHH and ALAS activities and bind to the cytosol binding protein (1) have halogen atoms in at least three of the lateral ring positions (positions 2,3,7 and 8); (2) have an order of potency of substitution BR >Cl >F, NO_2, and (3) have at least one unsubstituted ring position. A similar structure activity is found for the halogenated dibenzofurans; again the 2,3,7,8-tetrachloro- and 2,3,7,8-tetrabromo- congeners are most active, and

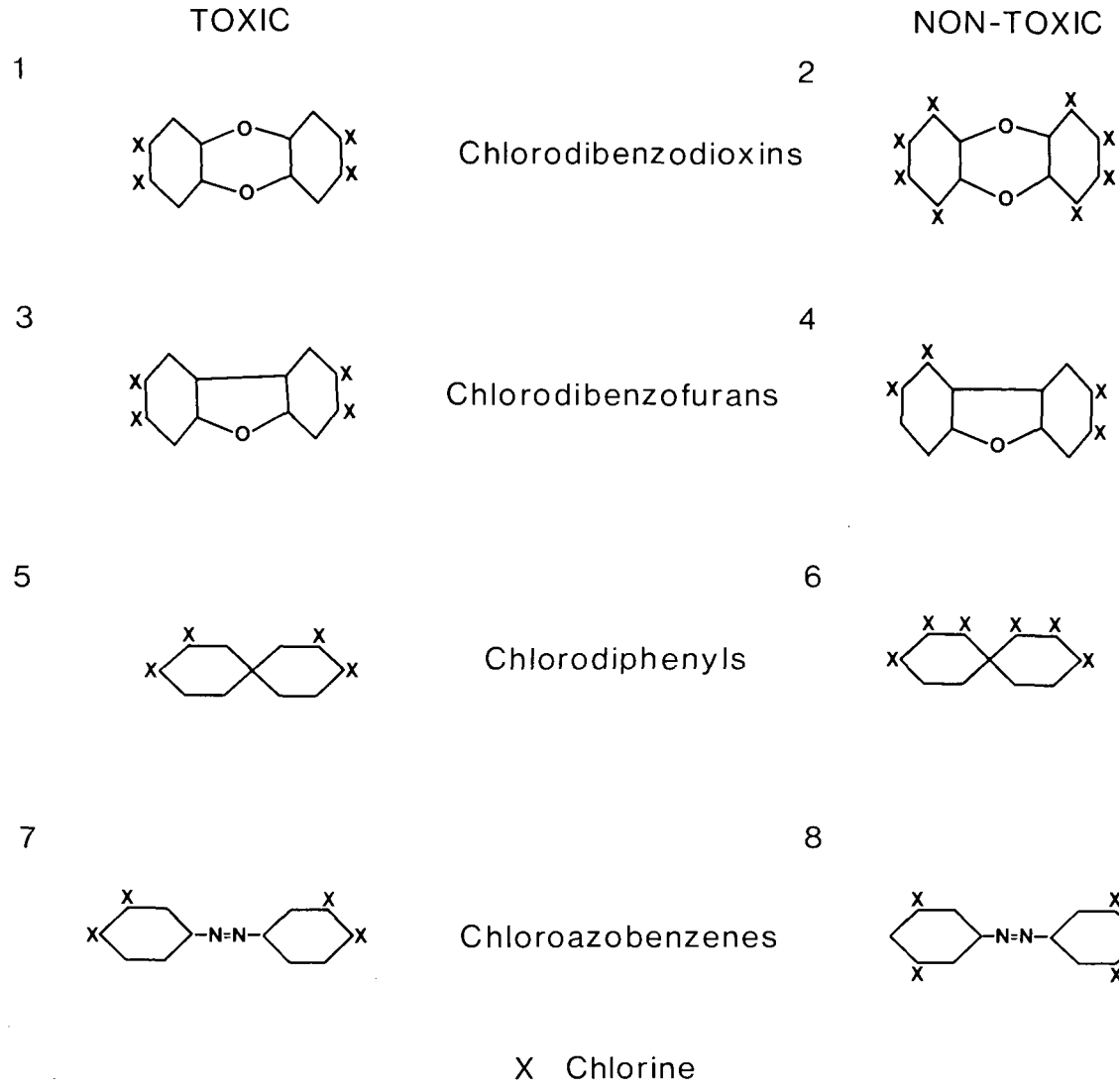

FIGURE A3-1 STRUCTURE AND TOXICITY OF FOUR CHLORINATED AROMATIC HYDROCARBONS (From Crow, 1978)
Note: "No available hydrogen (2), asymmetry (4), masking of the reactive bridge by chlorine atoms (6), and unadjacent chlorine atoms (8) all make the chemical relatively non-toxic"

TABLE A3-5 LETHALITY OF 2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN (Schwetz et al, 1973)

Species, Sex	Administration	Time of death, days post admin.	LD_{50} mg/kg	Dose mg/kg	Deaths/Treated (Numbers)
Rat, male	Oral	9 - 27	0.022	0.008	0/5
				0.016	0/5
				0.032	10/10
				0.063	5/5
Rat, female	Oral	13 - 43	0.045 (0.030-0.066)		
Guinea pig, male	Oral	5 - 34	0.0006 (0.0004-0.0009)		
Guinea pig, female	Oral	9 - 42	0.0021 (0.0015-0.0030)		
Rabbit, mixed	Oral	6 - 39	0.115 (0.038-0.345)		
	Skin	12 - 22	0.275 (0.142-0.531)		
	Intraperitoneal	6 - 23	--	0.032	0/5
				0.063	2/5
				0.126	2/5
				0.252	2/5
				0.500	3/5
Dogs, male	Oral	9 - 15		0.30	0/2
				3.00	2/2
Dogs, female	Oral	--		0.03	0/2
				0.10	0/2

Note: Responses to individual doses are given in those cases in which an LD_{50} could not be calculated. The LD_{50} for oral administration to rabbits was calculated using the method of Litchfield and Wilcoxon (1949); the remaining values were calculated using the Weil modification.

the octachloro-analogue is inactive. On the whole, the dibenzofurans follow the dibenzo-p-dioxins rather well."

(Note: AHH = aryl hydrocarbon hydroxylase, ALAS = δ-aminolevulinic acid synthetase)

3.1.2.1 Acute toxicity. Schwetz et al (1973) published acute lethality data for four CDDs, 2,7-DCDD, 2,3,7,8-TCDD, HCDD and OCDD. Since the greatest known hazard to health was associated with 2,3,7,8-TCDD, the majority of the data was on this CD (Table A3-5). In summary: a) the single-dose oral LD_{50} ranged from 0.0006 mg/kg in male guinea pigs to 0.115 mg/kg in rabbits of either sex; b) male rats and guinea pigs were more sensitive than females, although Moore (1978) noted in his review of toxicity of TCDD that a modestly elevated susceptibility of females to TCDD had been reported in several species; c) acute LD_{50}s were independant of method of dose administration; and, similarly, d) the dose of TCDD required to produce a toxic response was generally equivalent for all routes of exposure (Moore, 1978).

McConnell et al (1978a) determined that the oral LD_{50} of 2,3,7,8-TCDD in female rhesus monkeys (Macaca mulatta) following a single oral dose was <70 μg/kg.

Acute mortality data for 2,7-DCDD, HCDD, and OCDD were reported by Schwetz et al (1973) as follows:

> "Limited lethality data are available for 2,7-DCDD, HCDD and OCDD. HCDD (sample c) killed 1 of 2 and 0 of 2 male rats given oral doses of 100 and 10 mg/kg, respectively. No deaths occurred in four male mice given 2.0 grams/kg of 2,7-DCDD (sample a or b) orally or in two female rats given 1 gram/kg (sample a). For OCDD, oral doses of 1 gram/kg (sample d) to five female rats did not cause death; in four male mice, doses of 4 grams/kg also did not cause death. No signs of toxicity were observed in animals treated with either 2,7-DCDD or OCDD. The only sign of toxicity among animals treated with HCDD was loss of body weight."

McConnell and Moore (1976) presented, in an abstract, (also see McConnell et al, 1978b) information on the comparative toxicity of CDD isomers to mice and guinea pigs, as follows:

> "TCDD (i.e. 2,3,7,8-TCDD) was the most toxic of the isomers studied, although 1,2,3,7,8-pentachlorodibenzo-p-dioxin was almost as toxic. 1-NO_2-2,3,7,8 TCDD was more toxic than 1-NH_2-2,3,7,8. Hexachloro isomers were less toxic than the tetra and penta's and the trichloral isomers were the least toxic of

all. Pentachloropredioxins did not produce demonstrable effects even at 50,000 times the LD_{50-30} of TCDD. The lesions produced by the various isomers were consistent within a given animal species. Thymic and testicular lesions were found in both species. Subcutaneous edema, ascites, and hepatic lesions were characteristically found in dead mice, but not in guinea pigs. Adrenal cortical hemorrhage was a characteristic finding in guinea pigs, but was never observed in mice. Porphyria, as evidenced by fluorescence of various tissues under ultraviolet light, was observed in mice, but not in guinea pigs. Serum protein analysis showed discrepancies in mice only. Total serum protein was decreased due to lower levels of albumin. The alpha- and beta-globulin fractions were decreased, but gamma levels were within normal limits. Toxicity appears to be correlated in part with the degree of chlorination at the 2,3,7 or 8 position. Regardless of the isomer used, guinea pigs were more sensitive than mice to the lethal effects."

Moore et al (1976a) determined the single oral LD_{50-30} of 2,3,7,8-TCDF as between 5 and 10 μg/kg for Hartley guinea pigs. This data has now been confirmed in a recent study reported by Moore et al (1979). They also established that the LD_{50-60} for 2,3,7,8-TCDF in female rhesus monkeys was 1000 μg/kg, a dose that was about 20-fold higher than that estimated for 2,3,7,8-TCDD. When rats and mice were subjected to a massive dose of 6,000 μg/kg they exhibited only a mild toxicological reaction. The TCDF used in these tests was 88% pure with the remainder primarily a PnCDF.

Moore et al (1979) further reported that 2,3,7,8-TCDD

"... at a total dose of up to 6,600 μg/kg administered over a 30-day period failed to cause clinical signs of toxicity in mice. These results and those reported earlier indicate that doses of $\pm$6 mg/kg TCDF given orally (either as a single or divided dose) or subcutaneously do not cause overt toxicity in mice. This dose is 23-fold higher than the TCDD $LD_{50/30}$ in the same strain of mouse and 30 to 33-fold higher than the TCDD dose required to produce equivalent organ-weight effects."

3.1.2.2 Chronic toxicity. Schwetz et al (1973) stated that in chronic toxicity studies 2,3,7,8-TCDD and HCDD were acnegenic and highly embryotoxic. Both compounds were positive for the chick edema factor. The dioxins 2,7-DCDD and OCDD were not

chloracnegenic and caused little or no embryotoxicity. In addition, OCDD was negative for the chick edema factor.

Perinatal effects of CDDs were investigated in Wistar rats by Khera and Ruddick (1973). Oral treatment of pregnant dams with 2,3,7,8-TCDD at rates over 0.25 µg/kg/day effected development, while no adverse effects were seen at 0.125 µg/kg/day. Research with ^{14}C-2,3,7,8-TCDD administered at 2 µg/kg/day (2.99 µCi/mg) showed activity was primarily in the liver, and to a lesser extent in fat and brain, but with a single oral dose of 200 µg/kg administered in gestation days 16, 17, or 18, which was followed 6 hours later with tissue sampling, the ^{14}C was observed in the fetus and placenta. Khera and Ruddick (1973) summarized the perinatal effects of the other CDDs examined, as follows:

> "1,2,3,4-Tetrachlorodibenzo-p-dioxin elicited no apparent prenatal or postnatal effects when doses of up to 800 µg/kg/day were given orally for 10 days of gestation. Treatment with 250-2000 µg/kg/day of 2,7-dichlorodibenzo-p-dioxin (99% purity) had no significant effect on prenatal and postnatal measures of toxicity but caused a low incidence of cardiac lesions. 2,3-Dichlorodibenzo-p-dioxin and 2-chlorodibenzo-p-dioxin up to 2000 µg/kg/day had no adverse effect on survival, average weight, and skeleton of term fetuses."

McNulty (1977) reported that pilot tests indicated that young male rhesus monkeys (Macaca mulatta) were among the most susceptible of laboratory animals to 2,3,7,8-TCDD; a daily oral intake of less than 1 µg of 2,3,7,8-TCDD/kg was lethal.

Fries and Marrow (1975) fed rats 7 or 20 ppb of 2,3,7,8-TCDD in their diet for 42 days and observed that total retention of TCDD was closely related to total intake. They estimated that at steady state, total retention would be approximately 10.5 times the average daily intake. The study also revealed when feeding was stopped TCDD residues were eliminated from the body with half-lives of 12 and 15 days for males and females, respectively.

In a chronic toxicity study reported by Kociba et al (1976), rats were administered doses of TCDD at 0, 0.001, 0.01, 0.1, and 1.0 µg/kg/day, 5 days a wk for 13 wk. The highest dosage level, 1.0 µg/kg/day caused some mortality.

Recently, Kociba et al (1979) summarized their long-term toxicologic studies of 2,3,7,8-TCDD in Sprague-Dawley rats, as follows:

"The results of this study of rats ingesting TCDD for a lifetime serve as a basis for assessing the long-term chronic toxicity of TCDD. Continuous ingestion of a high dose level of 0.1 μg TCDD/kg/day (approximately 2200 ppt in diet) predictably caused multiple toxicologic effects. Liver toxicity was the most consistent observation, and this was accompanied by morphologic changes of the lymphoid, respiratory, and vascular tissues of the body. The incidence of hepatocellular carcinomas of the liver and squamous cell carcinomas of the lung, hard palate/nasal turbinates or tongue was increased at this dose level. Conversely, the incidence of tumors of the pituitary, uterus, mammary gland, pancreas and adrenal medulla was decreased at this high dose level of treatment. Similarly, the incidence of other spontaneous lesions such as chronic renal disease was also decreased at this high dose level.

"Lifetime ingestion by rats of 0.01 μg TCDD/kg/day (approximately 210 ppt in diet) caused a lesser degree of toxicity, primarily of the liver. However, there was no increase in the incidence of neoplasia at this dose level.

"Rats ingesting 0.001 μg TCDD/kg/day for two years had no adverse effects in spite of the fact the liver and fat each contained 540 ppt of TCDD at termination of the study."

3.1.2.3 Pathological and physiological effects. The pathological effects of 2,3,7,8-TCDD have been reviewed from the literature through 1975, in the National Research Council of Canada (NRCC) document on phenoxy herbicides (NRCC, 1978).

Kociba et al (1976) noted that with doses administered by gavage of 1 μg TCDD/kg/day to rats for 5 days/wk for 13 wk, there was decreased body weight and food consumption, icterus, increased serum bilirubin and alkaline phosphatase, pathomorphologic changes in the liver, lymphoid depletion of the thymus and other lymphoid organs, increased urinary excretion of porphyrins and delta-aminolevulinic acid, and minimal alterations of some hematopoietic components. They also stated that no discernible ill effects occurred in rats given 0.01 or 0.001 ug TCDD/kg/day, 5 days/ wk for 13 weeks.

As a follow-up study to their 13-wk chronic toxicity study (Kociba et al, 1976), Kociba et al (1978) conducted a 2-yr chronic toxicity and oncogenicity study of 2,3,7,8-TCDD in Sprague-Dawley rats, Spartan substrain. The rats were maintained for 2 years on a diet supplying 0.1, 0.01, and 0.001 μg of 2,3,7,8-TCDD/kg/day. Analysis of the diets indicated levels of 2200, 210, and 22 ppt of 2,3,7,8-TCDD. Kociba et al (1978)

stated that continuous ingestion of the diet containing approximately 2200 ppt of 2,3,7,8-TCDD for two years caused multiple toxicologic effects on the same order as found during the 13-wk study. Morphological changes occurred primarily in the hepatic, lymphoid, respiratory, and vascular tissues of the body. Terminal liver and fat samples from the rats at this high dose level contained 24,000 and 8100 ppt of 2,3,7,8-TCDD, respectively. Ingestion of the intermediate dose level of 0.01 µg of 2,3,7,8-TCDD/kg/day caused a lesser degree of toxicity. Terminal liver and fat content of 2,3,7,8-TCDD averaged 5100 and 1700 ppt, respectively. Lifetime ingestion of 0.001 µg of 2,3,7,8-TCDD/kg/day caused no effects considered to be of any toxicological significance. Liver and fat from rats in this latter group contained 540 ppt of 2,3,7,8-TCDD.

Allen et al (1977) included low levels (500 ppt) of 2,3,7,8-TCDD in the diet of female rhesus monkeys for 9 months. Death occurred in five of the eight animals between months 7 and 12 of the experiment at total exposure levels of 2-3 µg TCDD/kg body weight. Allen et al (1977) stated this compared to a single oral dose LD_{50-45} of 50-70 µg/kg of body weight. The morphological changes resulting from chronic exposure to TCDD at the level of 2-3 µg/kg included a marked effect on the haemotopoietic system. As exposure time lengthened, they noted that cellular deterioration in bone marrow and lymphoid tissue became widespread eventually leading to severe pancytopenia, or reduction in all of the cellular elements in the blood, prior to death.

During research on a PCB, Aroclor 1242, Fingerman and Fingerman (1977), described the effect of a contaminant, 1,2,3,4,5,6,7,8-octachlorodibenzofuran (OCDF), on molting of the fiddler crab, Uca pugilator. Following exposure to a concentration of 16×10^{-10}% of OCDF, there was only slight inhibition on the rate of molting, compared to complete inhibition of ecdysis by Aroclor 1242, even though in some organisms, such as chicks, Aroclor 1242 is far more toxic than the OCDF. The question of a potentiation or enhancement effect was not examined.

3.1.2.4 Biochemical effects. The biochemical effects of 2,3,7,8-TCDD are apparently species-specific and have been reviewed (NRCC, 1978). Generalizations of the biochemical effects of chlorodioxins in mice and guinea pigs have been summarized by McConnell and Moore, 1976 (App. 3, Sect. 3.1.2.1).

Higginbotham et al (1968) suggested that CPs could be precursors of a hydropericardium factor in chicks (i.e. a chick edema factor). This was based on their finding that the pyrolysate from technical grade 2,3,4,6-TTCP showed in an ec-glc test a peak pattern indicating the presence of the factor in comparison to a reference material

concentrated from the unsaponifiable fraction of a fatty acid material obtained as a by-product from the manufacture of oleic and stearic acids. The fraction was known to contain trace amounts of the hydropericardium factor. It was also known that CPs and their salts when pyrolyzed undergo condensation reactions to form CDDs.

Cantrell et al (1969) identified 1,2,3,7,8,9-HCDD isolated from a contaminated animal food fat as a factor in hydropericardium in chicks or chick edema disease.

Flick et al (1972) used mixtures of CDDs to study chick edema disease and in an abstract reported their findings as follows:

> "Feeding and injection studies have been performed with synthetic compounds which produce edema and pathological changes in S.C. White Leghorn chicks. Polychlorinated derivatives of dibenzo-p-dioxin were prepared either by chlorination of dibenzo-p-dioxin or by pyrolysis of 2,3,4,6-tetrachlorophenol and pentachlorophenol. The compounds prepared were administered to chicks by either oral feedings, oral intubation or intra-abdominal injection. Results of the feeding studies indicated that the hexachloro derivatives of dibenzo-p-dioxin most readily elicited the edema aspect of the chick edema disease but had low lethality, whereas the derivatives containing 3 and 4 chlorine atoms, per molecule also caused edema but were the most lethal of the compounds tested. Hepta-and octachloro-dioxins, although capable of producing evidence of disease, were less toxic than the other polychlorinated derivatives. The pyrolytic product of 2,3,4,6-tetrachlorophenol, a mixture of predominantly hexa-and heptachlorodibenzo-p-dioxins, was toxic when administered in single doses by way of oral intubation or by intra-abdominal injection."

In contrast to TCDD which is a potent porphyrogen in male mice, 2,3,7,8-TCDF did not produce porphyrin in chicks, even at lethal doses (i.e. 5 µg/kg/day for 21 days (Goldstein et al, 1976)).

Goldstein et al (1977), who investigated the hepatic effects of technical and pure grade PCP in female Sherman rats, reported that the biologically active CDDs and CDFs, present in some technical PCP preparations, have produced a number of liver changes in the female rats when separate lots of rats were fed technical or pure PCP at levels of 20, 100, and 500 ppm in their diet for 8 months. ("Technical pentachlorophenol was contaminated with 8 ppm hexa-, 520 ppm hepta-, and 1380 ppm octachlorodibenzo-p-dioxins and with 4 ppm tetra-, 42 ppm penta-, 90 ppm hexa-, 1500 ppm hepta- and 200 ppm octachlorodibenzofurans; pure pentachlorophenol contained less than 0.1 ppm of each

of these contaminants"). The hepatic effects, which were not attributable to the PCP alone but were consistent with the effects of CDDs and CDFs, included hepatic porphyria, increased liver weight, and increases in specific enzyme activity except for N-demethylase activity, which was essentially unaltered.

3.1.2.5 Immunosuppression. Research reports on immunosuppression effects following exposure to 2,3,7,8-TCDD have been reviewed in the phenoxy herbicide document from NRCC (1978). Based on the rather limited available documentation, the reviewers suggested that 2,3,7,8-TCDD probably has an immunosuppresion potential. Thigpen et al (1975) investigated the effect of subclinical levels of 2,3,7,8-TCDD on the response of mice to infection with either Salmonella bern or Herpevirus suis. Sublethal levels of 2,3,7,8-TCDD were used: 0.5, 1.5, 10 or 20 µg/kg, was given through a gastric tube once weekly for 4 weeks. They stated that their most important finding in the study was that extremely low levels of TCDD, which do not produce clinical or pathological change, still had the capacity to affect host defence. Vos (1977) also reviewed immunosuppression activity of 2,3,7,8-TCDD and concluded there was positive evidence for this relationship. He noted that TCDD caused thymus atrophy in all mammalian species studied. Immunosuppresion activity of the other PCDDs and PCDFs has not been researched to date.

Sharma et al (1978) in reporting on the reversal of immunologic and toxicologic effects of a single exposure of 2,3,7,8-TCDD in mice stated that:

> "Results indicate that TCDD in a single dose of 10 µg/kg is toxic to mice and the toxicity is evident by decreased thymus wt, increased liver wt, alterations in hematological parameters and histopathological lesions, primarily in liver. In general these toxic effects seems to be reversible and the reversal may be evident as early as 8 wk after exposure to TCDD."

Recently, Sharma and Gehring (1979) examined the effect of 2,3,7,8-TCDD on splenic lymphocyte transformation in mice following repeated exposure. The summary to their report stated that:

> "Male CD-1 mice were orally treated with 0.01, 0.1, 1 and 10 µg TCDD/kg body wt./week for up to 8 weeks. Randomly selected animals were sacrificed at 2, 4, and 8 weeks of exposure. . . . Splenic lymphocytes from these animals were cultured in vitro with or without the presence of phytomitogens, phytohemagglutinin, and pokeweed mitogen. The incorporation of ^{3}H-thymidine was measured as an indication of relative blast formation.

Exposure of animals to TCDD, even at the lowest level (0.01 μg/kg/wk for 2 weeks) caused a marked increase in the thymidine uptake by cultured lymphocytes. The blastogenic response of mitogens was reduced at high levels of TCDD exposure, indicating an immunosuppressive effect."

Faith and Luster (1979) investigated the effects of 2,3,7,8-TCDD exposure on parameters of immune function during the developmental period of Fischer/Wistar rats. The procedures and results of their research were briefly summarized as follows:

"Fetal and neonatal rats were exposed to TCDD through maternal dosing (5 μg/kg) on day 18 of gestation and on days 0, 7, and 14 of postnatal life (group 1). Another group of neonatal rats were exposed to TCDD through maternal dosing on days 0, 7, and 14 of postnatal life only (group 2). Body weights and relative thymus weights were found to be suppressed up to 135 days of age in group 1 but only up to 35 days of age in group 2. Parameters of cell-mediated and humoral immune function were investigated. TCDD suppressed cell-mediated immune function without affecting humoral immune function. TCDD-exposed animals had recovered normal cell-mediated immune function by 270 days of age.

"A group of inbred Fischer rats was exposed to TCDD as described for group 1 above. At 45 days of age these animals were utilized in lymphocyte homing studies. It was found that TCDD exposure alters homing patterns of lymphocytes from exposed animals when adoptively transferred to untreated animals. In addition, lymphocytes from nonexposed animals did not home normally when injected into TCDD-exposed recipients."

3.1.2.6 Teratogenicity, tumorigenicity, mutagenicity, and cytogenicity

Teratogenicity

The dioxin, 2,3,7,8-TCDD, is a known teratogen in mice and rats. Investigations by Moore et al (1973) and others were reviewed by Khera (1976), and NRCC (1978). Some of the fetal anomalies associated with exposure to 2,3,7,8-TCDD were subcutaneous edema and gastro-intestinal hemorrhage in the rat, and cleft palate and renal pelvic dilation in the mouse. In addition, Moore et al (1973) found that hydronephrotic kidneys were produced in mouse pups that nursed a mother treated with a single dose of 1 μg of TCDD during pregnancy or at time of parturition. Moore et al (1976b) determined that following administration of a single oral dose of 5 μg/kg of (^{14}C) TCDD to Fischer 344

rats that there was "continuous mobilization of TCDD from maternal tissue and ultimately its secretion in milk". Neubert et al (1973) reported a potentiation of teratogenic effects in mice from simultaneous administration of TCDD and 2,4,5-T, with one at a teratogenic level and the other at a threshold level.

Investigations by Schwetz et al (1973) identified the following dioxin compounds as being non-teratogenic in rats fed on days 6 through 15 of gestation at the levels indicated: 2,7-DCDD (100 mg/kg/day), HCDD (1.0 µg/kg/day), and OCDD (500 mg/kg/day). These authors pointed out that all CDDs are not alike in their toxicological properties (e.g. research has shown that the symetrical 2,3,7,8-TCDD was highly embryotoxic; the no-effect level for embryotoxicity was 0.03 µg of 2,3,7,8-TCDD/kg/day; whereas 1,2,3,4-TCDD was not embryotoxic at doses as high as 800 µg/kg/day). They further stated that under given test conditions HCDD was teratogenic in the rat at 100 µg/kg/day, given orally on days 6 through 15 of gestation, as evidenced by induction of cleft palate, whereas OCDD was not teratogenic at 500 mg/kg/day but did cause embryotoxicity. OCDD and 2,7-DCDD were neither teratogenic nor embryotoxic at 100 mg/kg/day.

Courtney (1976) conducted mouse teratogenic studies with 2,7-DCDD; 2,3,7-TriCDD; 1,2,3,4-TCDD; 2,3,7,8-TCDD; and OCDD, and concluded that relative to the high fetotoxicity and high teratogenicity of 2,3,7,8-TCDD, the related compounds studied were relatively non-toxic and were not teratogenic (Table A3-6). The anomalies in the data were explained as follows:

> "The mixture of dichloro- and trichlorodibenzo-p-dioxin produced a slight increase in the number of abnormal fetuses. At the lower dose this was partly due to an increase in kidney malformations which were a mild form of hydronephrosis. Since most of these fetuses (9/10) were from one litter and kidney malformations were not observed at the higher dose, it is doubtful that this malformation was produced by the compound under study. At both dose levels there was an increase in the incidence of clubfoot. This may reflect both a natural incidence of the malformation and uterine crowding, since these fetuses weighted slightly heavier and the litters were slightly larger than the controls. However, this does not negate a possible compound effect.
>
> "The 1,2,3,4 tetrachloro-isomer did not increase the incidence of malformation at any dose level by either oral or subcutaneous administration. Since this strain of mouse has a tendency to display clubfoot, the 8% incidence of this

TABLE A3-6 TERATOGENIC EVALUATION OF CHLORINATED DIBENZO-p-DIOXIN COMPOUNDS ADMINISTERED ORALLY IN CD-1 MICE (Courtney, 1976)

Dibenzo-p-dioxin Compound	Route	Dose/kg/day	No. of Litters	Av. No. Live Fetuses/ Litter	Av. No. Abnormal Fetuses/ Litter	% Anomalies/Total Fetuses		
						Cleft Palate	Kidney	Clubfoot
5%, anisole: corn oil[d]	oral	0.1 mL/mouse	15	11.0	0.8	0	1	4
2/3 mixture[a]	oral	100 µg	6	12.3	3.2	0	10[c]	9
	oral	200 µg	5	12.8	3.8	0	1	22[c]
1,2,3,4-tetrachloro-	oral	100 µg	4	11.8	0.8	2	0	2
	oral	250 µg	4	11.5	0.5	0	1	3
	oral	500 µg	5	11.6	0.2	0	0	0
	oral	1000 µg	5	11.8	1.0	0	0	0
2,3,7,8-tetrachloro-	oral	25 µg	7	10.9	4.6	3	34	3
	oral	50 µg	7	11.0	8.1	19	72	7
	oral	100 µg	6	9.7	8.3	66	71	13
	oral	200 µg	6	1.5	1.5	100	100	14
	oral	400 µg	5	0.4	0.4	100	50	50
15% anisole: corn oil[b,d]	oral	0.1 mL/mouse	5	11.2	0	0	0	0
octachloro-[b]	oral	5 mg	6	11.2	0.2	1	0	0
	oral	20 mg	6	11.6	0	0	0	0

[a]mixture = 40% 2,7 dichlorodibenzo-p-dioxin and 60% 2,3,7-trichlorodibenzo-p-dioxin

[b]sacrificed day 17 of gestation: all others sacrificed day 18

[c]Courtney suggested those deviant figures were not a result of the dioxin treatment

[d]control

anomaly observed at the 1000 µg/kg/day dose level needs further substantiation before being accepted as a compound effect.

"In contrast TCDD produced many abnormal fetuses at all doses studied and by both routes of administration. The majority of the malformations were cleft palates and hydronephrotic kidneys, both unilateral and bilaterial. A few other anomalies such as hydrocephalus and open eye were occasionally seen. TCDD administered subcutaneously produced a greater teratogenic response at a lower dose than administration by the oral route. Administration by the subcutaneous route at the lowest dose produced about 87% abnormal fetuses per litter. This made it difficult to demonstrate a dose related response since this was close to being a maximum response. At the higher dose with both routes of administration many fetuses were observed with marked edema and petechiae.

"The oral administration of 5 or 20 mg/kg/day of octachlorodibenzo-p-dioxin to pregnant CD-1 mice did not affect fetal development morphologically. The only malformation detected in this group of fetuses was a single cleft palate at the low dose."

Tumorigenicity

King et al (1973) stated that, based on preliminary results, unsubstituted CDD, 2,7,-DCDD, and OCDD possessed neither tumor promoting activity nor complete carcinogenic properties in mouse skin.

"In testing for complete carcinogenicity 0.2 ml of a solution of the test compound dissolved in acetone is applied three times weekly to the backs of mice. The octachloro-, dichloro-, and unsubstituted dibenzodioxin solutions in acetone contain 0.2, 3.0, and 80 mg/ml, respectively. For the study of promotion activity, each mouse was initially treated with 50 µg dimethylbenzanthracene (DMBA) 1 week prior to initiation of test compound application."

DiGiovanni et al (1977) studied the tumor initiating ability of 2,3,7,8-TCDD in mice using the two-stage, initiation - promotion system of carcinogenesis. TCDD when administered alone at a level of 2 µg/mouse was a weak tumor initiator after promotion for 32 weeks (0.1 papillomas/mouse, 14% of survivors with papillomas). They stated: "When TCDD was given concurrently with DMBA (dimethylbenz [a]anthracene) the number

of tumors observed increased slightly (2.2 papillomas/mouse, 63% of survivors with papillomas) when compared with the initiating ability of DMBA alone; i.e., when TCDD and DMBA were given together, an approximately additive effect was observed." The authors noted that the rate used was based in part on the ED_{50} of TCDD. The dose of 2 µg/mouse produced lethality in approximately 1/3 of the animals at 32 weeks.

A 2-yr feeding study with Sprague-Dawley rats fed 2,3,7,8-TCDD at 5 ppt/g in the diet (approximately equivalent to a weekly dose of 0.001 µg TCDD/kg body weight) showed statistically significant increases ($p=0.05$) in tumors at all sites examined, including the ear duct, kidney, and liver, as compared with the control animals (Van Miller et al, 1977).

Kociba et al (1978) concluded from their 2-year feeding study of 2,3,7,8-TCDD in rats (App. 3, Sect. 3.1.2.3) that continuous doses of 2,3,7,8-TCDD sufficient to induce severe toxicity (i.e. 0.1 µg of 2,3,7,8-TCDD/kg/day) increased the incidence of some types of tumors and reduced the incidence of other types. The increases were noted in the incidence of hepatocellular carcinomas in livers from female rats, and squamous cell carcinomas of the lung, hard palate/nasal turbinates, or tongue. Kociba et al (1978) also noted no increase in neoplasms in rats receiving sufficient 2,3,7,8-TCDD during the 2-yr study to induce slight or no manifestations of toxicity.

A report in Toxic Materials News (Feb. 21, 1979, pg. 64) noted that the U.S. National Cancer Institute had released results of an animal bioassay feeding study which indicated that 2,7-DCDD was not carcinogenic to rats or female mice.

Another report in Toxic Materials News (Aug. 1, 1979, p. 244) noted that 2,3,7,8-TCDF would be included as one of the 106 chemicals in a lifetime carcinogenicity bioassay program. The test protocol included prescreening in bacteria.

A recent study by Berry et al (1979) demonstrated that 2,3,7,8-TCDD

> " possesses remarkable inhibitory actions on skin tumor-initiation to polycyclic aromatic hydrocarbons (PAH). Almost complete inhibition of DMBA tumor initiation is achieved with a single nontoxic topical dose of 0.1 µg and with a dose of 0.01 µg approximately 80% inhibition was achieved. This potent anticarcinogenic effect may be related to the ability of TCDD to induce epidermal enzyme pathways responsible for detoxifying PAH carcinogens in the skin."

Mutagenicity

Hussain et al (1972) used three test procedures with the bacteria, Escherichia coli SD-4 and Salmonella typhimurium strain TA 1532 to demonstrate that, at levels above 2 µg/mL, 2,3,7,8-TCDD was mutagenic. They suggested the mutations observed might be through intercalation in DNA (Deoxyribonucleic acid).

A mutagenic evaluation of 2,3,7,8-TCDD by use of a dominant lethal test in male Wistar rats was conducted by Khera and Ruddick (1973). The male rats were dosed orally with 4, 8, or 12 µg/kg/day for 7 consecutive days followed by 7 sequential mating trials in the surviving males. Based on prenatal data in the sacrificed females, which included numbers of viable embryos, resorption sites, and corpora lutea, no apparent lethal mutations were noted during post meiotic stages of spermatogenesis. Histological examination of testes and epididymus in the surviving male rats, at 43 days post treatment, showed normal testes, but the epididymus had been involved in an inflamatory process with sperm granulomas formation.

Cytogenicity

Cytogenetic evaluation of DD, 2,7-DCDD, and 2,3,7,8-TCDD were made in two studies with male rats (Green and Moreland, 1975). In the first study when each of the three compounds was administered by intubation at 10 µg/kg/day for 5 days, no chromosomal aberrations were observed 6 hrs. after the last administration. The second study was restricted to 2,3,7,8-TCDD administered intraperitoneally at 5, 10, and 25 µg/kg and orally at 20 µg/kg. The rats receiving 15 and 20 µg/kg were sacrificed 24 h after injection, and the rats receiving the low rates of 5 and 10 µg/kg were sacrificed 29 days after injection. No evidence of chromosomal aberrations in the bone marrow of male rats was observed in any of the dioxin treated groups.

3.2 Environmental Toxicology of Chlorophenols

3.2.1 Microorganisms. Konrad and Gabrio (1976) studied both the effects of the active ingredients in 2,4,5-TCP and PCP, which were among a group of 43 pesticides, on dairy cultures and the suitability of milk contaminated in-vitro. Both the 2,4,5-TCP and PCP, when applied at concentrations of more than 100 ppm, exerted inhibiting effects upon the acid forming property of yoghurt, kefir, butter and cheese cultures. In comparison, most of the organophosphorus and chlorinated insecticides under investigation produced no detectable effects on the culture activity when used at concentrations up to 100 mg/kg.

Cserjesi and Roff (1975) in a test of various pesticides as possible agents to control moulds and sap stain fungi on unseasoned lumber, included as standards, TTCP and PCP. TTCP at concentrations of 0.46% (ai V/V) controlled Trichoderma virgatum and Penicillium sp., and at a concentration of 0.92% controlled Aureobasidium pullalans. PCP at a concentration of 0.25% controlled Trichoderma harzianum and at 0.125%, Phialophora sp. As noted by Cserjesi and Roff (1975), TTCP and PCP when used at commercial concentrations were least effective against Cephaloascus fragrans (brown mould). This species, controlled in the test by TTCP at 1.84% and PCP at 1.0%, has been controlled commercially only through addition of mercurials, such as phenylmercuric lactate. In Canada, there are two products registered as wood preservatives which contain mercury, either as phenylmercuric lactate or phenylmercuric acetate. Because economic levels of brown mold are not a frequent problem (probably less than one year in six in British Columbia) demand for, and use of, the wood preservatives which contain mercurials has been low.

Conkey and Carlson (1963) had tabulated the toxicity of biostatic agents suggested for use in the pulp and paper industry. The materials were screened against two species of bacterium frequently isolated from pulp and paper mill systems; a gram negative nonspore forming bacteria, Aerobacter aerogenes, and a gram positive spore forming bacteria, Bacillus mycoides. Two species of fungi were also included, Aspergillus niger and Penicillium expansum, which are both common to pulp and paper mill systems, stored pulp, and finished paper products. The test method used, which was fully described by Conkey and Carlson (1963), was basically an agar-Petri plate technique where the concentration level which showed no growth or cessation of growth of the organisms on the test substrate was considered as the point of inhibition. The inhibiting concentration of several formulations of chlorophenols against these organisms are presented in Table A3-7.

Unligil (1972) tested eleven strains of wood-rotting fungi from 6 species frequently isolated from wood in service in Eastern Canada. Of the following species and strains,

Coniophora puteana (Schum. ex Fr.) Karst (Strains A 328 and A302),
Coriolellus serialis (Fr.) Murr. (Strain A269),
Stereum radiatium Peck (Strain A293 and S488),
Stereum hirsutum (Willd, ex Fr.) S.F. Gray (Strains A265 and S392),
Coriolellus variiformis (Peck) Sarkar (Strains A355 and S603), and

TABLE A3-7 TOXICITY OF CHLOROPHENOLS SUGGESTED FOR USE AS BIOSTATIC AGENTS IN THE PULP AND PAPER INDUSTRY (Excerpted from Conkey and Carlson, 1963)[1]

Bioastatic agent	Active Ingredient	Inhibiting Concentration (ppm) (a.i. in substrate)			
		Aerobacter aerogenes (bacteria)	Bacillus mycoides (bacteria)	Aspergillus niger (fungus)	Penicillium expansum (fungus)
Biocide Dis-124	Polychlorinated phenols	55	10	20	35
Dowicide B	2,4,5-NaTCP	20	15	15	7
Dowicide F	2,3,4,6-NaTTCP	400	7	20	30
Dowicide G	NaPCP	200	4	25	30
Dowicide 2S	2,4,6-TCP	200	40	20	15
Nalco 21B	Mixture of saturated and unsaturated chlorophenols	200	25	55	550
Nalco 21M	NaPCP	200	5	45	40
Nalco 21S	2,4,5-NaTCP and NaPCP	25	5	35	55
Nalco 201	Blend of chlorinated phenols	50	9	35	65
Santobrite	NaPCP	250	4	35	30

[1]This Table lists the bioastatic activity of various chlorophenols and does not suggest that the listed products are presently available.

Hypoxylon rubiginosum (Pers. ex Fr.) Fr. (Strains C139 and C145),

Unligil (1972) suggested that C. puteana (Strain A328) could be used for evaluation of PCP containing preservatives. This particular strain decays wood rapidly and tolerates high concentrations of preservative. The threshold value of analytical grade PCP to this strain was approximately 0.008 mg/cm^3 of wood.

3.2.2 Mammals (other than human)

Swine

Schipper (1961) reviewed the toxicity of PCP to swine and conducted related studies. In his summary statement he stated that wood preservatives containing PCP or creosote may be extremely toxic to young swine coming into direct contact with freshly treated lumber containing excessive quantities of wood preservative. Liberal amounts of bedding aided in preventing the condition. Schipper (1961) also stated that wood preservatives containing PCP or creosote, when properly applied to wood which was thoroughly dried, would have little or no toxicity to swine.

Blevins (1965) reported a case of acute and lethal poisoning of baby pigs by PCP in a newly constructed farrowing house built with lumber excessively treated with PCP in used crankcase oil . His review of the literature indicated that animals with well developed urinary systems are most likely to recover from PCP poisoning; he further observed that baby pigs are notably deficient in that area.

Cattle

Spencer (1957) reported the death of two Hereford cows that had consumed 5% PCP in kerosene. No information was presented on total dose or on levels of PCP found in the organs, but at necropsy, eight hours after death, the liver and kidneys showed greatest damage, an extreme necrosis.

3.2.3 Humans

Canada

Bergner et al (1965) reported five cases of industrial PCP poisoning, one of them fatal, which occurred in employees of wood working plants in Winnipeg, Manitoba, in the summer of 1963. Inadequate precautions in handling and using a toxic material were the main reasons for the poisonings. When proper precautions were instituted, there were no further incidents reported. Bergner et al (1965) also reviewed other fatal cases of PCP

poisoning reported in the literature. They stated that there were some features common to all cases (e.g. the fatalities occurred in instances in which the workers involved were ignorant, careless or grossly negligent in observing a few simple protective and precausionary measures). Smith (1970) reemphasized the findings of Bergner et al (1965) and recommended safe handling procedures for use of PCP to reduce toxicity cases to a minimum.

Germany

Ten cases of industrial intoxication due to PCP were investigated and reviewed by Baader and Bauer (1951). The cases were all from one plant producing PCP through the HCB pathway (Sect. 2.1). Clinical symptomology of the PCP related poisonings was characterized by irritation of the mucosae and upper respiratory tract, neuralgic pain, and generalized acne of many months duration.

United States

Robson et al (1969) reviewed the world literature, published from 1952 to 1969, on poisonings from ingestion or absorption of PCP. Of the 51 cases reported, 30 had resulted in death, partly because no specific treatments were known and partly because of difficulty in diagnosis. In St. Louis, Mo., during the summer of 1967, a group of neonatal infants aged 6 - 14 days, were severely affected by PCP poisoning, and 2 deaths resulted. Over a period of 5 months, 11 other children were similarily intoxicated but to a lesser degree. Exchange blood transfusions were given to 6 of the 9 infants. This therapy resulted in immediate improvement and eventually complete recovery of the patients. The PCP poisoning syndrome, characterized by a profuse diaphoresis in all affected infants, had been produced by percutaneous absorption of PCP which had been used in the laundering of diapers and the infants' bed linen (Robson et al, 1969). Serum PCP levels dropped from a prior level of 11.8 mg/100 mL to 3.1 mg/100 mL in 24 h, following an exchange transfusion (Armstrong et al, 1969). In one infant that died approximately 3 h after the onset of the first symptom, autopsy tissue for the kidney, adrenal, heart and blood vessel, fat and connective tissue had PCP levels of 2.1 to 3.4 mg/100 g of tissue. PCP levels in diapers ranged from 2.64 to 17.20 mg/100 g, in shirts from 7.38 to 7.90 mg/100 g, in shirt backs from 22.4 to 195.0 mg/100 g, and in crib pads from 4.89 to 178.7 mg/100 g. The misused laundry product which led to the infant toxications was withdrawn by the manufacturer in September 1967.

It is interesting to note that during the course of the investigation by Armstrong et al (1969), PCP levels in serum and urine in adults attending premarital clinics, who were supposedly unexposed to PCP, averaged (mathematical mean) 0.004 mg/100 mL, and in infants at unaffected hospitals levels were 0.011 mg/100 mL in the serum, and 0.002 mg/100 mL in the urine.

Roberts (1963) reported a fatal aplastic anemia case following the patient's repeated exposure to 3% PCP and 1.5% TTCP. It should be noted however that the causative agent may possibly have been the CDD contaminant in the CPs. There had been no previous reports of aplastic anemia due to chlorophenol poisoning.

Casarett et al (1969) summarized a year's study of PCP in industrially exposed workers in Hawaii. They noted that the respiratory tract is a significant mode of exposure to PCP by workers, and that there are probable differences in excretion kinetics between single low-level exposures and chronic higher level exposures. They suggested that binding the plasma constituents (protein) occurs and that an appreciable PCP compartment occurs in tissues. They postulated that excretion rates after inhalation are dependent not only on exposure levels but on the steady state relationship of PCP concentrations in lung, blood, plasma protein, and tissue depots.

3.3 Environmental Toxicology of Polychlorinated Dibenzo-p-dioxins and Chlorodibenzofurans

In April 1973, the National Institute of Environmental Health Sciences sponsored a conference which critically reviewed and summarized the world literature and research activities on the chlorinated derivatives of dibenzodioxins and dibenzofurans. The proceedings of the conference were published in full in Environmental Health Perspectives, Experimental No. 5 (1973). Included in the proceedings was a paper by Huff and Wassom (1973). They reviewed the literature on CDDs and CDFs and compiled 242 references in an annotated bibliography which covered published documents from the years 1934 to 1973. Huff and Wassom (1974) also examined the toxicological aspects of the CDD and CDF contaminants which were associated with a) chick edema disease and b) chloracne, as did Kimbrough (1972).

Chick edema disease was first identified in 1957 when millions of broiler chicks died in the eastern and mid-western U.S. Characteristic symptoms in the chicks included both hydropericardium, or the presence of excessive fluid in the heart sac, and excess fluid in the abdominal cavity. Other symptoms such as subcutaneous edema and liver necrosis were followed in the third week by high mortality (Firestone, 1973). The

disease was due to the presence of a toxic chlorodioxin, 1,2,3,7,8,9-HCDD, in fleshing grease from hides processed with CPs (Huff and Wassom, 1974).

Chloracne is a common occupational dermatitis characterized by inclusion cysts, comedones, and pustules (Huff and Wassom, 1974). The agents responsible for producing chloracne were the contaminants in CPs, specifically the CDDs and CDFs.

Crow (1978) stated:

> "The chlorinated aromatic hydrocarbons which are known to have produced chloracne in humans are:
>
> 1) chlornaphthalenes
> 2) commercial polychlorinated biphenyls (PCBs)
> 3) polychlorinated dibenzofurans (PCDFs)
> 4) polychlorinated dibenzodioxins (PCDDs)
> 5) tetrachloroazobenzene (TCAB) and tetrachloroazoxybenzene (TCAOB).
>
> With the exception of the chlornaphthalenes and PCBs, all these compounds are contaminants formed accidentally during the manufacture of other chemicals."

As a further note of explanation, Crow (1978) stated:

> "The contaminants TCAB and TCAOB occur during the chemical reduction of dinitrochlorbenzene to the corresponding diphenyl hydroxylamine and dichloroaniline, both industrial significant intermediates. TCAB and TCAOB are never found in the final products and exposure therefore occurs only in the chemical industry. Little is known of the detailed chemistry of the commercial chlornaphthalenes. Whether their toxicity is due to contaminants or the pure chlornaphthalenes is unknown."

Crow (1978) also stated that: "The acneigenic potential of a chemical appears to be directly related to its overall toxicity. The importance of this fact is the implication that chloracne in an experimental animal can provide a very important screening test for systemic (not only skin) toxicity."

An item in Pesticide and Toxic Chemical News (Anonymous, 1977) noted that in 1949 an explosion in a 2,4,5-TCP plant, owned by Monsanto Co. in Nitro, W.Va., had resulted in chloracne and other symptoms among 278 workers.

An industrial accident resulting in chloracne occurred in 1953 in a German factory producing TCP, where 55 workers, who had been exposed to CDD, developed

chloracne. This and other incidents, in Amsterdam in 1963, and in the U.K. in 1968 (May, 1973), were reviewed by Hay (1976). Hay (1979) has listed the accidents in chemical plants manufacturing TCP for the period 1949 to 1976, and included those in plants in both Europe and U.S.A.

The most widely publicized incident involving the chloracnegenic agent, TCDD, was the accident at Seveso, Italy, July 10, 1976. Briefly, there was a "runaway reaction" in a reactor producing TCP. Pressure increased to the point where the hot vapor from the reactor was released to the atmosphere. After the volatiles evaporated, 2 kg of TCDD in the form of dust precipitated south of the factory, in an area inhabited by 2,000 people. Animal deaths followed 5 days later. Within a week there were reports of chloracne in children in the area (Hay, 1976).

Bonaccorsi et al (1978) related some of the commercial, health, and political factors which biased most attempts at identifying cause and effect relationships associated with exposure of plants, animals, and humans to TCDD prior to and following the Seveso incident. (The TCDD isomer was not further identified.) They noted that a government inquiry concluded that substantial amounts of TCDD had been dispersed over the area in previous years. Bonaccorsi et al (1978) stated that the continued reporting of confirmed cases of chloracne into 1978 documented "the persistence of a real and wide-spread risk of contamination for the population living in the area."

An item in the April 11, 1979, issue of Pesticide and Toxic Chemical News (Anonymous, 1979b) referenced a draft report by personnel of Givaudan Research Co. Ltd., and Hoffman-La Roche and Co. Ltd., which covered the two years following the Seveso explosion. The report noted that the chloracne effect was probably induced by TCDD exposure but that the incidence and severity of the effects were now close to those in provinces surrounding Seveso. They also reported that the frequency of abortion and congenital malformation in the TCDD contaminated area "remained well within the normal incidence in Europe." The news item (Anonymous, 1979b) also stated "The authors noted the TCDD accident link with an increase in the death rates of wild and domestic animals."

TCDD was the agent responsible for the poisoning of humans, horses, birds, cats, dogs, and rodents, following spraying of contaminated waste oil for dust control in three riding arenas and on a farm road in eastern Missouri in 1971 (Carter et al, 1975) (Kimbrough et al, 1977). Salvage or waste oil sludges had frequently been used to control dust in riding arenas and on dirt roads. The TCDD contaminant was traced to distillate

residues from 2,4,5-TCP production at a hexachlorophene plant which had discontinued production in 1971. In 1974, three years after the poisoning incident, sludge from the storage tank which had held the distillate residue contained 306 to 356 μg TCDD/g of sludge. In one sludge treated arena, of 85 horses which were exposed during exercising, 62 horses became ill and 48 died. The first death occurred within one month following exposure in the arena and the last death 2.5 yr later. Signs of toxicity in the affected horses were chronic emaciating weight loss, loss of hair, skin lesions, dependent edema, intestinal colic, dark urine, gross hematuria, conjuctivitis, joint stiffness, and laminitis. In addition to the laminea, the soles and frogs of the horses' feet were particularly inflamed (Carter et al, 1975). In regards to disposal of the contaminated oil on a farm road, Carter et al (1975) stated that "Seventy chickens that were exposed to the sludge oil on the farm road died within 2 weeks after the spraying." Human illnesses included one case of hemorrhagic cystitis in a 6-yr old girl.

Thalken et al (1975) conducted a field investigation on populations of beach mice, Peromyscus polionotus, and hispid cotton rats, Sigmodon hispidus, from a military site which had been treated with 114.1 kg ai 2,4,5-T/A, by repetitive treatments over a period of three years, 1962-64. Ten years after the last aerial application of the herbicide, the top 15.2 cm of test site soil had levels of TCDD ranging from 10 to 710 ppt. Liver tissue from the rodents inhabiting the site contained 210 to 1300 ppt of TCDD. In gross or histologic examination of 122 adults and 87 fetuses, no evidence of teratogenesis or toxicity was found. However an analysis of variance of liver and spleen weights for the beach mouse indicated significant differences between control and TCDD exposed animals although these differences were not explained by any histologic differences. The authors suggested that TCDD accumulated in liver tissue was from pelt contamination from burrowing and subsequent ingestion of soil particles via grooming.

Bollen and Norris (1979) investigated the influence of 2,3,7,8-TCDD on respiration in forest floor and Dystric Cryochrept soil samples from the H.J. Andrews Experimental Forest, Eugene, Oregon. The treatments were based on surface application rates of 2,4,5-T/ha of 0, 4.48×10^{-3}, 0.448, or 44.8 kg containing 0.1 ppm 2,3,7,8-TCDD. This is the equivalent of 13.1×10^{-9}, 13.1×10^{-7}, or 13.1×10^{-5} ppm 2,3,7,8-TCDD in forest floor; 5.2×10^{-9}, 5.2×10^{-7}, or 5.2×10^{-5} ppm 2,3,7,8-TCDD in soil. Results from the 4-week study indicated that 2,3,7,8-TCDD, at the rates used, had no effect on CO_2 evolution from the forest floor and that the rate of carbon metabolism was relatively constant. The researchers considered this unusual but suggested the reason could be that

the content of only moderately decomposible organic material in the soil was so high that its rate of oxidation would be nearly constant during the study period. Bollen and Norris (1979) did find that 2,3,7,8-TCDD significantly stimulated CO_2 evolution from the soil and in both the control and the treatments CO_2 production decreased linearly with time.

APPENDIX 4

APPENDIX 4

4 TOXICOLOGY OF CHLOROPHENOLS AND THEIR IMPURITIES IN AQUATIC SYSTEMS

The aquatic toxicology studies as related in this section emphasize that much of the early research on CP toxicology was undertaken because the CPs had superior molluscicidal and algicidal activity and were used extensively in aquatic environments. The rather lengthy Table A4-3 presenting toxicity data for CPs to aquatic biota was based on a Battelle Laboratory report by Becker and Thatcher (1973) for the U.S. Atomic Energy Commission. For each CP the table presents the information chronologically by publication date. (Note: The references from Becker and Thatcher (1973) for the toxicity data on the CPs have been included in the list of references, App. 11, although not all have been examined.) Physiological effects of CPs on aquatic biota were also frequently reported in the literature and are referred to in this appendix. Secondary effects of CPs, eg. taste and odor in water, and impairment of flavor in fish are documented. Information on the overall impact of CPs on aquatic ecosystems is also presented.

4.1 Laboratory Toxicology

4.1.1 Producers. The relationship of primary producers to the aquatic environment was briefly stated in the NRCC (1978) document on phenoxy herbicides, as follows:

> "The basis of all aquatic productivity is photosynthesis by algae, mainly the microscopic, free-floating forms known as phytoplankton. In shallow ponds and lakes, rooted vascular plants may also be important. This photosynthetic process is referred to as primary production".

The agents, such as NaPCP, which may have a toxic effect on algae and other primary producers, have a dual role and can be considered either beneficial, as when used as slime control agents in pulp mills, or deleterious, when they enter the aquatic environment and upset desirable ecosystems; in either event, it is necessary to know what effect particular concentrations of the toxic material will have on the photosynthesizing organisms. One of the earliest contributions in this area was from Gelfand (1941). In static tests to determine the suitability of NaPCP as a replacement for chlorine as an algicide for a 3.8×10^7 L spray pond, NaPCP at 15 ppm prevented initiation of algal growth in samples of filtered spray pond water with no live algal growth present. In water samples containing live growing algae, NaPCP at 15 ppm allowed algal growth for about 7

days before the algal growth was stopped; however, in pond water with 20 ppm NaPCP, algal growth stopped immediately.

Palmer and Maloney (1955) demonstrated that NaPCP at 2.0 ppm was toxic (growth relative to control in culture) or only partially toxic to algae for limited periods of time, 3 to 7 days (Table A4-3), depending on the species of algae. Any initial toxicity was overcome by the end of the 21 day test period.

Strufe (1968) noted a field study by Shiff and Garnett (1961) on effect of NaPCP on the microflora and microfauna of biologically stable pools in Southern Rhodesia as follows:

> "Shiff and Garnett (1961) studied the effects of different molluscicides on the microflora and microfauna of ponds, and found that following application of NaPCP at five p.p.m. the heaviest reduction of the microflora and microfauna took place within the first 24 hours. The total microflora and microfauna were reduced from an initial population level of about 30,000 individuals/litre to about 80 individuals/litre after 10 days. The population trends were back to normal within the next three weeks. Cladocera very quickly disappeared from the ponds treated with NaPCP and the recovery from the effect of this compound was very slow. On the other hand, Copepoda, Ostracoda and the alga Spirogyra recovered much faster.

Strufe (1968) in a review of NaPCP as a molluscicide, and its effects on producers, referred to the extensive investigations of Enigk and Düwel (1960) as follows:

> "Enigk and Duwel (1960) studied the effect of NaPCP on freshwater algae (Ankistrodesmus braunii) in a laboratory trial: 48-hour exposure to NaPCP at six to seven p.p.m. in Kandler's nutrient solution killed 65 percent of the algae. Destruction of the algae was manifested by ruptured cell structure and heavy accumulation of single cells. Following a short period of recovery (about eight days), the algae which survived exposure began to multiply normally again. A 48-hour exposure of fresh-water algae to 12 p.p.m. caused very heavy although incomplete destruction. Only 20 percent of the Protozoa survived 48-hour exposure to 12 p.p.m. of NaPCP; this percentage, however, quickly recovered and then began to reproduce normally again.
>
> "Enigk and Duwel (1960a) showed that broad-leaved plants such as coltsfoot (Tussilago) and plantain (Plantago) are extremely sensitive to NaPCP. Usually,

however, single leaves only are destroyed and not the whole plant. Moss is also severely damaged by NaPCP".

The water hyacinth, Crassipes eichornia, was relatively tolerant to NaPCP. Hirsch (1942) noted that 5.0 ppm of NaPCP was required to affect the appearance of the water hyacinth, and an 80 ppm dose was required for a complete kill (Table A4-3).

The effect of PCP on kelp, Macrocystis pyrifera, was investigated by Clendenning (1959). At a concentration of 2.66 ppm PCP eliminated all photosynthesis in kelp after 4 days exposure. The more water soluble NaPCP at a concentration of 0.3 ppm caused 50% inactivation of photosynthesis in 4 days (Clendenning and North, 1960) (Table A4-3).

Blackman et al (1955a) determined the concentration of various CPs in nutrient solution that induced 50% chlorosis in fronds of the duckweed, Lemna minor (Table A4-1). A similar order of toxicity (i.e. more highly chlorinated phenols have greater biological activity) was observed by Sund and Nomura (1963) in Hawaii when they noted the inhibition of seed germination in radish (Raphanus sativus), and sudan grass (Sorghum sudanense), by nine CPs (App. 3, Sect. 3.1.1.1 and Table A3-2). The practical application of Blackman's et al (1955a), and Sund and Nomura's (1963) test results was to incorporate PCP into a weed control program in irrigation ditches in Hawaii.

TABLE A4-1 CONCENTRATIONS OF CHLOROPHENOLS, IN NUTRIENT SOLUTION, REQUIRED TO INDUCE 50% CHLOROSIS IN FRONDS OF THE DUCKWEED, LEMNA MINOR (Adapted from Blackman et al, 1955a)

Compound	Conc. moles/L	Relative conc. ppm[1]
4-chlorophenol	2.2×10^{-3}	282.8
2,4-dichlorophenol	3.6×10^{-4}	58.7
2,4,6-trichlorophenol	3.0×10^{-5}	5.9
2,4,5-trichlorophenol	8.4×10^{-6}	1.7
2,3,4,6-tetrachlorophenol	2.6×10^{-6}	.6
2,3,4,5,6-pentachlorophenol	7.1×10^{-7}	.2

[1]No allowance made in calculations for Spec. Grav. of the nutrient solution.

4.1.2 Invertebrates. Batte and Swanson (1952) screened four CPs against liver fluke ova with the following results. Fluke ova (153 were used in the test) failed to hatch after an exposure to 2.5 ppm of NaPCP or 5 ppm of 2,4,5-TCP, for 24 hours at room temperature. Following exposure to 10 ppm of Na-2,3,4,6-TTCP or 10 ppm of 2,4-DCP there was slight reduction in fluke ova hatchability (i.e. 96.6% hatch, and 91.8% hatch, respectively).

Rubinstein (1978) investigated the effect of NaPCP on the feeding activity of a lugworm, Arenicola cristata. There was no marked effect at 40 µg/L of NaPCP, but feeding activity was significantly affected when NaPCP was at a level of 80 µg/L or higher. The lugworm, an inhabitant of sandy seashores, mixes organic material and oxygenated water into the substrate as it feeds to depths up to 30 cm. Rubinstein (1978) notes that inhibition of feeding activity affects sediment turnover.

Turner et al (1948) observed that NaPCP prevented the attachment and growth of mussels (Mytilus edulis), anemones, and barnacles, in sea water circulating systems when concentrations as low as 1.0 ppm were maintained continuously; whereas a solution of 0.1 ppm NaPCP was ineffective. This concentration was not effective in eliminating slime. The same tests showed that the organisms Molgula sp. and Bugula sp. could not survive after 1 days exposure to 1.0 ppm of NaPCP, and anemones, mussels, and barnacles after 4 days exposure to 1.0 ppm of NaPCP.

Batte and Swanson (1952) tested the molluscicidal activity of various compounds, including several CPs (Table A4-2) against two lymnaied snails, Pseudosuccinea coiumella Say and Fossaria cubensis Pfr., the intermediate hosts of liver fluke in Florida. Of the CPs tested, technical PCP gave 100% mortality to the snails following 24-h exposure in concentrations of 1 ppm.

Weinbach and Nolan (1956) noted that aerobic exposure of snails, Australorbis glabratus, to a low concentration of 2 ppm of PCP resulted in the accumulation of acetate, pyruvate, lactate, and inorganic phosphate in their tissues. PCP at lower concentrations stimulated respiration in snails, and at higher concentrations were inhibitory.

Okubo and Okubo (1965) investigated the influence of diluted sea water on the physiological activity of baby-neck clam, Venerupis japonica, and the toxic effect of PCP. The research was undertaken to determine whether the use of PCP followed by a heavy rainstorm could have contributed to the mass destruction of littoral fishes and clams on the coast of the Ariake Sea, Kyushu, Japan, in early July of 1961 and 1962. In essence,

TABLE A4-2 LETHAL CONCENTRATION OF CHLOROPHENOLS FOR 100% MORTALITY OF THE LYMNAEID SNAILS, PSEUDOSUCCINEA COLUMELLA SAY AND FOSSARIA CUBENSIS PFR., FOLLOWING 24 h EXPOSURE. (Excerpted from Bate and Swanson, 1952)

Compound	Lethal concentration (ppm)
o-chlorophenol (tech)	10 (less than 100% mortality)
p-chlorophenate (Na-salt, 25%)	10 (less than 100% mortality)
2,4-dichlorophenate (Na-salt, 25%)	10 (less than 100% mortality)
2,4-dichlorophenol (tech)	10
2,4,5-trichlorophenol (tech)	10
2,4,6-trichlorophenol (tech)	5
Na-2,4,5-trichlorphenate (85%)	2.5
Pentachlorophenol (20%)	1.25
Pentachlorophenol (tech)	1.0
Pentachlorophenol (8%)	0.833

Okubo and Okubo (1965) determined that in diluted sea water the osmotic pressure of body fluids of Venerupis can drop to a threshold level, equivalent to that for sea water at 5500 ppm in "chlorinity" (op.cit.), at which level the lethal concentration of PCP to Venerupis drops to 1/10 the normal value.

Fox and Rao (1978), who evaluated the effects of NaPCP in vivo and in vitro on certain hepatopancreatic enzymes in the blue crab, Callinectes sapidus, stated:

> "Fumarase, malate dehydrogenase and succinate dehydrogenase were inhibited by Na-PCP . . . in vivo, whereas isocitrate dehydrogenase was stimulated. Of those tested, lactic dehydrogenase was the least affected cytoplasmic (soluble) enzyme in vivo while pyruvate kinase and glucose-6-phosphate dehydrogenase were inhibited at least 50% by Na-PCP. Glutamate-pyruvate transaminase was also inhibited. Na-PCP . . . had an inhibitory effect on the various enzymes tested in vitro at concentrations of 10^{-4} M or higher. In general, the mitochondrial enzymes were more susceptible than cytoplasmic enzymes to . . . Na-PCP. The calcium activated ATPase from the microsomal fraction of the crab hepatopancreas was inhibited by Na-PCP . . . in vitro and in vivo."

Cantelmo et al (1978) used the grass shrimp, Palaemonetes pugio, to determine the effect of NaPCP on oxygen consumption in vivo, and the blue crab, Callinectes sapidus, for a study on effect of NaPCP on tissue respiration in vitro. In a summary statement, Cantelmo et al (1978) stated that their results indicated "that the biocidal

effects of PCP may not be solely due to its ability to uncouple oxidative phosphorylation but also due to a disruption of the overall metabolic activity." The detailed results of their study were abstracted by the authors as follows:

> "The oxygen consumption of the grass shrimp, Palaemonetes pugio, was determined at different stages of the molt cycle. At each stage of the molt cycle, the oxygen consumption varied in relation to periods of activity. In order to minimize the errors in establishing basal (control) rates of oxygen consumption, measurements were made over extended periods (18 to 24 hours). In contrast to the previous reports of progressive increases in oxygen consumption during proecdysial stages in other crustaceans, we noted significant increases in oxygen consumption just prior to and during the actual shedding of exoskeleton (ecdysis) in grass shrimp. The effects of sodium pentachlorophenate (Na-PCP) on oxygen consumption varied depending on the stage of the molt cycle, concentration of Na-PCP and extent of pre-exposure of shrimp to Na-PCP. At concentrations of 1.5 and 5.0 ppm, Na-PCP did not alter the oxygen consumption of shrimp in intermolt and proecdysial stages of the molt cycle. Late proecdysial shrimp exposed to 5.0 ppm Na-PCP exhibited an increase in oxygen consumption in relation to ecdysis to the same level as that of control shrimp. However, following ecdysis, the shrimp exposed to 5.0 ppm Na-PCP exhibited a dramatic decline in oxygen consumption and died within three hours. This increased sensitivity during the early postecdysial period appeared to be related to an increase in the uptake of Na-PCP at this stage compared to intermolt and proecdysial stages. A decline in oxygen consumption as noted above could be induced in intermolt shrimp by using higher concentrations of Na-PCP. Exposure of shrimp to 10 or 20 ppm Na-PCP, or to 5 ppm followed by 20 ppm Na-PCP caused an initial increase in oxygen consumption and a subsequent decline leading to death. The survival time of intermolt shrimp pretreated with 5 ppm Na-PCP was longer than that of shrimp exposed directly to 10 or 20 ppm Na-PCP. Although 20 ppm 2,4-dinitrophenol (DNP) caused an initial increase in oxygen consumption in intermolt shrimp, this was not followed by any deeline in oxygen consumption or death during a 24-hour exposure.
>
> "The effects of Na-PCP and DNP on tissue respiration in vitro were studied using the blue crab, Callinectes sapidus. At concentrations of 1 x 10^{-6} M and

5×10^{-5} M, these compounds did not alter the oxygen consumption of the muscle, gill and hepatopancreas. At a concentration of 5×10^{-3} M, both Na-PCP and DNP caused an inhibition of oxygen consumption of isolated tissues."

Doughtie and Rao (1978) had noted that in grass shrimp, Palaemonetes pugio, exposed to 1.0 ppm of NaPCP for the duration of the molt cycle, there was little evidence of any pathological changes until after ecdysis when it became very extensive and evident at the ultrastructural level in gills, hepatopancreas, midgut, and hindgut. The duration of the molt cycle in the grass shrimp, as referred to in the studies of Doughtie and Rao (1978), Rao et al (1978), and Conklin and Rao (1978), can be influenced by various physical and environmental factors as stated by Conklin and Rao (1978):

> "The length of the molt cycle varies with the size of the shrimp, season and other factors such as temperature and photoperiod. The average duration of the molt cycle for representative grass shrimp tested was 13 days."

Rao et al (1978) studied the effect of NaPCP at levels of 0.1, 0.5, 1.0 ppm, on limb regeneration in the grass shrimp during the molt cycle. They stated that depending on the concentration, NaPCP caused a complete inhibition of limb bud development, or reduction of limb bud growth without altering the intermolt duration. Effects were more noticeable during the initial phases of limb regeneration. The authors suggested that crustacean limb regeneration could be used as a sensitive bioassay for studying the effects of environmental pollutants.

Conklin and Rao (1978) observed that in static sea water tests the toxicity of NaPCP to grass shrimp varied in relation to the molt cycle. Intermolt and premolt shrimp had 96-h LC_{50} values of 2.63 and 2.74 ppm NaPCP. Those animals that molted during the 96 hour bioassays had an LC_{50} of 0.44 ppm NaPCP. They considered that the first few hours following ecdysis represented the most sensitive period of the molt cycle for the toxic effects of NaPCP. Conklin and Rao (1978) stated that this is the lowest of all the LC_{50} values reported previously for adult crustaceans and is comparable to those for fish and larval crustaceans.

The toxicity of NaPCP was determined by van Dijk et al (1977) for two marine decapods, Crangon crangon (Linn.) and Palaemon elegans (Rathke), and one brackish water decapod, Palaemonetes varians (Leach), all collected from Netherlands localities (Table A4-3). Unless otherwise noted the data were obtained from static tests in natural sea water at 15°C and pH 7.5 - 8.0. The 96-h LC_{50} values for NaPCP for the adults of C. crangon, P. elegans, and P. varians were 1.79 ppm, 10.39 ppm, and 5.09 ppm, respectively,

TABLE A4-3 TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
o-chlorophenol	Lepomis macrochirus (bluegill)	SB,FW,LS	8.1 8.2	48 h-TLm; 20°C 24 h-TLm; 20°C	Lammering & Burbank (1961)
o-chlorophenol	Lepomis macrochirus (bluegill)	SB,FW,LS	8.4 2.0	96 h-TLm; 25°C Taints flesh	Henderson et al (1961)
o-chlorophenol	Lepomis macrochirus (bluegill)	SB,FW,LS	10.0	96 h-TLm (All data at 25 C, soft water.)	Pickering & Henderson (1966)
	Pimephales promelas (fathead minnow)		11.6	96 h-TLm	
	Carassius auratus (goldfish)		12.4	96 h-TLm	
	Lebistes reticulatus (guppy)		20.2	96 h-TLm	
o-chlorophenol	"minnows"	SB,FW	58.0	24 h-TLm	Ingols & Gaffney (1965)
o-chlorophenol	Carassius auratus (goldfish)	SB,FW,LS	142-311 104 82.8 10.0	Total kill, 8 h: 27°C Killed 83%, 8 h; 27°C Killed 64%, 8 h; 27°C Killed 20%, 8 h; 27°C	Gersdorff & Smith (1940)
o-chlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	5.3	96 h-LT; 10 C	McLeese et al (1979)
m-chlorophenol	Carassius auratus	SB,FW,LS	70.5 20.6	8 h-LC_{100} 27°C 8 h-LC_{62} 27°C	Gersdorff and Smith (1940)
p-chlorophenol	Carassius auratus (goldfish)	SB,FW,LS	54.3 6.3	8 h-LC_{100} 27°C 8 h-LC_{54} 27°C	Gersdorff and Smith (1940)
p-chlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	4.6	96 h-LT; 10°C	McLeese et al (1979)
2,4-dichlorophenol	Salmo gairdneri (rainbow trout)	SB,FW,LS	5.0	Dead, 3 h; 12.8°C	Applegate et al (1957)

SB = Static Bioassay, CB = Constant-flow Bioassay, FW = Freshwater, SW = Sea (Salt) Water, LS = Lab Study, FS = Field Study

TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
2,4-dichlorophenol	Lepomis macrochirus (bluegill)	SB,FW,LS	5.0	Dead, 12 h	Applegate et al (1957)
	Petromyzon marinus (sea lamprey)		5.0	Dead, 12 h; larvae	
	Salmo gairdneri (rainbow trout)		5.0	Dead 6 h; 12.8°C	
	Lepomis macrochirus (bluegill)		5.0	"Ill." 0.5 h	
	Petromyzon marinus (sea lamprey)		5.0	"Ill." 1 h; larvae	
2,4-dichlorophenol	Ptychocheilus oregonensis (northern squawfish)	SB,FW,LS	10.0	Equilibrium loss, 0-1 h; dead, 1-3 h; 10.6°C	MacPhee & Ruelle (1969)
	Oncorhynchus kisutch (coho salmon)		10.0	Dead, 0-1 h; 10.6°C	
	O. tshawytscha (chinook salmon)		10.0	Dead, 0-1 h; 10.6°C	
2,6-dichlorophenol	Salmo gairdneri (rainbow trout)	SB,FW,LS	5.0	Dead, 13 h; 12.8°C	Applegate et al (1957)
	Lepomis macrochirus (bluegill)		5.0	Dead, 5 h	
	Petromyzon marinus (sea lamprey)		5.0	"Ill." 12 h; larvae	
2,6-dichlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	19.1	52 h-LT; 10°C	McLeese et al (1979)
3,4-dichlorophenol	Salmo gairdneri (rainbow trout)	SB,FW,LS	5.0	Dead, 3 h; 12.8°C	Applegate et al (1957)
	Lepomis macrochirus (bluegill)		5.0	Dead, 3 h	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
3,4-dichlorophenol	Petromyzon marinus (sea lamprey)	SB,FW,LS	5.0	Dead, 11 h; larvae	Applegate et al (1957)
3,5-dichlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	1.5	96 h-LT; 10°C	McLeese et al (1979)
	Mya arenaria (soft-shelled clam)		9.8	35 h-LT; 10°C	
Trichlorophenol	"sewage bacteria"	SB,FW,LS	60.0	50% inhibition of "cumulative gas production"	Ingols & Gaffney (1965)
Trichlorophenol	Ptychocheilus oregonensis (northern squawfish)	SB,FW,LS	1.0	Equilibrium loss, 0-1 h; dead, 2-4 h; 11.1°C	MacPhee & Ruelle (1969)
	Oncorhynchus tshawytscha (chinook salmon)		1.0	Equilibrium loss, 0-1 h; dead, 2-4 h; 11.1°C	
	Oncorhynchus kisutch (coho salmon)		1.0	Equilibrium loss, 0-1 h; dead, 1-2 h; 11.1°C	
	Ptychocheilus oregonensis (northern squawfish)		5.0	Equilibrium loss, 0-0.5 h; dead 0.5-1 h; 13.9°C	
	Oncorhynchus kisutch (coho salmon)		5.0	Dead, 0-0.5 h; 13.9°C	
	Ptychocheilus oregonensis (northern squawfish)		10.0	Dead, 0-1 h; 10.0°C	
	Oncorhynchus kisutch (coho salmon)		10.0	Dead, 0-1 h; 10.0°C	
	Salmo gairdneri (rainbow trout)		10.0	Dead, 0-1 h; 10.0°C	
	Ptychocheilus oregonensis (northern squawfish)		10.0	Equilibrium loss, 0-0.5 h; dead, 0.5-1 h; 20.0°C	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
2,3,4-trichlorophenol	Crangon septemspinosa (shrimp)	SB,SW,Ls	2.0	96 h-LT; 10°C	McLeese et al (1979)
2,3,6-trichlorophenol	Astacus fluviatilis (crayfish)	SB,FW,LS	5.4	8 d - LC_{50}; pH 6.5; 13°C	Kaila & Saarikoski (1977)
			19.0	8 d - LC_{50}; pH 7.5; 13°C	
			38.0-39.0	LD_{50}	
2,3,6-trichlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	2.7	96 h-LT; 10°C	McLeese et al (1979)
2,4,5-trichlorophenol (Dowicide 2)	Salmo gairdneri (rainbow trout)	SB,FW,LS	5.0	Dead, 0.5 -2h; 12.8°C	Applegate et al (1957)
			1.0	"Ill.", 4 h	
			0.1	No effect, 24 h	
	Lepomis macrochirus (bluegill)		5.0	Dead, 2 h; 12.8°C	
			1.0	No effect, 24 h	
	Petromyzon marinus (larvae) (sea lamprey)		5.0	Dead, 3 h; 12.8°C	
			1.0	No effect, 24 h	
2,4,5-trichlorophenol (Dowicide 2)	Ptychocheilus oregonensis (northern squawfish)	SB,FW,LS	10.0	Dead, 0-1 h; 10.6°C	McPhee & Ruelle (1969)
	Oncorhynchus tshawytscha (chinook salmon)		10.0	Dead, 0-1 h; 10.6°C	
	Oncorhynchus kisutch (coho salmon)		10.0	Dead, 0-1 h; 10.6°C	
2,4,5-trichlorophenol	Mya arenaria (soft-shelled clam)	SB,SW,LS	2.4	96 h-LT; 10°C	McLeese et al (1979)
2,4,6-trichlorophenol	Mya arenaria (soft-shelled clam)	SB,SW,LS	3.9	96 h-LT; 10°C	McLeese et al (1979)
2,4,6-trichlorophenol	Pimephales promelas (fathead minnow)	SB,FW,LS	0.1-1.0	96 h - TL_m	Barnhart & Campbell (1972)

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
2,3,4,6-tetra-chlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	11.8	96 h-LT; 10°C	McLeese et al (1979)
	Mya arenaria (soft-shelled clam)		11.8	96 h-LT; 10°C	
Sodium 2,3,4,6-tetrachlorophenate (Dowicide F)	Lymnaeid snails	SB,FW,LS	1.67	100% kill in 24 h	Batte et al (1951)
Pentachlorophenol	Macrocystis pyrifera (kelp)	SW,LS	2.66	Eliminated all photosynthesis in 4 d	Clendenning (1959)
			1.0	Eliminated photosynthesis in 2 d	
Pentachlorophenol	Lepomis cyanellus (green sunfish)	SB,FW,LS	20.0	Repelled, distress	Summerfelt & Lewis (1967)
			5.0	Not repelled, but distressed	
Pentachlorophenol	Lepomis macrochirus	SB,FW,LS	0.05	24 h - LC_{50}; soft water; Ave. 2 series; 24°C	Inglis & Davis (1972)
	(bluegill)		0.04	24 h - LC_{50}; med. hard water; Ave. 2 series; 24°C	
			0.05	24 h - LC_{50}; hard water; Ave. 2 series; 24°C	
			0.03	48 h - LC_{50}; soft water 24°C	
			0.03	48 h - LC_{50}; med. hard water; 24°C	
			0.04	48 h - LC_{50}; hard water; 24°C	
			0.02	96 h - LC_{50}; hard water; 24°C	
	Carassius auratus (goldfish)		0.17	48 h - LC_{50}; soft water; 24°C	
			0.08	48 h - LC_{50}; med. hard water; 24°C	
			0.11	48 h - LC_{50}; hard water 24°C	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Pentachlorophenol	Carassius auratus (goldfish)	SB,FW,LS	0.08	72 h - LC_{50}; soft water 24°C	Inglis & Davis (1972)
			0.07	72 h - LC_{50}; med. hard water; 24°C	
			0.06	72 h - LC_{50}; hard water; 24°C	
			0.06	96 h - LC_{50}; soft water; 24°C	
			0.06	96 h - LC_{50}; med. hard water; 24°C	
			0.05	96 h - LC_{50}; hard water; 24°C	
Pentachlorophenol	Crassostrea virginica (Eastern oyster)	SB,SW,LS	0.25	48 h - TL_m (eggs)	Davis and Hidu (1968) in Kemp et al (1973)
		SB,SW,LS	0.07	14 d - TL_m (larvae)	
Pentachlorophenol	Ictalurus punctatus (fingerling catfish)	LS	0.12	24 h - LD_{50} (reagent grade PCP)	Cliburn (1975)
			0.14	24 h - LD_{50} (commercial grade PCP)	
Pentachlorophenol	Carassius auratus (goldfish)	CB,FW,LS	0.27	24 h - LC_{50}	Adelman & Smith (1976) and Adelman et al (1976)
			0.22	96 h - LC_{50}	
	Pimephales promelas (fathead minnow)		0.22	24 h - LC_{50}	
			0.21	96 h - LC_{50}	
Pentachlorophenol	Pimephales promelas (fathead minnow) (juvenile)	SB,FW,LS	8.0	1 h - LC_{50} 18 - 22°C; pH 5.9	Mattson et al (1976)
			0.6	24,48,72, and 96 h - LC_{50}; 18 - 22°C pH 5.9	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Pentachlorophenol	Astacus fluviatilis (crayfish)	SB,FW,LS	9.0	8 d - LC_{50}; pH 6.5; 13°C	Kaila & Saarikoski (1977)
			53.0	8 d - LC_{50}; pH 7.5; 13°C	
			26.0	LD_{50}	
Pentachlorophenol	Cyprinodon variegatus (sheepshead minnow)	CB,SW,LS	0.44	96 h - LC_{50}	Parrish (1977)
Pentachlorophenol (analytical grade)	Cyprinodon variegatus (sheepshead minnow)	SB,SW,LS	0.329	96 h - LC_{50} 1 d old fry, 30°C	Borthwick & Schimmel (1978)
			0.392	96 h - LC_{50} 14 d old fry, 30°C	
			0.240	96 h - LC_{50} 28 d old fry, 30°C	
			0.223	96 h - LC_{50} 42 d old fry, 30°C	
Pentachlorophenol	Salmo gairdneri (Rainbow trout)	SB,FW,LS	0.13	96 hr - LC_{50} Juveniles (av.wt. 2.7 g), 14-15°C, pH 7.5-8.0, dechlorinated city water	Guo et al (1979)
Pentachlorophenol	Crangon septemspinosa (shrimp)	SB,SW,LS	3.3	66 h-LT; 10°C	McLeese et al (1979)
Sodium pentachlorophenate	"algae"	FW,FS	15.0	Growth stopped in 7 d	Gelfand (1941)
			20.0	Growth stopped immediately	
Sodium pentachlorophenate	Ericymba buccata (silverjaw minnow)	SB,FW,LS	5.0	Survived 23 min	Goodnight (1942)
			1.0	Survived 105 min	
			0.2	Alive after 3 d	
	Notropis umbratilis (redfin shiner)		5.0	Survived 16 min	
			1.0	Survived 100 min	
			0.2	Alive after 3 d	
	Pimephales notatus (bluntnose minnow)		5.0	Survived 42 min	
			1.0	Survived 147 min	
			0.2	Alive after 3 d	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Campostoma anomalum (stoneroller)	SB,FW,LS	5.0 1.0 0.2	Survived 13 min Survived 58 min Alive after 3 d	Goodnight (1942)
	Notropis whipplii (steelcolor shiner)		5.0 1.0 0.2	Survived 15 min Survived 65 min Alive after 3 d	
	Semotilus atromaculatus (creek chub)		5.0 1.0 0.4	Survived 30 min Survived 105 min Alive after 3 d	
	Fundulus notatus (blackstripe topminnow)		5.0 1.0 0.6	Survived 90 min Survived 435 min Alive after 3 d	
	Lepomis humilis (orangespotted sunfish)		5.0 1.0 0.2	Survived 25 min Survived 165 min Alive after 3 d	
	"tadpoles" (Rana pipiens)		5.0 1.0 0.6	Survived 75 min Survived 375 min Alive after 3 d (All data at pH 7.6, 16°C; temperatures of 9-24°C did not appear to effect toxicity.)	
	"bloodworms" (Chironomidae)		5.0	Killed	
	"crayfish" (Cambarus virilis)		5.0	Survived	
	"amphipods" (Hyalella) knickerbockeri)		5.0	Survived	
	"cladocera" (Daphnia pulex)		5.0	Survived	
	"dragonfly nymphs" (Epicordulia sp.)		5.0	Survived	
	"damselfly nymphs" (Ischnura sp.)		5.0	Survived	
	"isopods" (Asellus communis)		5.0	Survived	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Cristivomer namaycush (lake trout)	SB,FW,LS	---	Eggs were more resistant than mature fish	Goodnight (1942)
	"fish" (19 species tested)		0.2-0.6	Lethal range	
Sodium pentachlorophenate (Santobrite)	Crassipes eichornia (water hyacinth)	SB,FW,FS	5.0	Minimum to affect appearance	Hirsch (1942)
			80.0	Dose for complete kill	
Sodium pentachlorophenate (Santobrite)	Notropis spilopterus (spotfin shiner)	SB,FW,LS	5.0 1.0 0.4 0.4	18 min-TLm 74 min-TLm 234 min-TLm Critical concentration.	Van Horn (1943)
	Pimephales notatus (bluntnose minnow)		5.0 1.0 0.4 0.3	21 min-TLm 80 min-TLm 248 min-TLm Critical concentration.	
	Notropis atherinoides (emerald shiner)		5.0 1.0 0.4 0.2	16 min-TLm 87 min-TLm 418 min-TLm Lived, the critical concentration. (All tests at 18-20°C, tap water.)	
Sodium pentachlorophenate	"fish"	FW,FS	0.2	Toxic concentration	Fleming (1946)
Sodium pentachlorophenate (Santobrite)	"Mussel, anemones, barnacles"	SB,SW,LS	0.1 1.0	Ineffective Killed all organisms, 3 d	Turner et al (1948)
		CB,SW,LS	10.0	Killed all anenomes, tunicates, and bryozoa in 1 d; all barnacles in 3 d, all mussels in 5 d	
			1.0	Killed all tunicates and bryozoa in 1 d; all anemones, mussels and barnacles in 4 d	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Australorbis glabratus (snail)	FW,FS (stream)	9.5	Killed 95-100%, 2.4 km dowstream in 6 h flowrate dose	Berry et al (1950)
	"catfish, eels, guppies		9.5	Lethal under same conditions	
	"crayfish"		9.5	No effect under same conditions	
Sodium pentachlorophenate (88%)	Lymnaeid snails	SB,FW,LS	2.5	100% mortality in 24 h	Batte et al (1951)
Sodium pentachlorophenate	Kuhlia sandvicensis (marine Kuhliidae)	SW,LS	20.0	Medium reaction, 2 min exposure	Hiatt et al (1953)
Sodium pentachlorophenate (Santobrite)	Lepomis macrochirus (bluegill)	SB,FW,LS	0.35 0.10	24 & 48 h-TLm, acute 20°C "Safe concentration."	Turnbull et al (1954)
Sodium pentachlorophenate	Australorbis sp. (snail)	SB,FW,LS	1.0 2.5	Killed 94%, 48 h Killed 100%, 48 h	Vallejo-Freire et al (1954)
Sodium pentachlorophenate (Santobrite)	Notropis atherinoides (emerald shiner)	SB,FW,LS	0.2	Minimum lethal dose	Van Horn & Balch (1955)
Pentachlorophenol sodium salt (Dowicide G)	Notropis atherinoides (emerald shiner)	SB,FW,LS	0.5	Minimum lethal concentration, 100% survival in 120 h	Van Horn & Balch (1955)
			0.1	31% survival, mean survival time 102 h	
			0.5	100% kill, mean survival time 7.6 h	
Sodium pentachlorophenate (75%)	Cylindrospermum licheniforme (blue-green algae)	SB,FW,LS	2.0	Toxic, 3 d. (Toxic = growth relative to control in culture	Palmer & Maloney (1955)
	Microcystis aeruginosa (blue-green algae)		2.0	Toxic, 3 d	

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate (75%)	Scenedesmus obliquus (green algae)	SB,FW,LS	2.0	Partially toxic, 3 & 7 d	Palmer & Maloney (1955)
	Chlorella variegata (green algae)		2.0	Not toxic	
	Gomphonema parvulum (diatom)		2.0	Partially toxic, 3 & 7 d	
	Nitzschia palea (diatom)		2.0	Toxic, 3 d	
Sodium pentachlorophenate	Lebistes reticulatus (guppy)	FW,LS	2.0 4.0 8.0 15.0 25.0	Killed 94%, 1450 min Killed 100%, 200 min Killed 100%, 90 min Killed 100%, 40 min Killed 100%, 25 min	Klock (1956)
Sodium pentachlorophenate	"snails"	FW,FS	15-20	Controlled	Rudd & Genelly (1956)
	"centrarchid fishes"		6.0	Toxic, violent reactions after 10-20 min	
	"catfish"		6.0	Effect, but surfaced only after the majority of other fish had succumbed	
	"Aquatic insects"		15.0	Some killed	
Sodium pentachlorophenate (Dowicide G)	Salmo gairdneri (rainbow trout)	SB,FW,LS	5.0 1.0 0.1	Dead, 1 h; 12.8°C Dead, 4 h No effect, 24 h	Applegate et al (1957)
	Lepomis macrochirus (bluegill)		5.0 1.0 0.1	Dead, 3 h; 12.8°C Dead, 8 h No effect, 24 h	
	Petromyzon marinus (larvae) (sea lamprey)		5.0 1.0 0.1	Dead, 3 h; 12.8°C Dead, 4 h No effect, 24 h	
Sodium pentachlorophenate	Pomoxis annularis (white crappie)	FW,LS	.056-.075	"Caused losses"	Springer (1957)

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Lebistes reticulatus (guppy)	FW,LS	1.0	"Caused losses"	Springer (1957)
	"catfish"		9.5	Killed	
	Lebistes reticulatus (guppy)		9.5	Killed	
	"eels"		9.5	Killed	
	"crayfish"		9.5	Unharmed	
Sodium pentachlorophenate	Ictalurus punctatus (fingerlings)	SB,FW,LS	0.46	24 to 96 h-TLm, acute; 25°C	Clemens & Sneed (1959)
	(channel catfish)		1.5	4 h-TLm, acute; 25°C	
			5.4	1 h-TLm, acute; 25°C	
Sodium pentachlorophenate	Pimephales promelas (fathead minnow)	SB,FW,LS	0.32-0.35	24 h-TLm; pH 8.0, 15°C; as pH was reduced, or as temperature increased, the toxicity increased	Crandall & Goodnight (1959)
			1.0	260 min mean survival time; 10°C, pH 7.5-7.6	
			1.0	81 minmean survival time; 18°C, pH 7.5-7.6	
			1.0	46 min mean survival time; 26°C, pH 7.5-7.6	
Sodium pentachlorophenate (Santobrite)	Macrocystis pyrifera (kelp)	CB,SW,LS	0.3	50% inactivation of photo-synthesis in 4 d	Clendenning & North (1960)
Sodium pentachlorophenate	Lebistes reticulatus (guppy)	SB,FW,LS	0.5	44.6% killed, 90 min	Crandall & Goodnight (1962)
Sodium pentachlorophenate	Oncorhynchus kisutch (coho salmon)	SB,FW,LS	3.0	Median resistance times at different combinations of salinity and temperature are given.	Alderdice (1963)

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Mylilus edulis (bay mussel)	SW,LS	0.20	12% abnormal embryos, 28 ppt salinity	Dimick & Breese (1965)
			0.20	21% abnormal embryos, 24 ppt salinity	
			0.30	17.6% abnormal embryos, 28 ppt salinity	
			0.30	33.6% abnormal embryos, 24 ppt salinity	
			0.4	22.1% abnormal embryos, 28 ppt salinity	
			0.4	69.1% abnormal embryos, 24 ppt salinity	
Sodium pentachlorophenate	Australorbis glabratus (snail)	SB,FW,LS	2.0 12.0	6 h-TLm, acute 95% kill, 6 h	Seiffer & Schoof (1967)
Sodium pentachlorophenate	"tubificid worms"	SB,FW,LS	0.31 0.67 1.40	24 h-TLm; pH 7.5, 20°C 24 h-TLm; pH 8.5, 20°C 24 h-TLm; pH 9.5, 20°C	Whitley (1968)
Sodium pentachlorophenate (Santobrite)	Salmo gairdneri (steelhead trout)	SB,FW,LS	0.25	60 h - LC_{50}; yearlings; 10°C	Chapman (1969)
	Salmo gairdneri (rainbow trout)	CB,FW,LS	0.17	48 h - LC_{50}; 3 - 12 month old trout; 18°C	
	Salmo trutta (brown trout)		0.17	48 h - LC_{50}; 3 - 12 month old trout; 18°C	
Sodium pentachlorophenate	"fish"	FW,LS	0.06	Can be lethal under laboratory conditions	Walker (1969)
Sodium pentachlorophenate	Salmo gairdneri (rainbow trout)	CB,LS	0.15	48 h - TL_m	Alabaster (1969) in Kemp et al (1973)
Sodium pentachlorophenate	Carassius auratus (goldfish)	FW,FS	---	Residues detrimental to early developmental life stages, causing heavy mortality and teratogensis	Lennon et al (1970)
Sodium pentachlorophenate	Crassostrea gigas (oyster)	SW,LS	0.027	4.3% abnormal embryos in 48 h	Woelke (1972)

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TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Crassostrea gigas (oyster)	SW,LS	0.069	72.4% abnormal embryos in 48 h	Woelke (1972)
			0.11	100% abnormal embryos in 48 h	
Sodium pentachlorophenate	Salmo gairdneri (Rainbow trout)	SB, LS, pH7.0	0.098	96 h - LC_{50} 12°C	Davis and Hoos (1975) (nomographic calc.)
		SB, LS, pH7.0	0.096	96 h - LC_{50} 12°C	
		SB, LS, pH7.0	0.050	96 h - LC_{50} 11°C	
		SB, LS, pH7.1	0.106	96 h - LC_{50} 12°C	
		SB, LS, pH5.7	0.047	96 h - LC_{50} 10°C	
	Oncorhynchus kisutch (coho salmon)	SB, LS, pH7.0	0.092	96 h - LC_{50} 10°C	
		SB, LS, pH7.0	0.032	96 h - LC_{50} 11°C	
	Oncorhynchus nerka (sockeye salmon)	SB, LS, pH7.2	0.050	96 h - LC_{50} 13°C	
		SB, LS, pH7.7	0.130	96 h - LC_{50} 8°C	
Sodium pentachlorophenate	Pimephales promelas (fathead minnow)	CB,FW,LS	0.21	48 h - LC_{50}; 15°C	Ruesink & Smith (1975)
			0.21	96 h - LC_{50}; 15°C	
			0.37	48 h - LC_{50}; 25°C	
			0.34	96 h - LC_{50}; 25°C	
			0.21	Lethal threshold concentration (LTC) 15°C	
			0.33	LTC; 25°C	
Sodium pentachlorophenate	Salvelinus fontinalis (brook trout) (adult)	CB,FW,LS	0.32	24 h - LC_{50}	Cardwell et al (1976)
			0.12	219 h - LC_{50}	
	Pimephales promelas (fathead minnow) (juvenile)		0.34	21 h - LC_{50}	
			0.15	336 h - LC_{50}	
	Carassius auratus (goldfish) (adult)		0.37	21 h - LC_{50}	
			0.19	336 h - LC_{50}	
	Lepomis macrochirus (bluegill) (juvenille)		0.30	30 h - LC_{50}	
			0.22	336 h - LC_{50}	

SB = Static Bioassay, CB = Constant-flow Bioassay, FW = Freshwater, SW = Sea (Salt) Water, LS = Lab Study, FS = Field Study

TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate	Palaemonetes pugio (grass shrimp)	SB,SW,LS	2.63	96 h - LC_{50}; intermolt	Conklin & Rao (1977)
			2.74	96 h - LC_{50}; early premolt	
			0.44	96 h - LC_{50}; molt	
Sodium pentachlorophenate	Crangon crangon (a marine decapod)	SB,SW,LS	1.79	96 h - LC_{50}; adult; 15°C	van Dijk et al (1977)
			0.11	96 h - LC_{50}; larvae; 15°C	
	Palaemon elegans (a marine decapod)		10.39	96 h - LC_{50}; adult; 15°C	
			0.08	96 h - LC_{50}; larvae; 15°C	
	Palaemonetes varians (a brackish water decapid)	SB,LS	5.09	96 h - LC_{50}; adult; 15°C	
			0.36	96 h - LC_{50}; larvae; 15°C	
Sodium pentachlorophenate (analytical grade)	Lagodon rhomboides (pinfish)	CB,SW,LS	0.053	96 h - LC_{50}	Schimmel et al (1978)
		SB,SW,LS	0.038	96 h - LC_{50}; 48-h prolarvae; 20°C	Borthwick & Schimmel (1978)
	Mugil cephalus (striped mullet)	CB,SW,LS	0.112	96 h - LC_{50}	Schimmel et al (1978)
	Fundulus similis (longnose killifish)		> 0.306	96 h - LC_{50}	
	Penaeus aztecus (brown shrimp)		> 0.195	96 h - LC_{50}	
Sodium pentachlorophenate (analytical grade)	Crassostrea virginica (eastern oyster)		0.076	192 h - EC_{50}	
		SB,SW,LS	0.040	48 h - EC_{50}; developing embryos; 25°C	Borthwick & Schimmel (1978)
	Palaemonetes pugio (grass shrimp)		0.649	96 h - LC_{50}; 24-h larvae; 25°C	

SB = Static Bioassay, CB = Constant-flow Bioassay, FW = Freshwater, SW = Sea (Salt) Water, LS = Lab Study, FS = Field Study

TABLE A4-3 (Cont'd) TOXICITY OF CHLOROPHENOLS TO AQUATIC BIOTA (Adapted from Becker and Thatcher, (1973), with additions)

Chemical Compound	Test Organism	Test Conditions	Concentration (ppm)	Remarks	Reference
Sodium pentachlorophenate (analytical grade)	Palaemonetes pugio (grass shrimp)	CB,SW,LS	> 0.515	96 h-LC_{50}	Schimmel et al (1978)
Sodium pentachlorophenate (Dowicide G)	Lagodon rhomboides (pinfish)	SB,SW,LS	0.066	96 h - LC_{50}; 48h-prolarvae	Borthwick Schimmel (1978)
	Cyprinodon variegatus (sheepshead minnow)		0.516	96 h - LC_{50} 14 d old fry	
Sodium pentachlorophenate	Oncorhynchus tschawytscha (chinook salmon)	CB,FW, LS pH7.0	0.078	96 h - LC_{50} 12°C juvenile	Iwama & Greer (1979)

SB = Static Bioassay, CB = Constant-flow Bioassay, FW = Freshwater, SW = Sea (Salt) Water, LS = Lab Study, FS = Field Study

and for the larvae 0.11 ppm, 0.08 ppm and 0.36 ppm, respectively. The effect of NaPCP vs moulting was investigated by van Dijk et al (1977) using larvae of P. elegans. The period of moulting had no significant effect on the sensitivity of P. elegans larvae to NaPCP. The effect of temperature on sensitivity of adults of the brackish water decapod, P. varians, to NaPCP was studied. Toxicity seems to increase with temperature over a base of 15°C., roughly by a factor of 2 for each 10°C. P. varians adults were also less sensitive to NaPCP in sea water diluted to 30% as recorded at 96 h, than in sea water at either 100% or 70% (van Dijk et al, 1977). If observations on adult mortality in the 30% sea water were carried through to 192 h then there was a slight increase, bordering on significance, of mortality, but it was still significantly lower than after 96 h in 70% sea water.

Kaila and Saarikoski (1977) investigated the toxicity of PCP and 2,3,6-TCP to the crayfish, Astacus fluviatilis L., at two pH levels 6.5 and 7.5, in static tests. The eight day LC_{50} estimates obtained were 53 ppm for PCP and 19 ppm for TCP, at pH 7.5 (13°C). Lowering the pH to 6.5 increased the toxicity of PCP by a factor of 5.9 and that of TCP by 3.5. The 96-h LC_{50} values @ pH 6.5 for PCP and TCP were 9.0 ppm and 5.4 ppm, respectively. The authors stated that both factors were significantly smaller than could be expected if only the concentration of the non-ionized chlorophenol molecules were essential. The suggested explanations offered to account for the difference between expected and actual toxicity factors were that the toxicities measured by the LC_{50} values were also affected by the ionic concentration, or that the resistance of the crayfish was influenced by the pH level (Kaila and Saarikoski, 1977). On a weight basis and at pH 6.5 TCP was more toxic than PCP. On the other hand, the LD_{50} estimates, obtained by injection, were lower for PCP than for TCP, 26 and 38 ppm respectively. The authors suggested that PCP may penetrate more rapidly into the crayfish, and they further stated that this was in agreement with the smaller degree of ionization of TCP due to its higher pK_a value. The pK_a of PCP is 5.00 and that of 2,3,6-TCP is 5.98 (Farquharson et al, 1958) (Table A1-1).

Schimmel et al (1978) developed, in flow-through tests, NaPCP, 96-h LC_{50} toxicity data for three estuarine invertebrate animals. Two organisms and their toxicity values were grass shrimp (Palaemonetes pugio), >515 µg/L, and brown shrimp (Penaeus aztecus), >195 µg/L. In addition they noted that the 192-h EC_{50} (effect measured was shell deposition) for the eastern oyster (Crassostrea virginica) was 76.5 µg/L.

McLeese et al (1979) developed lethal threshold (LT) data for shrimp (Crangon septemspinosa) and soft-shelled clam (Mya arenaria) exposed to various CPs (Table A4-3). It was stated that "The 96-h threshold was taken as the geometric mean of the highest concentration with no deaths and the next higher concentration (step by a factor of 2) at which all 3 test animals died. A lethal threshold was recognized before 96 h with several of the compounds." McLeese et al (1979) noted that when tests were conducted on the clams, that mono-CPs and PCPs caused no mortalities, but deaths occurred with DCPs, TCPs, and TTCPs. This suggested to McLeese et al (1979) that there may be lower and upper limits on the ability of clams to sense and avoid structurally related chemicals by closing, and that this subject would warrant further investigation.

4.1.3 Vertebrates

Eel

Holmberg et al (1972) have detailed the effect of technical grade Na-PCP on the eel, Anguilla anguilla, when exposed to levels of 0.1 ppm PCP in sea water at pH 8.1 for 8 days and PCP in freshwater at pH 7.1 for 4 days. The authors stated that PCP exposure caused changes which indicated a hypermetabolic state with accelerated utilization of tissue energy reserves. These effects seemed to persist in spite of a recovery period in clean water for about 2 months.

Boström and Johansson (1972) examined the effect of PCP on enzyme activity in the liver of eel, Anguilla anguilla. In a comparison of enzyme activity ratios in-vivo, a shift was noted between different metabolic pathways; also, in-vitro experiments showed that PCP inhibited all the investigated enzymes.

Fish

Gersdorff and Smith (1940) investigated the toxicity of phenol and the three isomeric mono-CPs to goldfish, Carassius auratus. By utilitizing the minimal product of the concentration and survival time, the authors calculated the relative toxicity of the CPs as compared with phenol as follows: ortho, 1.15; meta, 1.51; and para, 1.89.

Inglis and Davis (1972) determined that water hardness, within the range found in fish-bearing waters of the United States, had no significant effect on the toxicity of PCP as measured in 96 h static bioassay tests run in 2 series, to bluegill sunfish, Lepomis macrochirus, or to goldfish, Carassius auratus. (Table A4-3). The test waters, which contained calcium/magnesium ion ratios of 1:1 and 5:1, had concentrations of total

hardness (calculated as $CaCO_3$) of 13.0 (very soft), 52.2 (soft), 208.7 (medium), and 365.2 (hard) ppm.

The following examples illustrate the diverse results in bioassay tests reported in the literature. Mattson et al (1976) determined, in short term static bioassay tests, that the 96-h LC_{50} of PCP diluted in Lake Superior water at pH 5.9, to juvenile fathead minnows, Pimephales promelas, was 0.6 mg/L (Table A4-3). On the other hand, Adelman et al (1976) reported the 96-h LC_{50} for technical grade NaPCP to fathead minnows, Pimephales promelas, and goldfish, Carassius auratus, as 0.21 and 0.22 mg/L, respectively, in flow through tests at 25°C, and pH 7.4 - 7.8 (Table A4-3). They also reported that the threshold LC_{50} for PCP for these species, 0.21 mg/L, was reached in 6 days in flow through tests. Adelman et al (1976) stated that, "Tests were conducted for 11 days, or until no mortality occurred for 2 days, whichever came first. Two days with no deaths was used as the criterion for achieving a threshold LC_{50}." In the threshold LC_{50} tests, goldfish were initially more resistant to PCP than fathead minnows, but by termination there was no significant difference in LC_{50}'s between the two species. Data from this bioassay study, in which PCP was compared with 3 other toxicants, was used by Adelman and Smith (1976) to recommend the fathead minnow as the standard fish species for bioassay tests. The fathead minnows were primarily selected on the basis of their relatively constant response to a broad range of toxicants when tested under similar conditions, their small size, and their capability for use in complete life cycle tests.
The authors also stated that standard bioassay test results will probably continue to be reported as LC_{50}'s on a fixed time basis (i.e. 24-h LC_{50} or 96-h LC_{50}) (Table A4-3).

Ruesink and Smith (1975) investigated the relationship of the 96-h LC_{50} to the Lethal Threshold Concentration (LTC) with NaPCP and fathead minnow in short term laboratory bioassays at two temperatures. They stated that the LTC was probably a better descriptor for the toxicity of a compound than an LC_{50} for an arbitrary time period since it represents a biologically defined end-point. For NaPCP, the LTC was approximately the same as the 96-h LC_{50} and both concentrations were equally affected by temperature (i.e. an increased LC_{50} or LTC value with an increase in temperature) (Table A4-3). Ruesink and Smith (1975) defined their terminology as follows: "The lethal threshold was reached when 50% of the fish survived, and no additional mortality occurred during a further 20% extension of exposure time. . . . The time to the beginning of the period of 0 mortality is the time to lethal threshold. The median lethal concentration for this time is the lethal threshold concentration." Ruesink and Smith (1975) recommended

that ". . . acute bioassays not be terminated until the lethal threshold concentration has been determined, and that safety factors which extrapolate from acute lethal levels to no effect levels should be applied to the lethal threshold concentration."

Norup (1972) investigated the toxicity of slime control agents used in paper processing and determined that the threshold concentration (7-d LC_{50}) of NaPCP for guppies, Lebistes reticulatus, was near 2 ppm. The active ingredients for the static bioassay tests were made up from a commercial NaPCP formulation containing 79% NaPCP and 11% Na salts of other CPs, diluted in tap water with the hardness corresponding to 358 ppm $CaCO_3$, at pH 7.6 - 7.8, and at 24°C. Norup (1972) observed that some guppies which had previously been acclimated to 1.0 and 0.1 ppm NaPCP for 10 days were able to survive for 3 - 8 h in a concentration of 5.0 ppm NaPCP, which is lethal to non-acclimated fish. Norup (1972) also compared the efficacy of NaPCP as a slimicidal agent with mercury containing agents and concluded that NaPCP was less efficient as a controller of slime organisms but was equally toxic to fish. Norup (1972) also conjectured that resistance by fish to sublethal levels of NaPCP may ". . . lead to increased tolerance of accumulated PCP in the organism where severe metabolic distortions, delayed sexual maturity and increased mortality may result."

Strufe (1968) in a review article on molluscicides reported that Klock (1956) made use of the high susceptibility of guppies (Lebistes reticulatus) for making determinations of active ingredient after field application of NaPCP. Vaughn (1954), as reported by Strufe (1968), observed in field trials of NaPCP for control of molluscs in the Dominican Republic that in flowing water, three species of fish, Gobiomoris durmitor, Awavus taiasica, and Agnostomus monticola, were killed with a NaPCP concentration of 15 ppm, but in stagnant water, 2 ppm was sufficient to kill the fish.

Cliburn (1975) stated that because of the low solubility of PCP in water (17 ppm), the majority of the fish toxicity data has been developed using the water soluble NaPCP (Table A1-2). He therefore determined that the 24-h LD_{50} value for reagent grade PCP to fingerling catfish, Ictalurus punctatus, was 0.12 ppm and for commercial grade PCP was 0.14 ppm.

Trabalka and Burch (1977) utilized carp embryos and Daphnia pulex to assess the toxicity of a number of water soluble chlorinated organic compounds in chlorinated cooling waters from electric power stations and from chlorinated effluents from domestic sanitary treatment plants. Their work indicated low or moderate toxicity (96-h LC_{50}; 10 - 100 mg/L) for most of the water soluble chlorinated organic compounds identified to

date. They stated that the toxicity observed in aquatic organisms exposed to synthetic mixtures of identified chlorinated organics could be attributed primarily to the content of CPs.

Goodnight (1942) investigated the toxicity of NaPCP (ai. 90.5%) to several species of fish and invertebrates (Table A4-3). Known impurities in the NaPCP consisted primarily of Na salts of other chlorophenols and neutral salts. NaPCP concentrations above 0.2 ppm were fatal to the more sensitive species of fish such as the silverjaw minnow (=silver-mouthed minnow), Ericymba buccata, although hardier species, such as creek chub, Semotilus atromaculatus, and blackstripe topminnow, Fundulus notatus, survived at 0.4 and 0.6 ppm, respectively (Table A4-3). Goodnight (1942) noted that lethal concentrations of NaPCP and PCP increased the metabolism of fish, as shown by increased respiratory movements and increased blood pressure as evidenced in rupture of the smaller capillaries with bleeding occurring about the gills, mouth, and pectoral regions. The toxicity of NaPCP, indicated by fish survival time, increased as the pH was lowered. Goodnight (1942) also observed that silver-mouthed and blunt-nosed minnows, Campostoma anomalum, were able to detect and avoid NaPCP at concentrations above 10 ppm but not below 5 ppm. Eggs of lake trout, Cristivomer namaycush, were noted as being more resistant than mature fish to NaPCP, although the developmental stage of the egg was not given. Newly hatched young in the yolk sac stage were more sensitive than either the eggs or more mature lake trout. Aquatic invertebrates used by fish as food were relatively insensitive to NaPCP at 5.0 ppm (Table A4-3).

Cardwell et al (1976) determined the median lethal threshold (MLT) concentration of NaPCP for brook trout as 0.118 mg/L after 219 h exposure, compared to an acute toxicity value of LC_{50} of 0.315 mg/L at 24 h (Table A4-3). MLT is that concentration of NaPCP when acute toxicity ceases to 50% of the test specimens. Acute toxicity tests utilizing an intermittent flow bioassay system indicated that the MLT for trout and the other three species of fish tested - fathead minnow, goldfish, and bluegill - was at least 0.11 mg NaPCP/L, which the authors stated was one-half or less than the values stated in much of the earlier literature. But the authors also stated that this was somewhat higher than the MLT of 0.057 mg NaPCP/L and levels of 0.00174 to 0.0018 mg/L affecting growth and food conversion efficacy in 50% of the underyearling sockeye salmon, Oncorhynchus nerka, exposed in a continuous flow system as reported by Webb and Brett (1973). The 96-h LC_{50} was determined as 63 ppb PCP when tests were performed at 15°C, pH 6.8, and dissolved oxygen values of 90-100% air saturation (Webb and Brett, 1973).

Iwama and Greer (1979) determined that the incipient 96 h LC_{50} of NaPCP for juvenile chinook salmon (Oncorhynchus tschawytscha) at a loading density of 13.8 g/L (g fish/unit volume test water) was 0.078 mg/L; the 95% confidence limits were 0.057 and 0.110 mg/L (Table A4-3). Iwana and Greer (1979) stated that the NaPCP 96 h LC_{50} obtained in their continuous-flow bioassay lay in the midrange of 96 h LC_{50} values determined for related species of juvenile salmonids by static methods in an inter-laboratory bioassay standardization study reported by Davis and Hoos (1975) (Table A4-3). In this latter study a loading density of 0.5 g/L (a maximum recommended loading) was used and the range of NaPCP toxicity for the different species were: rainbow trout 0.047 - 0.106 mg/L; coho salmon 0.032 - 0.092 mg/L; sockeye salmon 0.05 - 0.130 mg/L.

In young Coho salmon, Onchorynchus kisutch, exposed to 0.1 mg of KPCP/L, in a flow-through system (the 24-h LC_{50} for KPCP in the salmon was 0.15mg/L), Hanes et al (1968) noted a net loss of 22% of available fatty acids through a higher catabolic rate compared to the untreated controls. They also stated that "The excess net catabolism of each fatty acid in salmon under KPCP poisoning was directly proportional to the available mass of that fatty acid."

Krueger et al (1968) examined the bioenergetic aspects of KPCP poisoning in cichlid fish. When Cichlasoma bimaculatum were exposed to the non-lethal level of 0.2 ppm of KPCP at 25°C, the intake of food was increased and energy losses were also increased. Growth was decreased. They further observed that the cost of specific dynamic action was higher and the cost of exercise was increased above the cost of similar exercise in non-poisoned controls.

Chapman and Shumway (1978) conducted a study to determine the effects of technical grade NaPCP on the early developmental stages of the steelhead trout (Salmo gairdneri) and reported:

> "In an experiment where embryos were exposed to Na-PCP from fertilization to hatching, 100% mortality occurred within one week after fertilization at concentrations down to 300 ppb (µg/L), within 24 hours posthatch, 100% mortality occurred down to 50 ppb of Na-PCP. Alevin dry weight at hatch was decreased by exposure to Na-PCP and hatching was delayed. In 5-day tests, alevins usually died within 24 hours at concentrations down to 200 ppb, but little mortality occurred at lower concentrations.
>
> "Continuous exposure to Na-PCP from fertilization to complete yolk absorption produced 100% mortality at 40 ppb Na-PCP but little mortality at 20 or

10 ppb. However, in water containing 5 mg O_2/L, 20 ppb Na-PCP was 100% lethal and at 3 mg O_2/L, 10 ppb was 100% lethal. Little mortality occurred at these oxygen levels in the absence of Na-PCP. Oxygen consumption rates of alevins in 40 ppb Na-PCP were higher than those of control alevins. Exposure to Na-PCP reduced yolk utilization efficiency and growth. The bioenergetic data obtained in the study are consistent with the concept that PCP disrupts energy metabolism."

Three species of estuarine fish were exposed to NaPCP in flow-through toxicity tests (Schimmel et al, 1978). The test species and their 96-h LC_{50} values were: the longnose killifish (Fundulus similis), >306 µg/L; pinfish (Lagodon rhomboides), 53.2 µg/L; and striped mullet (Mugil cephalus), 112 µg/L.

Amphibians

Goodnight (1942) determined the toxicity of NaPCP to tadpoles, Rana pipiens, while investigating the sensitivity of 19 species of fish to NaPCP over a concentration range of 0.2 to 5.0 ppm. Compared to the minnows and sunfishes in the test, tadpoles were classed among the hardy species. They survived concentrations of 5.0 ppm NaPCP for 75 minutes which was only exceeded by the blackstripe topminnow, Fundulus notatus, which survived 5.0 ppm of NaPCP for 90 minutes (Table A4-3). Enigk and Duwel (1960), as reported by Strufe (1968), stated that tadpoles and adult frogs were quickly killed by NaPCP at molluscicidal concentrations. They had observed that most aquatic animals belonging to the gill-breathing group and which are devoid of or possess only a poorly developed cuticle, are particularly susceptible to the effect of NaPCP.

4.1.4 Toxicity of Chlorophenol Impurities to Aquatic Organisms. There is little information on the toxicity of PCDDs to aquatic organisms including fish. The lack of information may be due in part to the delayed response of fish to toxic effects from PCDDs and to the fact that most toxicity tests for fish do not go past 96 h. Some research has been done by Miller et al (1973) who investigated response to 2,3,7,8-TCDD in guppies (Poecilia reticulatum), coho salmon (Oncorhynchus kisutch), rainbow trout (Salmo gairdneri), and three aquatic invertebrates: a snail (Physa sp.), a worm (Paranais sp.), and mosquito larvae (Aedes aegypti). They noted that a difficulty in studying the toxicity of TCDD to fish was that the response to the chemical was delayed. Initial response to the chemical did not occur for 5 to 10 days after the beginning of the exposure period, and mortality often extended over a two month period. It is logical to

assume that a similar delayed response might occur in fish exposed to toxic levels of other PCDDs and might only show up in long-term chronic studies. Miller et al (1973) concluded from their studies with the most toxic dioxin, 2,3,7,8-TCDD, that effects of exposure for 24 - 96 h of young salmon to TCDD in water at levels greater than 23 ng/g were irreversible, and death resulted in 10 - 80 days. Duration of exposure was less important than level of exposure except as threshold response levels were approached. They stated that the critical exposure period may be somewhat less than 24 hours in static water toxicity tests in which TCDD concentration may change markedly with time. Low levels of TCDD reduced growth of young rainbow trout. TCDD at 0.2 ppb had no effect on pupation of mosquito larvae, but reduced the reproduction success of a pulmonate snail and an Oligochaete worm.

Norris and Miller (1974) reported that exposure of guppies, Poecilia reticulatum, to levels of 2,3,7,8-TCDD as low as 0.1 ppb for 120 h resulted in mortality 32 days later. They also noted that smaller fish were considerably more sensitive to TCDD than larger fish.

Beatty et al (1976) summarized their investigations of 2,3,7,8-TCDD administered interperitoneal in the American bullfrog, Rana catesbeiana, as follows:

> "Doses of TCDD as high as 1 mg/kg failed to have any significant effect upon survival or completion of metamorphosis in tadpole and doses of up to 500 µg/kg had no effect on survival of adult frogs. Histopathological examination of various tissues from the metamorphosed tadpoles and adult frogs failed to show any abnormalities".

As noted earlier in this review (Sect. 3.1), 2,3,7,8-TCDD has been determined in samples of CPs only in 2,4,5-TCP and 2,4,5-NaTCP (Firestone et al, 1972).

Hawkes and Norris (1977) reported the "no-effect" level for survival, growth, feeding activity, and fin erosion in rainbow trout (Salmo gairdneri) receiving 2,3,7,8-TCDD orally was between 2.3 ppm and 2.3 ppb.

In research on the CDFs, Zitko and Choi (1973) reported that when juvenile Atlantic salmon were fed for 140 days with dry fish food containing 2.7, 5.7, 2.8, and 9.1 µg/g wet weight of di-, tri-, tetra, and octa-CDF in their diet, only the OCDF was detected in the follow-up fish tissue analysis. The tissues analyzed consisted of muscle and gut of fish that died between the 81st and 135th day of the experiment, and of fish surviving for 140 days. Detection limits were 0.01 µg/g for the OCDF and 0.02 µg/g for the others. Mean levels (µg/g wet weight) of OCDF in muscle and gut of dead and live

fish were as follows: muscle: dead, 0.03; live, 0.01; gut: dead, 0.21; live, 0.02. The authors suggested that because of the high toxicity and low residual levels of CDFs, a more sensitive analytical method was required.

Zitko et al (1973) noted that 2,8-DCDF has a low acute toxicity to immature brook trout, since no mortality resulted after administrations by gelatin capsule of a single dose of 122 mg/kg. The authors suggest that the low acute toxicity and also the low residual level of 2,8-DCDF may be due to poor absorption of the compound in the gut and to its excretion in the form of conjugated hydroxy derivates (App. 5, Sect. 5.2).

4.2 Field Toxicology

4.2.1 Effects on Non-target Organisms

4.2.1.1 Primary effects. The virtual absence of documentation of effect of CPs on non-target organisms in the aquatic environment can be attributed to both lack of field research programs to develop such information and an apparent lack of proper follow-up investigative reporting of the known cases of contamination of the aquatic environment from CPs. The latter situation was suitably summed up in the "Royal Commission of Inquiry into the Use of Pesticides and Herbicides, May 30, 1975" in British Columbia (MacKenzie et al, 1975), as follows:

> "Hundred of isolated cases of fish, mammal and bird kills occur each year. According to the Fish and Wildlife Branch, however, it is estimated that only a small percentage of these cases are reported, investigated and documented. Even large scale die-offs, particularly of less popular species, may receive virtually no attention or investigation. Attempts by this Commission to obtain data concerning fish and wildlife mortalities, which occurred as a consequence of pesticide poisoning, revealed that such information is scant and difficult to obtain. There is no centralized source which compiles such information."

The assumption can be made this would apply not only to pesticides but also to industrial chemicals and that the situation in B.C. is probably indicative of that in the rest of Canada.

MacKenzie et al (1975) compiled data on fish kills in British Columbia salmon waters from the period 1960 to 1973. Of 62 kills reported, 17 were caused by pesticides. Of these 17 cases, which constituted an estimated 6 - 7% of all fish kills in B.C., four apparently were associated with use of PCP and TTCP for treatment of wood and poles (Table A4-4).

In the review paper on molluscicides, Strufe (1965) summarized Enigk and Dübwel (1960a) on the effect of NaPCP on producers, as follows:

> "Field trials revealed that contact of water milfoils (Myriopbyllum) and reeds (Pbragnites) with NaPCP chiefly results in heavy damage to the foliage but such injury is only of a temporary nature. After a relatively short time numerous young shoots are formed, and the plants recover within six to eight weeks. Similar results were obtained by Halawani et al. (1951). In field trials carried out in Egypt, these authors noted that aquatic plants of different species were not harmed by brief application of the NaPCP. Hunter et al. (1952) found the toxicity of NaPCP to aquatic plants was greater on dicotyledons than on monocotyledons".

Strufe (1965) further reported that Enigk and Dübwel (1960)

> "noted that pastures and ditches treated with NaPCP for controlling Galba truncatula became re-populated with Protozoa and algae within a few weeks of the application. Local conditions permitting (inactivation of the chemical by ultraviolet light and adsorption by mud), areas treated at 10 ppm were re-colonized by Protozoa and algae within 16 days."

4.2.1.2 Secondary effects. Chlorinated phenolic compounds, even when present in minute amounts can cause taste and odor problems in water (Burttschell et al, 1959) and impairment of flavor in fish and other organisms present in these waters (Fetterolf, 1964; Chatterjee, 1974). In discussing the chlorination of phenols, the reaction products, and the presence of tainting materials, Burttschell et al (1959) noted that during the chlorination of phenols the reaction "proceeds by progressive substitution of available ortho and para positions . . . . Several taste-producing substances and other relatively nontaste-producing ones comprise the mixture of chlorination products." Figure A4-1 (from Burttschel et al, 1959) illustrates the course of chlorination of a 20 ppm phenol solution reacted with 40 ppm chlorine, at pH 8, with the parenthesized figures referring to the approximate amounts present after 18 h. Burttschel et al (1959) suggested that "Earlier efforts had failed to establish the identity of the products from the molar ratio of chlorine to phenol because of the number of concurrent and consecutive reactions which take place including extremely complex kinetic relationships". In their summary, Burttschell et al (1959) state that the presence of chlorophenolics can be detected by taste when the reaction systems have resulted in production of 2,6- and 2,4-DCP and 2-CP in amounts in excess of their combined threshold value.

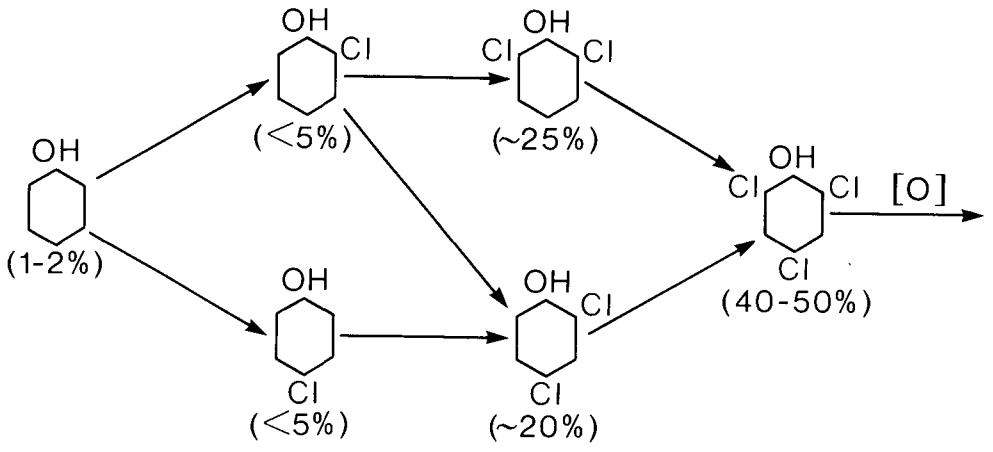


FIGURE A4-1 COURSE OF CHLORINATION OF PHENOL
(Burttschel et al, 1959)

Following the occurrence of off-flavor in eels, trout, and herring taken in Roskilde Fjord, Denmark, which received effluent from a pesticide factory, Boetius (1954) determined in laboratory experiments the concentration (vol./vol) of o-CP required to impart off-flavor to eels in brackish water and oysters in 30% salt water, both at 16°C. A concentration of 0.1 ppb o-CP tainted flesh of eels after 10 days exposure and oysters after 4 days exposure.

Fetterolf (1964) investigated taste and odor problems in fish from Michigan waters. In a review of the literature, he noted that Winston (1959) had reported off-flavor in fish exposed to minute concentrations of chemicals including three chlorophenols. The concentrations responsible for noticeable taste were 0.005 mg/L of 2,4-DCP, 0.015 mg/L of o-chlorophenol, and 0.05 mg/L of p-chlorophenol. Fetterolf (1964) listed four methods by which fish living in freshwater may take up a taint causing material:

> "1) Across the gill membrane into the blood; 2) Across the gut membranes into the blood; 3) Absorption through skin; and 4) Adsorption to the mucosa."

TABLE A4-4 FISH KILLS ATTRIBUTED TO CHLOROPHENOLS, BRITISH COLUMBIA (1963 - 1973) (Adapted from Mackenzie et al, 1975).

Location	Month-Year	Species	Total number killed	Agent	Circumstances	Comments
Sooke Basin	5-63	ocean perch	1000	PCP suspected	treatment of lumber	
Little Campbell River (Surrey) (Tributary)	8-72	trout, salmon, stickle-back	1000	PCP	spraying of hydro poles	
Victoria Harbour	12-72	anchovy, herring	10 tons	PCP suspected	pole and sawmill wood treating plant	kills have occurred several times
Mamquam Channel	10-73	adult and juvenile coho salmon,	500	PCP and TTCP	lumber treatment tank overflowed	court conviction
		shiners	500			

Of these, he considered the gill as probably the major organ for uptake. He also associated off-flavor fish with phenols in effluent discharged from a petroleum refinery and a bleached kraft pulp and paper mill.

Shumway and Palensky (1973) bioassayed 22 organic compounds, including 9 CPs, for impairment of flavor in rainbow trout, largemouth bass, and bluegills. The Estimated Threshold Concentration (ETC) (i.e. the highest estimated concentration of a material that will not impair the flavor of the flesh of exposed fish) ranged from 0.4 ppb of 2,4-DCP to 84 ppb of 2,3-DCP as observed for bass and trout respectively (Table A4-5). The data were acquired in 48 h exposure tests except for the 2,4-DCP trout test where the exposure was for 96 h. In their investigation the off-flavor of trout exposed to 2,4-DCP reached maximum in less than 33.5 h, but they also noted that the corollary was true, in that following exposure to 2,4-DCP for 24 h, trout lost the acquired off-flavor in 33.5 h in fresh water. They observed that the rate of acquisition of the off-flavor was apparently faster than the rate of loss, and that the degree of off-flavor was directly related to the concentration of the compound to which the fish was exposed.

TABLE A4-5 THE ESTIMATED THRESHOLD CONCENTRATION (ETC) FOR SEVERAL CHLOROPHENOLS CAUSING IMPAIRED FLAVOR IN FISH; THE HIGHEST CONCENTRATION (HC) NOT IMPAIRING FLAVOR, AND LC_{50} DATA. (Excerpted from Shumway and Palensky, 1973)

Compound	Fish	ETC (ppb)	HC (ppb)	LC_{50} (ppm)
phenol	trout	--	5,600	
m-chlorophenol (3-CP)	trout	25	1,000	10
m-chlorophenol (3-CP)	bluegill	--	1,000	
o-chlorophenol (2-CP)	trout	60	100	
p-chlorophenol (4-CP)	trout	45	21	
2,3-DCP	trout	84	32	10
2,4-DCP	trout	1	0.01	
2,4-DCP	bass	0.4	0.1	
2,4-DCP	bluegill	14	10	
2,5-DCP	trout	23	10	
2,6-DCP	trout	35	10	
2,4,5-TCP	trout	--	320	1
2,4,6-TCP	trout	52	10	

Chatterjee (1974) reported on the tainting of fish in the Upper Great Lakes, and stated that the source of phenolic compounds associated with tainting of fish originated in effluent from plants producing kraft and groundwood pulps and discharging into water leading to Thunder Bay and Nipigon Bay of Lake Superior, and the North Channel of Lake Huron, but these phenolic compounds were not further identified. Chatterjee (1974) also pointed out that tainting compounds present in the commercial fisheries waters and acquired by fish can result in rejection of tainted fish at the market place and also a possible reduction of fish populations. It was postulated that the latter was a result of an avoidance reaction by fish from their usual spawning beds due to the presence of a chemical barrier. A short review on the "avoidance" reaction of fish is contained in the NRCC publication by Marier (1973).

4.2.2 Effect on Systems. Little information has been available on overall impact of chlorophenols on marine and estuarine communities. At a symposium on PCP in 1977, Tagatz et al (1978) reported on the effect of PCP on development of estuarine macrobenthic communities. Macrofauna in aquaria were exposed for 13 weeks to three levels of PCP plus a control with no PCP. Each treatment was replicated 10 times. The authors stated:

> "The averages and ranges of PCP concentrations measured in water were 1.8 µg/L (0.5-4.1), 15.8 µg/L (8.9-22.0), and 161 µg/L (135-230). No PCP was detected in water samples from the control aquaria. The sediment samples from control aquaria and aquaria exposed to 1.8 µg PCP/L did not contain detectable amounts of PCP. A range of 3 to 6 µg PCP/kg was found in sediment from aquaria exposed to 15.8 µg PCP/L; 41 to 71 µg PCP/kg was found in sediment from aquaria exposed to 161 µg PCP/L. Limits of detection were 0.2 µg PCP/L in seawater and 2.5 µg PCP/kg (dry weight) in sediment. Recoveries averaged greater than 80% for water and sediment samples.
>
> "At the end of the experiment, macrofauna established in control and experimental aquaria was examined. Mollusks, arthropods and annelids were numerically dominant among the macrofauna. Although exposure to 1.8 µg PCP/L had no effect, the higher concentrations of PCP caused marked reduction in the numbers of individuals and species. Mollusks were the most sensitive taxonomic group to PCP. These results and our previous studies on the effects of a nine-week exposure to PCP on the establishment of macrobenthic communities indicate that discharge of PCP into natural waters could

alter the normal colonization by benthic animals and could impact various ecological relationships among localized populations."

Cantelma and Rao (1978) utilized the same test equipment and procedures as Tagatz et al (1978) to determine the effect of PCP on the establishment of meiobenthic communities. ("Meiobenthic community" covers the range of invertebrate organisms in marine sediments. Similarily, "microbenthic community" refers to the range of invertebrate organisms in freshwater sediments.) In two separate tests meiofauna had been exposed to PCP average concentrations of 7, 76, and 622 µg/L for 9 weeks and PCP average concentrations of 1.8, 15.8, and 161 µg/L for 12 weeks. PCP concentrations of 1.8, 7, and 15.8 µg/L did not effect the biomass and density of nematodes, the dominant group of all meiofauna encountered. At the intermediate concentration of 76µg PCP/L there was a statistically significant increase ($p < 0.01$) in nematode biomass and densities, while the higher concentrations of PCP caused a decrease. Although species diversity indices of control aquaria did not differ significantly from those of PCP-exposed aquaria, nematodes classed as epistrate feeders were most abundant in the control aquaria and in aquaria with PCP concentrations of 1.8, 7, 15.8, and 76µg/L. Deposit feeders were relatively abundant among the nematodes in aquaria exposed to 161 and 622µg PCP/L. Cantelma and Rao (1978) suggested the nematode population shifts resulted from variations in macrobenthic fauna and food (algae) supply caused by the biocidal effects of PCP and also due to the toxic effects of PCP on meiofauna.

The ecological effect of 2,3,7,8-TCDD, a dioxin contaminant found in both TCP and the phenoxy herbicide, 2,4,5-T, has been reported, but only as a by-product of a primary investigation involving herbicides. In 1962, at Elgin Air Force Base, Florida, a program was initiated to design, develop, and test aerial dissemination systems for herbicides. Over a period of 8 years, 1962 - 1970, an area of approximately 2.6 km^2, used as the herbicide application site, was treated with massive doses of 2,4-D; 2,4,5-T; picloram, and cacodylic acid. One 92 acre test plot within the experimental area received 39,547 kg of 2,4,5-T from 1962 through 1964. The 2,4,5-T was known to have had high levels of TCDD. Soil samples taken from this area in 1974 had 10 to 710 ppt of TCDD which was stratified within the top 6" of soil. The quantitative changes during the ecological succession that occurred in the vegetation and insect communities of this area have been noted by Young (1974), and Young et al (1975). No evidence was presented to show the influence, if any, of TCDD on the plant community. Although vegetation coverage of the test area had significantly increased during the study period, and

significant increases had taken place in the number of arthopod specimens and varieties, there was little overall change in calculated community diversity (Young et al, 1975). Species diversity studies were conducted in 1969, 1970, 1973, and 1974 in two aquatic test ecosystems within the test area. Although TCDD was not water soluble it had been carried into the aquatic environment on eroded soil. TCDD levels of 10 to 35 ppt were found on silt where eroded soil had entered the water. There was no significant change in composition of ichthyofauna during the study period between streams in the treatment and control areas. Following sampling of both invertebrates and vertebrates from the streams, low levels of TCDD (12 ppt) had been found in only two species of fish, sailfin shiner (Hotropis hypselopterus), and mosquitofish (Gambusia affinis).

4.2.3 Water Quality Criteria.

International Joint Commission (IJC) - Great Lakes Water Quality Board

The IJC Great Lakes Water Quality Board (1974) recommended in their report that levels of phenolic compounds should not exceed 0.001 mg/L (1 µg/L) in raw public water supplies to protect against taste and odour in domestic water. The definition used by the IJC for phenolic compounds is as follows:

> "Phenolic compounds are defined (Standard Methods, 1971) as hydroxy derivatives of benzene and its condensed nuclei." (Reference: Standard methods (1971) American Public Health Association, American Water Works Association, and Water Pollution Control Federation (1971), Standard methods for the examination of water and waste water, 13th ed (American Public Health Association, Washington, D.C.), 874 p.)

They noted that the Canadian Water Quality Standards established a limit of 2 µg/L of phenolic compounds in the raw water supply unless reduced to this limit by applied treatment. They also remarked that "Water quality criteria for toxic effects of phenol and phenolic compounds to aquatic life reveal that requirements for protection of water supplies are considerably more stringent."

Cote (1976) sums up the situation as follows:

> "It would appear that the particular causative agents of tainting and their mode of action have not been resolved. One fact, however, has received general agreement: minute concentrations (ppb range) of organic chemicals create taste and odor problems in fish flesh."

United States

Prior to publication of the final Water Quality Criteria the United States Environmental Protection Agency (E.P.A.) had published, and requested public comment on, the Water Quality Criteria for chlorophenols which are part of the 65 pollutants listed as toxic under Sect. 307 (a) of the U.S. Clean Water Act of 1977 (U.S.E.P.A. 1978b, 1979a, 1979b).

The Dow Chemical Company had petitioned the U.S.E.P.A., Aug. 11, 1978, requesting the removal of 2,4-DCP, 2,4,5-TCP, and PCP from the toxic pollutant list on the grounds that the chemicals failed to meet the E.P.A. criteria for listing chemicals as toxic pollutants on the bases of persistance, degradability, and toxicity. E.P.A. made available for public comment Dow's submission in a Federal Register notice (FR 44(217):64555-9) dated November 7, 1979. E.P.A. also summarized in the notice the information factors to be addressed by a petitioner to have a chemical deleted from the toxic pollutant list.

Using the guidelines as set out in the Federal Register (U.S.E.P.A. 1978b) for deriving Water Quality Criteria for the protection of aquatic life, the E.P.A. calculated the criteria for 2-CP, 4-CP, 2,4-DCP, and 2,4,6-TCP for freshwater aquatic life (Table A4-6). Criteria for the remaining CPs could not be derived using the guidelines since there were insufficient data, as was the case for all CPs for saltwater aquatic life. The two derived values used for the criteria include a) the final chronic value for tainting of fish flesh, and b) the final acute value. For 2,4,6-TCP, for example, the two criteria values are a) 52 µg of 2,4,6-TCP/L as a 24-h average, and b) the maximum concentration should not exceed 150 µg 2,4,6-TCP/L at any time. The equivalent 24-h average and ceiling figures in µg/L for 2-CP, 4-CP, and 2,4-DCP are 60 and 180, 45 and 180, and 0.4 and 110, respectively (Table A4-6).

In the Federal Register notice of March 15, 1979 (U.S.E.P.A. 1979a) which dealt with suggested water quality criteria for 2-CP, 2,4-DCP, and PCP, there was also a section devoted to 2,3,7,8-TCDD. Although there were no data presented from which water quality criteria for either freshwater or saltwater aquatic life could be developed, the following statement was made as part of the criteria summary:

> "Basis for the Criteria: Freshwater Aquatic Life. Although few data are available for this compound, sufficient information exists to indicate a potential high environmental hazard. A variety of aquatic organisms biocon-

TABLE A4-6 UNITED STATES WATER QUALITY CRITERIA - SUMMARY OF AVAILABLE DATA FOR SPECIFIC MONO-, DI-, TRI-, TETRA-, AND PENTACHLOROPHENOLS

Data Categories[7]	Aquatic env.[2]	Chlorophenols (Conc. µg/L)[1,4,5] 2-CP	4-CP	2,4-DCP	2,4,5-TCP	2,4,6-TCP	2,3,4,6-TTCP	2,3,5,6-TTCP	PCP
Final fish acute value	F	1 800	540	770	63	150	20	24	25
	S		790		250			280	25
Final invertebrate acute value	F	180	180	110	110	240	12	23	14
	S		510		66			380	8.5
Final acute value	F	180	180	110	63	150	12	23	14
	S		510		66			280	8.5
Final fish chronic value	F	290	NA[3]	NA	NA	NA	NA	NA	NA
	S		NA		NA			NA	9.6
Final plant value	F	500 000	4 800	50 000	1 200	5 900	600	2 700	7.5
	S		3 300		890			440	290
Final chronic value	F				1 200		600	2 700	7.5
	S		3 300		890			440	9.6
Final chronic value for tainting of fish flesh	F	60	45	0.4		52			
0.44 x final acute value[6]	F	79	79	48	28	66	5.3	10	6.2
	S		220		29			120	3.7

Notes:
(1) All concentrations in µg/L and rounded to two significant figures.
(2) Aquatic environments: F = freshwater, S = saltwater
(3) NA = Data not available (blank also indicates that information from which data could be derived was not available)
(4) Data for 2-CP; 2,4-DCP; and PCP from Fed. Reg. 44(52):15926-15981. March 15, 1979.
(5) Data for 4-CP; 2,4,5-TCP; 2,4,6-TCP; 2,3,4,6-TTCP; 2,3,5,6-TTCP from Fed. Reg. 44(144):43660-43665. July 25, 1979.
(6) "The figure of 0.44 is an "adjustment factor" applied to LC50 data to determine the concentration likely to be lethal to be between 0 percent and 10 percent. It is based on 219 acute toxicity tests which showed that the mean concentration lethal to 0-10 percent of the test population was 0.44 times the LC50." Fed. Reg. 43(97):21508.
(7) No data was available for either freshwater or saltwater a) final invertebrate chronic value, or b) residue limited toxicant concentration.

centrated 2,3,7,8-tetrachlorodibenzo-p-dioxin to 20,000 times or more. When coho salmon were exposed for 96 hours and placed in uncontaminated water for observation over a 60-day period, there was 12 percent mortality of those fish exposed to 0.000056 µg/L." (U.S.E.P.A. 1979a).

APPENDIX 5

APPENDIX 5

5 MODE OF ACTION AND METABOLISM OF CHLOROPHENOLS, CHLORODIBENZO-p-DIOXINS, AND CHLORODIBENZOFURANS

The literature citations in this section on mode of action and metabolism of the CPs and their impurities reflect the lack of knowledge. The information presented in this chapter is primarily an update based on the papers published since 1975, but also includes some brief background statements from the earlier review literature.

De Bruin (1976) had the following general statement in his introductory remarks to a discussion on metabolic fate of xenobiotic compounds (i.e. foreign compounds):

> "One of the most valuable contributions that biochemistry has made, to the improvement of understanding of the modes of action of foreign compounds, consists of the elucidation of their metabolism in living organisms. Metabolism, in broad terms, covers the total physical and chemical fate of a compound in animals."

In remarks on metabolic stability of xenobiotic substances de Bruin (1976) commented:

> "Animal resistance to metabolic attack is favoured by the two distinctive properties of xenobiotic substances -- namely high polarity and high volatility. Such compounds are especially liable to be excreted rapidly, in unchanged form, by either renal or pulmonary mechanisms.
>
> "Prominent among highly polar substances, which are metabolically stable, are acidic compounds with low pK_a values . . . .
>
> "A typical example of an acidic phenolic compound which shows a relative lack of biotransformation is pentachlorophenol (pK = 5.3)".

5.1 Mode of Action

5.1.1 Chlorophenols

Various researchers have investigated the effect of PCP on enzyme systems. Desaiah (1978) examined the effect of PCP on the ATPase system in the rat hepatic, brain, and kidney fractions using in-vitro techniques. This recent study corroborated earlier reports by Weinbach (1956), Weinbach (1957), Farquharson et al (1958), and

Weinbach and Garbus (1965), that PCP may be acting as an uncoupler of oxidative phosphorylation at low concentrations and inhibiting the same at high concentrations. He also indicated that the Na^+ -K^+ATPase (cation transporting enzyme) may be the locus of action of PCP.

Van Overbeek (1964) explained the significance of "uncoupling agents":

> "Inside the mitochondria, adenosine triphosphate (ATP) is generated. This ubiquitous high energy phosphate is needed for all biological activities that require energy. The mitochondria generate ATP by sending a stream of electrons from stored food such as sugars to oxygen of the air. This is respiration. This flow of electrons is coupled, geared as it were, to the generation of ATP. In this manner the stored energy from foods such as sugars is converted to the readily usable energy of ATP. The chemicals known as uncoupling agents unmesh the gears of this mechanism. Respiration then becomes free-wheeling, but ATP generation stops."

Fukami (1976) briefly defined oxidative phosphorylation as follows:

> "With the exception of bacteria and photosynthetic organisms, the energy required to maintain living organisms is supplied from oxidative phosphorylation of mitochondria. Oxidative phosphorylation is a coupled reaction consisting of two complex enzyme systems, the respiratory chain (electron transport system) and the energy transfer system (phosphorylation), which utilizes redox energy liberated from the respiratory chain for the synthesis of ATP".

Miller et al (1977) have postulated that part of the mammalian toxicity of halogenated antibacterials, which include the CPs, is due to their perturbation of mammalian membranes.

Saarikoski and Kaila (1977) investigated the basic modes of action of PCP and 2,3,6-TCP by observing the effect of these non-specific pesticides on the spontaneous impulse activity of the abdominal tonic motor system in the crayfish, Astacus fluviatilis L. The researchers proposed two mechanisms: first, that the CPs induced depolarization of some excitable cell membranes belonging to the tonic motor system; and second, that there may be a direct effect to the chemical aspect of synaptic transmissions.

Arrhenius et al (1977), following an investigation of subcellular distribution of PCP, observed that:

"Pentachlorophenol (PCP) is a potent uncoupler of mitochondrial phosphorylation in vitro and also interferes with microsomal detoxication functions in vitro. This favours flavin mediated oxygenation compared with flavin cytochrome P-450 dependent reactions. Gas chromatographic analysis of subcellular fractions, obtained by zonal centrifugation showed markedly lower PCP concentration in mitochondria and a high accumulation in microsomes compared with cytosol. This increases the likelihood that PCP in vivo causes a malfunction in microsomal detoxication."

5.1.2 Chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans

Buu-Hoï et al (1972a) noted that although 2,3,7,8-TCDD was known as a potent toxic and teratogenic compound, and that its mode of action was unknown, they were able to demonstrate in male and female Wistar rats that the 2,3,7,8-TCDD produced deep perturbations in several enzymatic systems and that the liver was one of the main targets. The histopathologic evidence for this was presented by Buu-Hoï et al (1972b) and they also identified the thymus, heart and lungs as additional organs showing pathological deterioration.

Vos (1978) concluded from an extensive, in-depth review of the literature on the effects and mode of action of 2,3,7,8-TCDD that:

"The mechanism of the toxic action of TCDD is still unknown, as is the pathogenesis of most lesions. Research efforts on TCDD should concentrate in this area. In particular, the mechanism of the development of chloracne should be investigated: vitamin A deficiency or disturbances in lipid metabolism may play a role in the etiology of this lesion. Similarly, the mode of action of TCDD induced thymic atrophy should be further pursued. Finally, it would be of interest to investigate whether thrombocytopenia, hermorrhages and thrombosis are the result of endotoxin shock."

Kitchin and Woods (1979) examined the effects of 2,3,7,8-TCDD on hepatic microsomal mixed function oxidase (MFO) enzyme systems in female rats. In their discussion of the study they noted that:

"The results of the present study indicate that TCDD, a hepatotoxic, environmentally persistent chlorinated dioxin, selectively alters cytochrome P-448-associated toxification and detoxification reactions in mammalian

liver, and, moreover, is capable of inducing this response at remarkably low concentrations in the cell."

Kitchin and Woods (1979) further observed that:

"The extreme potency of TCDD has been demonstrated by the estimate that only 65 molecules of TCDD per hepatocyte are required to elicit a measurable increase in benzo(α)pyrene hydroxylase. Because of this exceptional potency, it is unlikely TCDD acts via a nonspecific effect on microsomal membranes."

Kitchin and Woods (1979) found in their study that the ED_{50} of orally administered 2,3,7,8-TCDD with respect to induction of benzo(α)pyrene hydroxylase in female rats was 0.63 μg/kg in comparison to an ED_{50} of 0.27 μg/kg for intraperitoneally administered 2,3,7,8-TCDD in male rats, as reported in the literature. They also found that the lowest effective dose level of 0.002 μg/kg (2 ng/kg) was 1/100 of the previously published lowest dose effect of 2,3,7,8-TCDD in mammals. They further stated that the dose-response relationship indicated there was a 300-fold difference between the lowest response dose and the ED_{50}.

Kitchin and Woods (1979) stated that:

"The minimal responsive dose is lower than the concentration of TCDD that has been determined in some environmental soil (Kimbrough et al., 1977) and fish (Baughman and Meselson, 1973b) samples obtained from areas highly contaminated with TCDD".

Neal et al (1979) stated that there is insufficient information to postulate a mechanism for the acute lethal effects of TCDD. Neal et al (1979) using currently available information suggested that:

"It appears at this time the most reasonable hypothesis concerning the biochemical mechanism of the lethality of TCDD is that it binds to a receptor in the cytosol fraction of mammalian cells, is transferred into the nucleus and increases or decreases the synthesis of a critical protein(s) or enzyme(s). This increase or decrease in critical protein or enzyme levels leads eventually to a generalized dysfunction of a number of different cell types leading eventually to death. What biochemical function or functions are affected by this increase or decrease in protein or enzyme synthesis is unknown at this time."

No information was obtained on the mode of action of chlorodibenzofurans.

5.2 Metabolism

Matthews and Kato (1979) classified halogenated aromatic compounds into three broad classes, Type I, II, and III, according to their decreasing polarity. They noted that Type I compounds, the most polar of the halogenated aromatics, include the CPs. In their generalized summary statement, on the metabolism and disposition of the CPs in mammalian species, Matthews and Kato (1979) noted that almost all halogenated aromatics are sufficiently lipophilic to be readily absorbed from the gastrointestinal tract, and the determinants in the disposition of these compounds once they enter the body are their polarity and rate of metabolism. Matthews and Kato (1979) stated that the CPs, Type I compounds, are excreted primarily as the parent compounds or conjugates of the parent compounds, and that the biologic half-lives of CPs may vary with the degree of chlorination and species exposed but usually do not exceed 24 h. PCDDs and PCDFs are Type II halogenated aromatics since they have an intermediate polarity due to substitution(s) other than organic halogens. Regarding Type II compounds, Matthews and Kato (1979) stated that:

> "Type II compounds may be polar enough to be excreted in the bile prior to their metabolic alteration, but since they are readily absorbed from the intestine, most of the unaltered compound that is excreted in the bile will be reabsorbed from the intestine and returned to the liver. Therefore, the elimination of Type II compounds is largely dependent upon their rate of metabolism, and since the metabolism and the greatest tissue concentration of Type II compounds occurs in the liver, the decrease in the body burden of Type II compounds can usually be described by a single exponential component."

5.2.1 Chlorophenols

5.2.1.1 Aquatic

Published research on the metabolism of PCP has been reviewed by C.M. Menzie of the Fish and Wildlife Service, U.S. Dept. of the Interior, in three publications. The first was in "Metabolism of Pesticides" published in 1969, followed by two updates in 1974 and 1978 (Menzie, 1969, 1974, 1978). These reviews highlighted the main findings on metabolism of PCP documented in published research reports through 1975. Menzie

(1978) noted, however, that even during the period of preparation of his most recent update, considerable additional literature had been published.

The following information on metabolism of PCP was taken primarily from Menzie (1974) (1978) with additional appropriate comments extracted from the literature he cited, plus more recent references in the literature.

> "In shellfish (Tapes philippinarum) PCP was rapidly absorbed and distributed into various tissues; and then it was quickly eliminated. Most of the accumulated PCP in tissues was undecomposed and either free or in bound form. The bound form was identified as the sulfate ester of PCP (Kobayashi et al., 1969, 1970a, 1970b)." (Menzie, 1974).

Kobayashi et al (1969) authored the first work on metabolism of PCP in aquatic organisms. Their initial work indicated that PCP was adsorbed particularly into the Bojanus organ and liver, while in their 1970 report they concluded that PCP can be largely detoxicated in the shellfish by the conjugation with sulfate. (Kobayashi et al, 1970b).

> "The protoporphyrin enzyme peroxidase, detected in snails, catalyzed oxidation of PCP to 2,2',3,3',5,5',6,6',-octachlorobiphenylquinone. In vitro studies with horseradish peroxidase also produced this compound (Nabih and Metri, 1971)" (Menzie, 1974).

Nabih and Metri (1971), also noted that the compound 2,2', 3,3', 5,5', 6,6' - octachlorobiphenylquinone showed potent molluscicidal activity.

> "The amount of PCP accumulated by goldfish (Carassius auratus) increased with time. At 0.1 ppm, the concentration factor at 120 h was about 1000; at 0.2 ppm, about 580. Excretion was rapid with active elimination with half eliminated after 10 h in PCP-free water. Most of the PCP in the fish had not undergone decomposition. It appeared that most of PCP transferred to the hepatopancreas was detoxified by sulfate conjugation or by decomposition. Excretion of PCP was in the form of a conjugate identified as pentachlorophenylsulfate (Akitake and Kobayashi, 1975; Kobayashi and Akitake, 1975a and b)." (Menzie, 1978).

As an additional note, Kobayashi and Akitake (1975a) observed that when fish, previously exposed to ^{14}C-labelled PCP, had been transferred to water containing non-

radioactive PCP there was excretion into and uptake of PCP from the medium. The concentration in the fish increased by the amount expected from absorption minus that expected from excretion until it reached a concentration of about 100 µg-PCP/g - body weight, at which time mortality occurred. Most of the PCP found in the fish had not undergone decomposition.

A Chemical Abstracts synopsis of Tokunaga's (1967) paper on distribution of PCP in fish noted that:

> "Radiography and beta particle emission techniques were used to determine the distribution of pentachlorophenol among fish organs. The highest pentachlorophenol concentration was found in the gill, followed by hepatopancreas, heart, skin, digestive organs, kidney and muscle."

Kobayashi and Akitake (1975b) also determined that PCP absorbed by fish accumulated in various organs, especially the gall bladder. Concentration of PCP in the bile was 539 µg/g after 24 h exposure to 0.2 ppm of PCP. An increase in concentration of PCP in the bile continued after the fish were removed to clean water, with a decrease noted in other organs. PCP in the gall bladder ultimately reached a level of 1,077 µg/g, a concentration factor of 5400. The gall bladder contained 41% of the total PCP detected in the fish. They suggested that probably a large proportion of PCP and ^{14}C found in the gall bladder transferred from the hepatopancreas after detoxification by conjugation or decomposition. They concluded that characteristic accumulation of PCP in gall bladder indicated that fish can dispose of PCP by active elimination, such as conjugation and decomposition.

Kobayashi et al (1976, 1977) isolated and identified glucuronide and pentachlorophenyl -β- glucuronide from the bile of goldfish, Carassius auratus. Kobayashi et al (1975) found that sulfate conjugation is one of the most general detoxication mechanisms for some phenolic compounds in goldfish.

An investigation into the metabolism of PCP and pentachloroanisole (PCA) in rainbow trout, Salmo gairdneri, was reported by Glickman et al (1977) and again by Lech et al (1978). Glickman et al (1977) concluded that:

> "Thin layer chromatographic and GC-MS analyses of the tissues of the PCP exposed trout indicated that there was no methylation of PCP in any of the tissues studied. Bile from PCP-exposed trout contained high concentrations (250 µg/g) of PCP, mostly as the glucuronide conjugate, but no other

metabolites were detected. However, bile from PCA-exposed trout contained PCP glucuronide (10 μg/g) as well as PCA, indicating demethylation of this compound in vivo by rainbow trout. Inclusion of 1 mg/liter of piperonyl butoxide in the PCA exposure system decreased the formation of PCP from PCA."

Glickman et al (1977) noted that the half-lives for PCP residues in blood, liver, fat, and muscle of rainbow trout were measured in hours, whereas the half-lives for PCA residues in these same tissues were measured in days.

Statham et al (1976) in studies with rainbow trout, Salmo gairdneri, established that several xenobiotica, including PCP, could be conjugated with glucuronic acid and excreted into bile in high concentrations. In the case of PCP the bile-to-water radioactivity was 5,360:1 after 24 h exposure. They suggested that " . . . analysis of bile of wild or caged fish from a suspect site may be useful as a qualitative monitoring aid for certain types of xenobiotics in water."

5.2.1.2 Terrestrial

"In the urine of a rabbit orally administered PCP-Na, pentachlorophenyl β-glucuronide and chloranil were found. Chloranil was also observed in internal organs of mice two hours after intraperitoneal injection (Tashiro et al., 1970). "^{14}C-PCP was administered to mice by subcutaneous or intraperitoneal injection. Most of the activity (72-83%) was excreted in the urine in four days; about half, in 24 hours; and only a trace (0.05%), in expired air. High activity was observed in gall bladder and its contents, wall of stomach fundus, contents of G.I. tract, and liver. In the urine, in addition to unchanged PCP, about 8% of activity was in the form of a PCP conjugate, not further identified. Tetrachlorohydroquinone (TCH) was also detected (Jakobson and Yllner, 1971)." (Menzie, 1974).

Jakobson and Yllner (1971) concluded that their results showed there was both gastric and biliary secretion of PCP and/or its metabolites, and excretion in the feces.

The most recent update by Menzie (1978) on metabolism of PCP contained selected references to research published from 1973 to 1975, as follows:

"Sprague-Dawley rats and NMRI mice were administered PCP in olive oil or propylene glycol. Most of the PCP was excreted unchanged. One metabolite was identified as tetrachlorohydroquinone (TCH). Both PCP and TCH were present in small amounts as conjugates (Ahlborg et al., 1974)."

Ahlborg et al (1974) also noted that TCH had been found in the urine of workers occupationally exposed to PCP.

De Bruin (1976) summarized the metabolism of substituted phenols as follows:

> "Most compounds undergo the direct conjugative reactions typical for phenol. Conjugation is usually confined to a single OH group in the case of the polyhydric phenols. Aromatic hydroxylation constitutes a minor biological reaction to which phenols are subjected. The introduction of reactive substituents, such as -COOH, $-NO_2$ and $--NH_2$, enables the phenolic compound to follow additional metabolic routes, although even then conjugation remains the most favoured transformation. However, when the phenol is of pronounced acidic character (e.g. mono- and dihydroxy benzoic acids, pentachlorophenol) its conjugative ability is diminished and elimination in unaltered form correspondingly increased. This is illustrated by the chlorinated phenols. With increasing number of chlorine substituents the phenols become more acidic (decreasing pK values), and the extent of conjugation is reduced (Dodgson et al, 1950; Deichmann et al, 1943: Cserjesi, 1972). Pentachlorophenol (PCP), besides being eliminated in free state, forms pentachlorophenyl-β-glucuronide which is excreted as a minor metabolite. In addition, PCP gives rise to the production of tetrachlorohydroquinone; these two metabolites are the only metabolites of PCP detected so far (Jacobson and Yllner, 1971; Tashiro et al, 1970; Ahlborg et al, 1974)."

Ahlborg (1977) reviewed the metabolism of PCP and observed that Braun and Sauerhoff (1976) (Braun et al, 1977) had reported the presence of PCP-glucuronide in urine from exposed rats. Ahlborg (1977) went on to state that:

> "The level of conjugated pentachlorophenol they found (9.4%) is in the same range as found by us (9-16%). However, they only found unconjugated tetrachloro-p-hydroquinone which is in clear contrast to our findings where the greater part of tetrachloro-p-hydroquinone occurs as a conjugate."

Ahlborg (1977) further stated:

> "Studies of the metabolism of pentachlorophenol (Ahlborg et al, 1974; Ahlborg et al, 1977; and Ahlborg and Thunberg, 1977) thus show that rapid dechlorination occurs in rats. The dechlorination is mediated by liver microsomal

enzymes and their activity can be enhanced by pretreatment with inducing agents such as phenobarbital, 3-methylcholanthrene and TCDD. The dechlorination products formed are tetrachloro-p-hydroquinone and trichloro-p-hydroquinone. The dechlorination proceeds with an initial hydrolytic dechlorination to tetrachloro-p-hydroquinone followed by a reductive dechlorination to trichloro-p-hydroquinone (Figure A5-1).

Other reports have indicated the presence of dechlorination products such as tetrachloro- and trichlorophenols (Engst et al., 1976) and tetrachloro-p-benzoquinone (Tashiro et al., 1970). In our studies, these findings have not been verified."

Ahlborg (1977) discussed the metabolism of TTCPs as follows:

"Of the three isomers, only 2,3,5,6-tetrachlorophenol is metabolized to a significant degree (paper VI (i.e. Ahlborg and Larsson, 1977)), urinary excretion of tetrachloro-p-hydroquinone constituting about 35 per cent of given dose. Trichloro-p-hydroquinone was identified as a minor metabolite of both 2,3,4,5- and 2,3,4,6-tetrachlorophenol."

Braun et al (1978) summarized an evaluation of the pharmacokinetics and metabolic fate of PCP in rats and monkeys (Braun et al, 1977; Braun and Sauerhoff, 1976) which noted species differences, including the fact that rats metabolized PCP and monkeys apparently did not. When Braun et al (1978) compared a pharmacokinetic model for PCP in man with those constructed for PCP in monkey and rats, they stated that:

"in terms of the excretion patterns, neither the rat nor the monkey are exactly like man. However, it does appear that the pattern of excretion by humans is more similar to the rat than the monkey in that both the rat and man do conjugate PCP with glucuronic acid."

Edgerton et al (1979) found in a PCP feeding study with adult female Sherman rats that the major metabolite was tetrachlorohydroquinone, as previously reported by Ahlborg et al (1977), and minor metabolites, not previously reported in the literature, were 2,3,4,5-TTCP and tetrachloropyrocatechol. They confirmed by gc-ms the presence of these phenolic metabolites as their methyl ethers in urine from the general population and from an occupationally exposed worker.

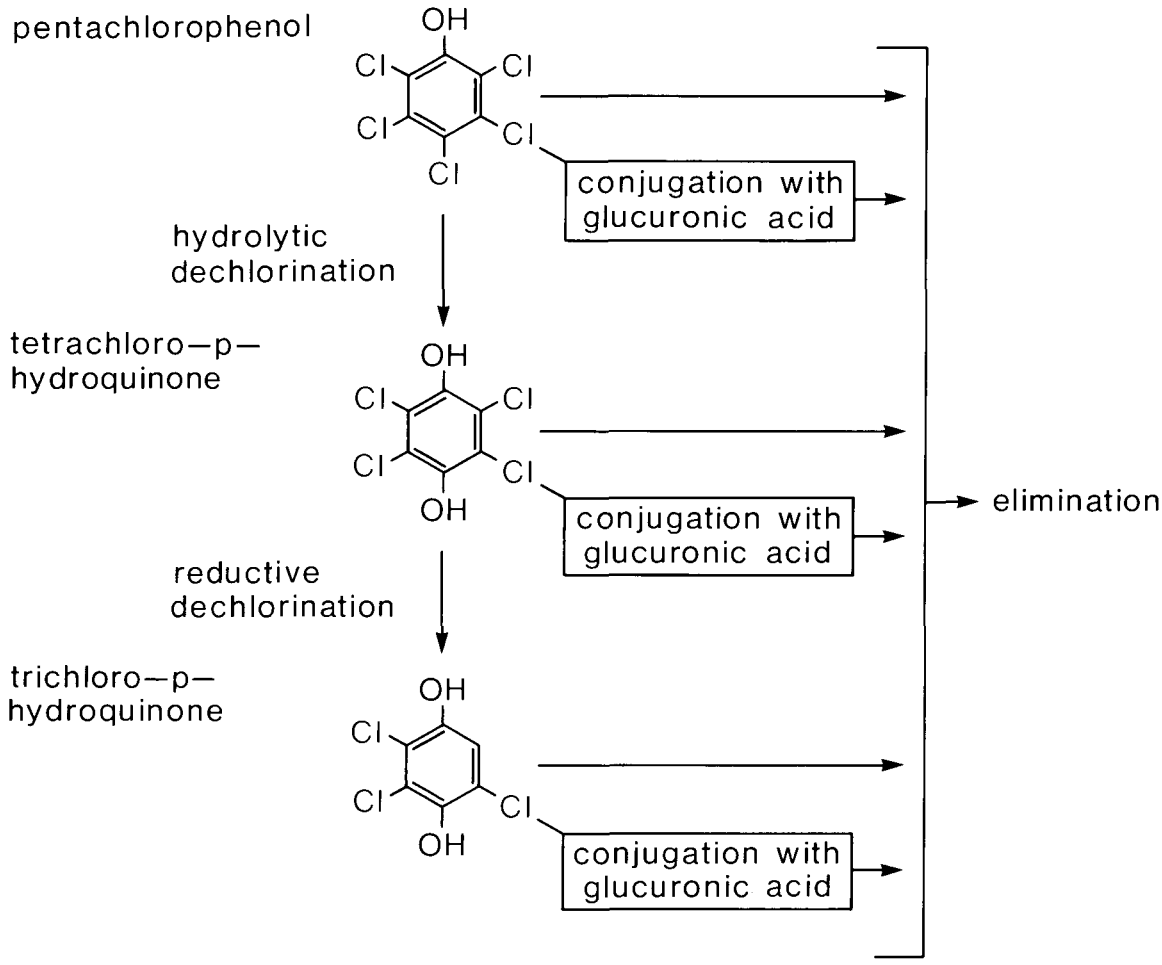


FIGURE A5-1 PROPOSED SCHEME FOR THE METABOLIC FATE OF PENTACHLOROPHENOL IN THE RAT. (Ahlborg, 1977).

5.2.2 Chlorodibenzo-p-dioxins

Rose et al (1976) reported on the fate of TCDD in rats given a single oral dose of 1 µg of (^{14}C)-2,3,7,8-TCDD/kg and repeated oral doses of 0.01, 0.1, or 1.0 µg of (^{14}C)-2,3,7,8-TCDD/kg/day, five days a wk for 7 wk. They found that following the single oral dose of 1.0 µg of (^{14}C) TCDD/kg, ^{14}C activity was detected only in feces and not in urine. The half-life of ^{14}C activity in the body was 31 $\pm$ 6 days. At 22 days after the single oral dose, concentrations of ^{14}C were located principally in the liver and fat. In those rats which received repeated oral doses of ^{14}C-TCDD, the half-life of ^{14}C activity in their bodies was 23.7 days. The major route of excretion was via the feces. Rose et al (1976) stated in their summary that:

> "The results of this study in rats indicate that TCDD approaches steady-state concentrations in the body within 13 weeks, and the rate constant defining the approach to steady-state concentrations is independent of the dosage of TCDD over the dose range of 0.01 - 1.0 µg of TCDD/kg/day."

Tulp and Hutzinger (1978) investigated the qualitative aspects of PCDD metabolism in rats with emphasis on structure of the metabolites formed. Because so little information exists in the literature on metabolism of PCDDs, the following information was extracted from their report:

> "We used mainly PCDD of low chlorine content since, by analogy with other chloro-aromatic compounds, larger amounts of metabolites could be expected than from higher chlorinated species. Also, to be able to administer relatively high doses, PCDD of known low toxicity were used.
>
> "In rats, dibenzo-p-dioxin, 1-chlorodibenzo-p-dioxin, 2-chlorodibenzo-p-dioxin, 2,3-dichlorodibenzo-p-dioxin, 2,7-dichlorodibenzo-p-dioxin, 1,2,4-trichlorodibenzo-p-dioxin and 1,2,3,4-tetrachlorodibenzo-p-dioxin are metabolized to mono- and dihydroxy derivates, whilst in case of dibenzo-p-dioxin and both the two monochloro isomers, also sulphur containing metabolites are excreted. Primary hydroxylation exclusively takes place at the 2-, 3-, 7- or 8-position in the molecule. In none of the experiments metabolites resulting from fission of the C-O bonds (ortho, ortho'- dihydroxychlorodiphenyl ethers, chlorocatechols) or hydroxylated derivatives thereof, were detected. No metabolites were found from octachlorodibenzo-p-dioxin.

"The metabolites from this study may help explain the fact that no metabolites of TCDD have been identified: PCDD metabolism occurs mainly (exclusively) via 2,3-epoxides, and since in TCDD these positions are blocked, the reaction is less likely to take place."

5.2.3 Chlorodibenzofurans

Zitko et al (1973) fed relatively high levels of 2,8-DCDF to immature brook trout (Salvelinus fontinalis). In the crude mixture of organic compounds in the water containing the excreta from the fish, they identified by ms the presence of a hydroxy-dichlorodibenzofuran.

Morita and Oishi (1977) noted the lack of research on metabolism, distribution, clearance, effect on reproduction, etc. of PCDFs in mammals. Morita and Oishi (1977) examined clearance and tissue distribution of PCDFs in mice following a single intraperitoneal dose of 0.50 mg of PCDF, which consisted of a mixture of tetra- to hexa-CDFs. Analysis of organs three days after treatment indicated that PCDFs were mainly located in liver, spleen, and fat tissues. Morita and Oishi (1977) noted that four weeks or eight weeks after single intraperitoneal administration of PCDFs, they had disappeared from most of the organs. The biological half life of PCDFs in mice was estimated to be two weeks.

APPENDIX 6

APPENDIX 6

6 DEGRADATION AND TRANSPORT OF CHLOROPHENOLS AND THEIR TRANSFORMATION PRODUCTS IN THE ENVIRONMENT

This appendix presents a brief summary of the pertinent literature on the degradation and transport of the CPs in various media, including water, soil and air. The contributions of chemical, photochemical, and microbiological processes to the degradation of the CPs are also discussed. Since the degradation processes occur over a period of time, the physical-chemical factors which influence environmental transport of the CPs are considered, under the headings of adsorption, diffusion, and volatilization. Additional information on movement of the CPs through one media to another are also discussed under the headings of exudation, leaching, surface movement and atmospheric movement.

6.1 Degradation

Freiter (1979) in a summary statement on stability of CPs in the environment listed three generalizations, which might not always agree with specific experimental data; they were 1) CPs are much more environmentally stable than the parent unsubstituted phenol, 2) as the number of Cl atoms increases, the rate of decomposition decreases, and 3) compounds containing a meta-Cl (e.g. 3-CP and 2,4,5-TCP) are more persistent than compounds lacking a Cl atom in a position meta to the hydroxyl group.

6.1.1 Chemical Degradation

6.1.1.1 In water. Aly and Faust (1964) investigated the fate of 2,4-DCP in lake water. In a buffered, biologically active system at pH 7.0, with a temperature of 25^{o}C, and with a concentration of 100, 500 and 1,000 µg/L, 50% of the phenol was decomposed in 6 days, with complete disappearance from the 100 µg/L system in 9 days. Under unbuffered, anaerobic conditions, and with high organic matter content, the 2,4-DCP persisted for over 43 days. Strufe (1968) reviewed the effect of electrolytic content of water and velocity of a stream on the activity of NaPCP. Although the solubility and activity of NaPCP are not largely influenced by salts dissolved in water of between 5 and 30^{o} of hardness, iron, lead, and copper salts may deactivate NaPCP by the formation of insoluble complex compounds. For example, in water with an iron content of 30 ppm the concentration of NaPCP diminished progressively from 10 ppm to 2 ppm in 120 days. In

regards to effect of stream velocity, a fast flowing stream maintained a given level of NaPCP activity further downstream from the point source than in a slow moving stream.

Pierce and Victor (1978) had the opportunity to determine the degradation products formed from PCP in a freshwater lake following the accidental release into the environment of a PCP-fuel oil waste from a wood-treatment plant. The major PCP degradation products observed in the contaminated lake water were pentachloroanisole ($PCP\text{-}OCH_3$); 2,3,5,6-TTCP, and 2,3,4,5-TTCP. Varying quantities of the methyl ether (anisole) of both TTCP isomers were also observed, but in low concentrations. Pierce and Victor (1978) stated that the 2,3,4,6-TTCP isomer was not observed but may have been present in small quantities. Also, it may not have been separated out in the gc system (Fox, 1978). Pierce and Victor (1978) observed that the concentration of 2,3,5,6-TTCP in the various lake water samples was low but it followed a pattern similar to PCP, indicating that 2,3,5,6-TTCP was present in the oil solution when it entered the lake. The concentration of 2,3,5,6-TTCP relative to PCP in the industrial waste-holding pond indicated that additional 2,3,5,6-TTCP may have been formed by photodegradation in the holding pond (Pierce and Victor, 1978). It was also noted that $PCP\text{-}OCH_3$ appeared to have been formed within the aquatic environment, probably in the sediment.

6.1.1.2 In soils. Although PCP has been classified as non-persistent in the environment (Arsenault, 1976), the categorization of chemicals, including CPs, as to their persistence in soil may be somewhat discretionary, as pointed out by Katan et al (1976) who investigated binding of (^{14}C) parathion in soil. They commented on their findings as follows:

> "Binding of parathion residues to soil results in a pronounced reduction of its insecticidal activity. This is a beneficial environmental consequence of the binding - at least for a short term. However, unless we know under which circumstances and in what forms the bound residues can be released or reactivated, or interact with other compounds in the environment, "loss" of toxicity should not be regarded as permanent.
>
> "Our results regarding the relatively rapid disappearance of the extractable parathion residues in soil confirm other reports. However, because of the concomitant formation of bound residues, as a consequence of its rapid degradation, the classification of this highly toxic pesticide as "non-persistent" is arbitrary until the fate of its bound residues is revealed. This may also be true for other "nonpersistent" pesticides."

In as much as PCP has been used as a herbicide in rice paddies in Japan for three decades, it was not unexpected that the majority of the research on degradation of PCP in soils, although somewhat limited, has been done in Japan. From a review of PCP degradation in soils by Kuwatsuka (1972), the following summary was abstracted. As a general rule, but with some exceptions, the degradation rate of PCP in soils was largely related to the organic matter content; the lower the soil organic matter content, the lower the rate of PCP degradation. Cation exchange capacity, and soil pH were less related to chemical degradation. Soil texture, clay content, degree of base saturation, and free iron oxides were not closely related to the rate of PCP degradation. Kuwatsuka (1972) stated that:

> "PCP degradation was assumed to proceed by both chemical and microbial means based on the results of experiments on soil sterilization, soil temperature, and the degradation products of PCP. Chemical degradation is, however, presumed to be caused and promoted by microbial action as no degradation occurred in Higashiyama soil containing almost no organic matter."

The products of PCP degradation detected by gc as listed by Kuwatsuka (1972) were as follows: 2,3,4,5-, 2,3,4,6-, and 2,3,5,6-TTCPs; and 2,3,6- 2,4,6-, 2,3,5-, and 2,3,4-; and/or 2,4,5-TCPs. The major products were 2,3,4,5-TTCP and 2,3,6-and 2,4,6-TCP. Similar finding were reported by Ide et al (1972).

From work in France, Casanova and Dubroca (1973) reported that preplant soil treatments with PCP (containing approx. 6.2% HCB impurity) in a greenhouse lettuce culture, resulted in residues of both PCP and HCB in the soil and lettuce. The Chemical Abstract summary did not report the time interval between treating and sampling.

Pierce and Victor (1978) who had determined the degradation products formed from PCP in lake water also verified that the same degradation products were present in sediment exposed to PCP, and included: 2,3,5,6- and 2,3,4,5-TTCP and the methyl ether of PCP and both TTCP isomers. It was suggested that the PCP-OCH_3 may have been produced in the sediment prior to its distribution in the water, although its solubility in water was low.

6.1.2 Photochemical Degradation. Hiatt et al (1960) investigated the action of sunlight on NaPCP since they recognized that photochemical degradation of NaPCP might have been a factor which reduced the efficacy of NaPCP as an agent for the control of snail vectors of schistosomiasis in certain streams in South Africa. The summary of their

findings stated that irradiation of dilute aqueous solutions (10 ppm) of NaPCP with light in the wavelength range of 290 to 330 nm caused a chemical alteration of NaPCP with a concomitant loss of moluscicidal activity. The degradation of NaPCP was measured by the colorimetric method. The reaction followed first order kinetics and the velocity of the reaction was directly proportional to light intensity. The velocity constant was 3.4×10^{-4}/s at a light intensity of approximately 0.04 watts/cm^2 between 290 and 330 nm.

In Japan, following the observation that NaPCP was readily decomposed by sunlight after its application in rice fields for the control of barnyard grass, Panicum crusgalli L., Kuwahara et al (1966a) noted that the photochemical reaction of NaPCP in an aqueous solution, when exposed to sunlight, led to various decomposition products (Fig. A6-1). The major products were chloranilic acid (I) and a yellow compound identified as $C_{12}HO_4Cl_7$, the structure of which was 3,4,5-trichloro-6-(2'-hydroxy-3',4',5',6'-tetrachlorophenoxy)-o-benzoquinone (II). In a subsequent paper, Kuwahara et al (1966b) identified three minor decomposition products as tetrachlororesorcinol (III), and two hydroxy-p-benzoquinones: 1) a red compound with the molecular formula, $C_{12}HO_4Cl_7$ and the structural formula 2,5-dichloro-3-hydroxy-6-pentachlorophenoxy-p-benzoquinone (IV), and 2) an orange-red compound, $C_{12}H_2O_5Cl_6$, or 2,6-dichloro-3-hydroxy-5-(2',4',5',6',-tetrachloro-3'-hydroxyphenoxy)-p-benzoquinone (V).

An additional yellow decomposition product was later identified by Kuwahara et al (1969) as $C_{18}H_2O_6Cl_{10}$ with a structure as follows: 3,5-dichloro-4-(2,3,5,6-tetrachloro-4-hydroxyphenoxy)-6-(2,3,4,5-tetrachloro-6-hydroxyphenoxy)-o-benzoquinone (VI). The reaction mechanisms involved in the formation of these products after the photochemical degradation of NaPCP were described and summarized by Munakata and Kuwahara (1969). In general, all the photodegradation products had stronger fungicidal activity but weaker phototoxicity and lower fish toxicity than NaPCP.

Ultraviolet (2537A) irradiation of PCP in organic solvents produced only a single major decomposition product, 2,3,5,6-TTCP plus a small amount of another phenolic substance assumed to be an isomeric TTCP (Crosby and Hamadmad, 1971). Crosby and Hamadmad (1971) concluded that although photodecomposition of PCP was observed, both in solution and in solid film, light was probably relatively unimportant in the loss of PCP from the environment. They noted that this was in contrast to the instability of NaPCP which had been observed by Kuwahara et al (1966a).

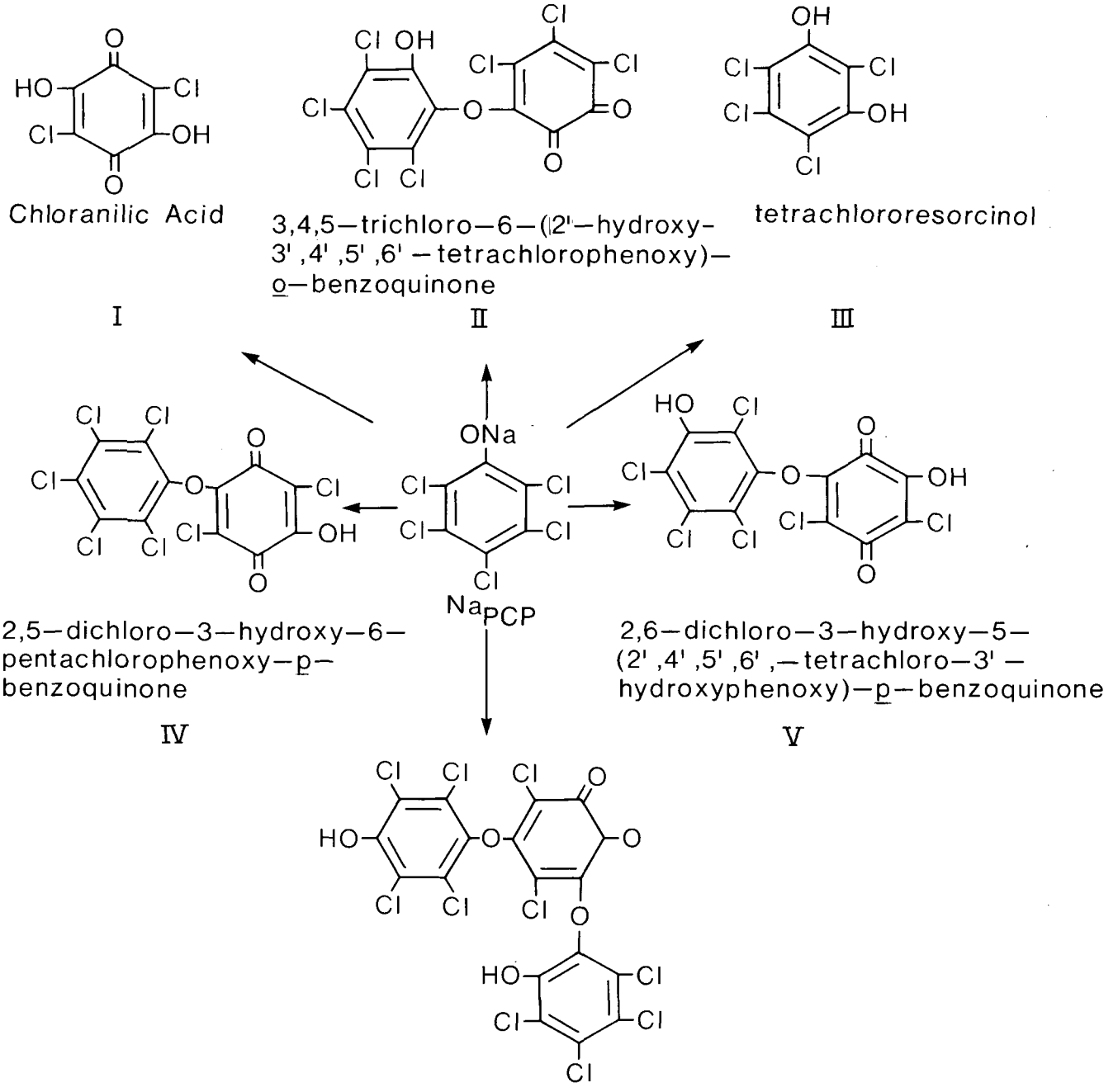


FIGURE A6-1 PHOTOCHEMICAL DEGRADATION PRODUCTS OF NaPCP IDENTIFIED BY KUWAHARA ET AL (1966a, 1966b, 1969). (Adapted from Menzies, 1974).

Recently, Wong and Crosby (1978) reported on the photodegradation products which evolved when dilute aqueous solutions (100 ppm) of PCP were irradiated with summer sunlight at Davis, Calif., or UV light in the wavelength region of 300 - 450 nm for 7, 20, and 30 days. After 7 days the photodegradation products included: CPs, tetrachlorodihydroxyl benzenes and non-aromatic fragments such as dichloromaleic acid. Subsequent irradiation, to 20 days, of the tetrachlorodiols resulted in the formations of hydroxylated trichlorobenzoquinones, trichlorodiols, dichloromaleic acid and non-aromatic fragments. The dichloromaleic acid, when irradiated, produced chloride ions and carbon dioxide. Wong and Crosby (1978) further stated that prolonged irradiation of PCP or its photodegradation products for 30 days yielded colorless solutions containing no ether-extractable volatile materials; and evaporation of the aqueous layer left no observable polymeric residue such as humic acids. Crosby and Wong (1976) also reported that OCDD was formed when a high concentration of NaPCP was irradiated (App. 7, Sect. 7.1.1). Wong and Crosby (1978) during their presentation at the 1977 PCP symposium had indicated that the velocity of the degradation reaction was directly related to the pH of the aqueous solution. At pH 7.3 there was almost no PCP remaining after 20 h exposure to simulated sunlight, while in sunlight total degradation of PCP was achieved within 5-7 days. At pH 3.3 the degradation reaction was slow with more than 50% of the PCP remaining after 48 h exposure.

Yasuhara et al (1977) investigated the possibility of using photodecomposition as a tool for deodorization of odorous CPs. They concluded that photolysis in the presence of hydrogen peroxide at a concentration of 1000 ppm was useful to decompose odorous 2-CP, which they stated had a threshold level of 2 ppb.

6.1.3 Microbiological Degradation. The microbiological breakdown of CPs was reviewed by Cserjesi (1972) and more specifically for PCP by Arsenault (1976) and Stranks (1976). In spite of the fact that CPs are for the most part used as anti-fungal agents or antimicrobials, these same CPs can be detoxified by microorganisms including fungi. Some microorganisms are implicated in the formation of chloroanisoles from NaPCP, as detailed in this section.

One of the factors first noted concerning degradation of CPs, was the importance of the ring position of the chlorines. Alexander and Aleem (1961) hypothesized that the aromatic nucleus of halogenated phenols remained intact for long periods in compounds containing the halogen in a position meta to the phenolic hydroxyl.

Stranks (1976) had noted the investigations of Lyr (1963) as follows:

> "Lyr (1963a) was one of the first to show that fungi, including some woodrot fungi, were capable of detoxifying PCP; phenol oxidases, i.e. tyrosinase and peroxidase, were the enzymes. The stability of halophenols was found to increase with increasing chlorination of the aromatic ring. Detoxification occurred through inactivation of the hydroxyl group. Ingols and Stevenson (1963) also observed increased resistance to biodegradation with increased chlorination of the phenol ring."

Various authors have discussed the effect on rate and extent of biodegradation of CPs by both "adapted" and "non-adapted" microorganisms. Ettinger and Ruchhoft (1950) observed that low concentrations (1 ppm) of o- and p-CPs when added to dilutions of domestic sewage, were not always removed in periods of 20 to 30 days at 20°C. Similar concentrations of these mono-CPs were removed at this temperature during similar periods of storage in polluted surface waters; therefore, they concluded that removal of the monochlorophenols required specialized microflora. Ettinger and Ruchhoft (1950) further suggested that bacterial flora capable of destroying these CPs would soon be established in a relatively pure stream if the wastes containing the CPs were discharged regularly, rather than intermittently.

Cserjesi (1967) reported that Trichoderma viride and T. virgatum were the only fungi he had tested (which included T. harzianum, Cephaloascus fragrans, Graphium sp., Penicillium sp, and Ceratocystis pilifera) which were capable of significant degradation of PCP present at concentrations of approximately 10 ppm in a malt extract solution. Unligil (1968) concluded that, although PCP was subject to fungal depletion, as observed in laboratory studies, direct application of these results to the field would not be possible because of the much higher concentration of PCP in treated wood in service.

In a study to determine the compatability of wood preservatives and the biological purification system for sewage, Pauli and Franke (1972) observed that both PCP and 2,4,5-TCP were not degraded in sewage even after 14 days exposure; in contrast to p-cresol and o-phenylphenol which degraded remarkably easily and after a very short adaptation period. On the other hand, an EPA project in 1971 ("Biological treatment of chlorophenolic wastes", Water Pollution Control Research Series 12130, EGK 06/71) which demonstrated the biodegradability of PCP in a sewage treatment plant was summarized by Arsenault (1976) as follows:

> "In a demonstration (under an EPA grant[116]) of the biological treatment of PCP waste in a sewage treatment plant, mixtures of PCP in aeration lagoon influent were aerated continuously and analyzed. In two separate experiments the PCP concentration fell from 39.5 ppm to 0.5 ppm in three days and from 81 ppm to 0.6 ppm in 30 hours. These experiments again prove that PCP can be degraded in a sewage treatment plant."

Kirsch and Etzel (1973), who had noted that information regarding biodecomposition of PCP was relatively scarce and quite vague, had investigated PCP biodegradation in heterogeneous cultures of microorganisms. Under specific idealized laboratory conditions, acclimated, proliferating, and nonproliferating mixed culture bacterial populations biodegraded NaPCP as measured by the substantial release of radioactive carbon during 24-h exposure period. The rate and extent of CO_2 liberation was highest in nonproliferating cultures where NaPCP was the sole carbon source. In addition, the rate of CO_2 liberation in a nonproliferating population was proportional to the biomass concentration at low cellular levels; but, at high cell concentration, rate limitation was observed. Kirsch and Etzel (1973) also stated

> "Finally, another factor that may influence the apparent rate of PCP oxidation is the condition of cells when the analysis is made. In the resting cell suspension exposed only to PCP, oxidation of PCP is more rapid than in cells supplied organic nutrient such as nutrient broth plus PCP. This suggests that PCP is probably not a primary substrate but serves rather as a secondary substrate that does not compete favorably with more easily degraded materials. In fact, it would not be at all surprising to find that PCP is co-metabolized by actively growing cells which use some other aromatic moiety as a primary energy source."

In the same context, degradation of a substance can be enhanced through the application of the principle of co-metabolism as defined by Stranks (1976):

> "In a study of the biodegradation of the herbicide 2,3,6-trichlorobenzoate he (i.e. Horvath, 1972) has shown that this substance could be more readily broken down by the flora of lake water if Na benzoate (a probable intermediate) were added to the system. Similarly the utilization of alkyl benzene sulfonate by a Pseudomonas species was enhanced by the addition of phenol (Horvath and Koft 1972), as was the oxidation of chlorophenols by Rhodotorula

glutinis (Walker 1973). Even glucose, a nonrelated analogue, can be used to enhance the degradation of chlorobenzoates (Horvath 1973). In these instances the principle of cometabolism is recognized as operative: the more easily metabolized cosubstrate supports growth of a greater population of active organisms equipped with an enzyme complement capable of degrading chlorinated aromatics. With this system, up to 100% degradation of aromatic material to CO_2 and water has been observed. Conceivably similar systems occur naturally in the global ecosystem. These would respond similary to the introduction of toxic chemicals such as PCP to the environment."

Watanabe (1973) observed that: "In the soil perfused with 40 ppm PCP solution, PCP was decomposed and five chlorine atoms of PCP were liberated as chloride ion after about three weeks". Following the initial period of PCP decomposition further addition of PCP accelerated the rate of PCP degradation and de-chlorination. The de-chlorination process corresponded approximately to PCP disappearance. Pseudomonas, or a closely related bacteria, were isolated from the soil.

Walker (1973) reviewed previous reports on phenol degradation by yeast species, including Rhodotorula glutinis and R. minuta. In the investigation, the study of co-metabolism was extended to the oxidation of halogenophenols by phenol-grown cells of a strain of R. glutinis isolated from Rothamstead soil. These cells oxidized 3- and 4-CPs to 4-chlorocatechol. Phenol-grown cells consumed 0_2 in the presence of 2-, 3-, or 4-CP and 2,4-DCP.

Chu (1972) isolated from a mixed microbial population a Gram-variable bacillus, designated as KC-3, which utilized PCP as a sole carbon source for growth with concurrent mineralization of the compound to carbon dioxide and chloride. Chu (1972) stated that: "When all 19 chlorophenols and unsubstituted phenol were tested as growth substrates, individually, only 2,3,4,6-TTCP and 2,4,6-TCP were able to support the growth of KC-3. Pentachlorophenol metabolism of culture KC-3 was shown to be inducible." Chu (1972) also stated that: "Under selected experimental conditions all multichlorinated phenols were found to be utilized to some extent by culture KC-3, while monochloro-phenols and the unsubstituted phenol were not catabolized." Several additional multi-halogenated phenols which were removed from the solution by respiring cells did not support growth (Chu and Kirsch, 1973).

Reiner et al (1978) in a review on microbial metabolism of PCP proposed a hypothetical pathway for the biodegradation of PCP by the bacterial culture, KC-3 (Fig. A6-2).

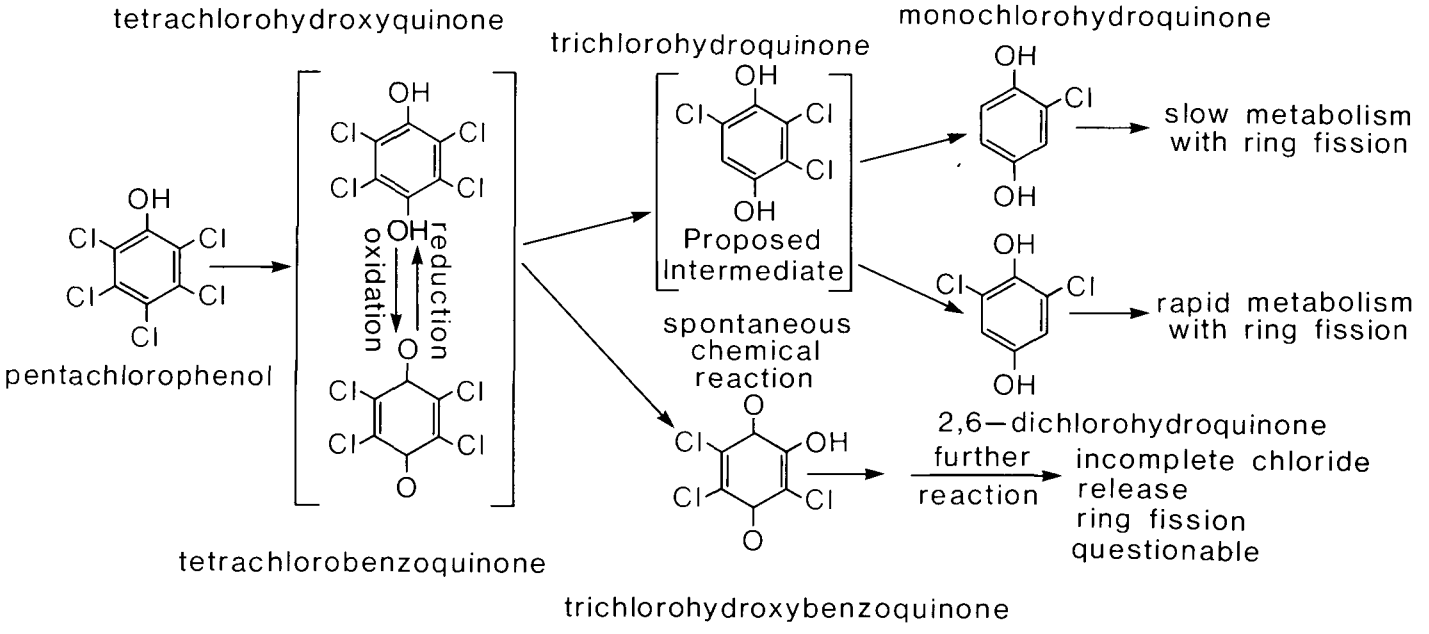


FIGURE A6-2 HYPOTHETICAL PATHWAY FOR THE BIODEGRADATION OF PENTACHLOROPHENOL BY THE BACTERIAL CULTURE, KC-3 (Reiner et al, 1978).

Following detection of 2,3,4,6-tetrachloroanisole and pentachloroanisole in chickens that had been exposed to CPs in broiler house litter (Curtis et al, 1972), it was firmly established that the 2,3,4,6-tetrachloroanisole had been formed by methylation of 2,3,4,6-TTCP by at least three of the fungi found in poultry litter, Scopulariopsis brevicaulis, Aspergillus sydowi, and Penicillium crustosum (Curtis et al, 1974). Chloroanisoles at very low levels impart a musty taint to chicken tissue and eggs and can be readily detected by odour (Table A6-1). The mechanism and routes of uptake of the chloroanisoles from the litter to the poultry have not been fully investigated; since chloroanisoles are significantly volatile, one suggested route was through inhalation. Another possibility was uptake through dermal contact resulting from chickens roosting in the litter (Curtis et al, 1974).

TABLE A6-1 ODOUR DETECTION THRESHOLD CONCENTRATIONS FOR CHLOROANISOLES IN AQUEOUS SOLUTIONS (Adapted from Curtis et al, 1972)

Compound	Threshold conc. (in µg/g solution[1])
Pentachloroanisole	4×10^{-3}
2,3,4,6-Tetrachloroanisole	4×10^{-6}
2,4,6-Trichloroanisole	3×10^{-8}
2,3,6-Trichloroanisole	3×10^{-10}

[1]Tests were made with 23 subjects in triplicate and were significant at the 1% level.

Land et al (1975) had identified 2,4,6-TCP in several samples of wood shavings at levels around 50 ppm. They also stated that taint can be caused by the corresponding anisole, 2,4,6-TCA, at levels below 10 ppb in the chicken.

Engel et al (1966) had reported that the "musty" taint in chicken eggs had been attributed to the presence of 2,3,4,6-tetrachloroanisole in litter shavings. Engel et al (1966) further reported that: "Related chlorinated anisols and phenols were tested for activity: 2,4,6-trichloroanisol was more active than 2,3,4,6-tetrachloroanisol; but 2,4,5-trichloroanisol, pentachloroanisol, 2,4,6-trichlorophenol, 2,3,4,6-tetrachlorophenol, and pentachlorophenol were inactive."

On the other side of the ledger, Vela-Muzquiz and Kasper (1973) reported that microbial flora of fertile soils were incapable of utilizing PCP or NaPCP as a sole source of carbon. When the substances at 40 ppm were added to nutrient broth or soil cultures with yeast extract, the growth of soil bacteria, fungi, actinomycetes, etc., was completely inhibited. Cultures of Flavobacterium and Enterobacter were isolated which grew in the presence of PCP and NaPCP but did not degrade these compounds.

Tyler and Finn (1974) studied the kinetics of growth of a pseudomonad on 2,4-DCP. They reported that:

> "Highest specific growth rate using 2,4-DCP was 0.12/h at 25°C in a pH range from 7.1 to 7.8. Growth was strongly inhibited by 2,4-DCP above a concentration of 25 mg/liter. Growth on 2,4-DCP was described by Monod kinetics at subinhibitory concentrations but the inhibition by 2,4-DCP exhibited an unusual linear response to substrate concentration, and did not fit a model based on noncompetitive inhibition. The lag phase of batch cultures

was found to depend on both 2,4-DCP concentration and prior adaptation of the inoculum."

Gee and Peel (1974) reported that in a co-metabolic system:

"Isolates of 26 fungal species from broiler house litter were screened for their ability to metabolize and methylate 2,3,4,6-tetrachlorophenol over a five-day period, by adding the chlorophenol to cultures after mycelial pellets were established on a complete growth medium. Under these conditions, 99 of the 116 isolates tested metabolized the chlorophenol and 68 of these produced 2,3,4,6-tetrachloroanisole. The proportion of the chlorophenol methylated to the chloroanisole differed widely with the isolate, even within species. The highest methylation was observed with Penicillium corylophilum; certain other isolates, notably of P. brevicompactum, metabolized almost all the chlorophenol without forming the chloroanisole. Progress studies with these species suggested that there is more than one route for the metabolism of 2,3,4,6-tetrachlorophenol. In tests with suspensions of mixed bacterial populations from broiler house litter, the chlorophenol was metabolized but no methylation was detected."

Pitter (1976) determined the biological degradability of 94 aromatic compounds including o-CP, p-CP, and 2,4-DCP. These organic substances were a sole source of carbon for the bacteria of the activated sludge adapted for 20 days. The amount of the compounds removed by biological action were 95.6, 96.0, and 98.0%, respectively. Pitter (1976) suggested that the factors affecting biological degradability could be divided into three groups:

"1. physico chemical factors (temperature, solubility, degree of dispersion of the compound in the medium, pH, dissolved oxygen). 2. biological factors (history of the microbial culture, its age, manner and time of its adaptation, toxicity of the compound, effect of other substrates) and 3. chemical factors (size of molecule, length of chain, kind, number and position of substituents in the molecule, stereochemistry)."

PCP has been used on upland rice fields in Japan and usually has been applied once or twice per year and applications continue for several consecutive years. (The use of PCP has been prohibited on low land rice fields because of its toxicity to fish.) Watanabe (1977), who noted the lack of field microbiological data, conducted field

experiments to study the build-up of PCP-decomposing as well as PCP-tolerant microorganisms after application of PCP to the same plots for three consecutive years. Two plots were treated with 10 kg PCP/ ha and two plots with 20 kg PCP/ ha; four plots were left untreated. Watanabe (1977) observed that within 6 weeks after an initial application of PCP, PCP-decomposing microorganisms increased by about 3 orders of magnitude. The increased populations, which carried through to the following spring, were maintained for the next 2 years at levels which never fell below the initial number. In 1972 and 1974, PCP-tolerant bacteria increased immediately after the PCP application until 2 weeks later and then declined. In 1973, the higher population of PCP-tolerant bacteria in PCP-treated plots were maintained until autumn. This variation could not be explained.

Suzuki (1977) examined the metabolism of PCP - ring ^{14}C by a microorganism (Pseudomonas sp.) isolated from soil. He stated that:

> "The microorganism degraded PCP-^{14}C rapidly, and released $^{14}CO_2$ equivalent to approximately 50% of the PCP-^{14}C added to the bacterial cell suspension in 1 hour of incubation. The results of amino acid analysis of the bacterial cells incubated with PCP-^{14}C showed that radioactive carbon derived from PCP-^{14}C was incorporated rapidly into the cell constituents, and that the pattern of ^{14}C-amino acids in the cell constituents was not much different between the 15 minute and 24 hour incubation periods. Intermediate metabolites of PCP isolated from the incubation medium were identified as tetrachlorocatechol and tetrachlorohydroquinone by spectral analyses."

Suzuki (1977) further stated:

> "These result are an implicit proof of the cleavage of the benzene ring. It is generally accepted that in the metabolism of aromatic compounds by microorganisms the conversion of the substrates to ortho or para dihydroxyphenol derivatives occurs prior to the cleavage of the benzene ring (Evans, W.C., J. Gen. Microbiol., 32, 177 (1963)). Therefore, the isolation and identification of tetrachlorocatechol and tetrachlorohydroquinone suggested that these products appear to be the intermediate metabolites before the ring cleavage in the degradation of PCP. Since TCHQ and TCC degraded rapidly as soon as they were produced, it was possible that a significant level of accumulation of these metabolites did not occur."

Gibson and Bourquin (1977) at a Conference on Water Chlorination discussed the microbial degradation of halogenated hydrocarbons and in the abstract of their paper noted:

> "Certain identified products of chlorination treatments, mono and dichloro substituted aromatic hydrocarbons, may be expected to be degraded by biochemical mechanisms established for pre-existing halogenated substrates. However, increased halogenation of organic compounds results in decreased degradation potential and microorganisms can be expected to do little more than modify the structure of persistent chemicals. Removal of halogen substitution must occur prior to the microorganism being able to use the compound. A number of different enzyme systems, some specific and some fortuitous, have been identified which effect dehalogenation. Halide replacement by hydroxyl can occur by hydrolysis; reductive dechlorination in anaerobic environments can occur, and dehydrodehalogenation can occur resulting in olefin production.
>
> "With the present available information, some predictions can be made concerning degradation of halogenation organics; however, much remains to be learned before we can predict with some certainty the degradability of most of these compounds."

Rott et al (1979) investigated the decomposition of NaPCP by Alcaligenes eutrophus, Aeromonas hydrophila var. hydrophila and var. anaerogenes, Azotobacter chroococcum, Azotobacter vinelandii, Flavobacterium aquatile, Pseudomonas fluorescens, Cytophaga johnsonae, Corynebacterium aquaticum, Brevibacterium testaceum, and Arthrobacter globiformis. Metabolites were characterized and identified by comparing them with authentic substances. This preliminary study, prior to a total radiochemical balance study with ^{14}C-labeled PCP, showed that PCP-acetate, the major metabolite, was formed by six strains of microorganisms in amounts of 0.01 up to 6.2% of the starting compound. Other metabolites were present in much smaller amounts (e.g. pentachloroanisole was formed by five strains of microorganisms but the maximum conversion was 0.02% of the starting material). The other transformation products amounted to less than 1% in every case. Rott et al (1979) noted that ten metabolites had been isolated and identified. They suggested that the main steps to metabolize NaPCP were as follows: methylation and/or acylation of the hydroxyl groups, dechlorination to tetrachlorophenols,

dechlorination and methylation to tetrachloroanisoles, and hydroxylation to tetrachlorodihydroxybenzenes, followed by acylation to the diacetates.

6.2 Transport of Chlorophenols

Letey and Farmer (1974) reviewed the transport of pesticides in soil. In a general introduction of the subject they defined mass flow and diffusion, the two main processes by which chemicals which enter the soil environment become distributed. They suggested that mass flow occurs as a result of external forces on the medium in which the chemical molecules are either dissolved or suspended, present in the vapor phase, or adsorbed. As a result the chemicals, including pesticides, move as the water and/or soil particles move. They considered mass flow due to air movement in soil as negligible.

In apposition to mass flow is diffusion. Letey and Farmer (1974) defined diffusion as "the process by which matter is transported as a result of random molecule motion caused by the molecule's thermal energy. The random molecular motions gradually cause the molecules to become uniformly distributed in the system. There is, therefore net movement from positions of higher concentration to positions of lower concentration."

The transport of CPs will be discussed primarily under the major mechanisms, adsorption and diffusion, with some attention to other factors such as leaching, and surface and air movement.

6.2.1 Adsorption. The transport of CPs after they enter the soil, water, or air media can be affected by one or more mechanisms, one of which is deposition by adsorption. As stated by Howard et al (1978):

> "Adsorption is a chemodynamic parameter similar to the water solubility, partition coefficient, and dissociation constant of a chemical and has significant impact on transport processes. Adsorption is usually determined by shaking soil (or sediment) with an aqueous solution of the chemical until equilibrium is reached. The adsorption coefficient, K, varies considerably from soil to soil and is particularly affected by the soil organic matter and clay content."

Haque and Freed (1974) reviewed the chemodynamics of adsorption as it relates to pesticides in the air and soil. Chlorinated phenols were not discussed specifically but it was stated that "Although the bulk of the adsorption may be from solution, adsorption to a certain extent also occurs from the chemicals present in the

vapor state." They pointed out that: "Adsorption of pesticides from aqueous solution is in most instances an exothermic process." The magnitude of the heat of adsorption was considered to give a qualitative indication of the adsorption system, i.e., physical (such as intercalation), chemisorption, or hydrogen-bond formation. Haque and Freed (1974) further note that:

> "In general, for pesticide adsorption the heat of adsorption ranges only a few kcal/mole, indicating a physical type adsorption or in some cases weak hydrogen bonding between adsorbate and the surface. Formation of a chemical bond or chemisorption has rarely been observed in neutral pesticide-soil systems. For most of the neutral organic molecules the adsorption is of the physical type, in which there is first the formation of a monolayer on the surface followed by a build-up of multilayers."

Hartley (1964) discussed the effect of adsorption on the availability of a herbicide in soil as follows:

> "The primary effect of adsorption of herbicide on the soil particle is to reduce, sometimes to a very small fraction of the whole, the concentration of herbicide freely available in the soil water."

A highly adsorptive soil, although initially reducing activity of a compound, such as PCP (Choi and Aomine, 1972), would tend to prolong its activity, with the period of activity being determined by the amount of the compound adsorbed on the soil as a utilizable reserve (Hartley, 1969).

Choi and Aomine (1974a) who investigated the adsorption of PCP by soils, summarized their findings as follows:

> "The adsorption studies using soils various in the species of clay minerals and organic matter content indicate:
>
> 1) That apparent adsorption occurs to the greatest extent on the strong acid soil system compared to the moderate acid soil system, regardless of the species of clay mineral and organic matter content. And there is no adsorption on the slightly acid or neutral soil system.
>
> 2) The apparent adsorption involves adsorption of molecules and/or anions and precipitation of molecules in the micell and the external liquid phase.

3) The magnitude of adsorption occurs in the decreasing order of humusallophanic, allophanic, montmerillonitic, and halloysitic soils.

4) The major factor governing the magnitude of apparent adsorption is pH."

Choi and Aomine (1974b) clarified the mechanism of PCP adsorption and precipitation in soils. They noted that the precipitation of PCP took place around base-unsaturated clay particles when the concentration of PCP exceeded solubility; and that the adsorption of PCP involves anion exchange reaction as well as physical adsorption. They further stated that soil type determined whether the PCP was primarily adsorbed as anions or as molecules. It is important to note that the research was carried out with PCP in hexane because the amount of PCP absorbed on soils from aqueous solution was so small that instrumental analyses such as infrared spectroscopy, X-ray diffraction, and differential thermal analyses were inapplicable for studying the interaction between clay and PCP.

Kaufman (1976) reviewed the chemistry, degradation and mode of action of phenols as herbicides. He considered adsorption as one of the principal factors affecting the fate and behaviour of pesticides in soil and water systems. Kaufman (1976) briefly summarized the work of Nose (1966), Nose et al (1963), and Nose et al (1964), who had investigated PCP adsorption on 14 soils and 4 types of clay minerals. A positive correlation was observed between the PCP adsorption coefficient, K, and soil factors such as cation exchange capacity, heat of wetting by water, and heat of wetting by toluene. PCP adsorption was pH dependent (i.e. the lower the pH the greater the adsorption of PCP). They suggested that PCP adsorption was based on the attractive force between the charge of the -OH group induced by polarization and the surface charge of the soil particle.

As stated by Kaufman (1976), Green and Young (1970) had found PCP to be most mobile in high pH soils and least mobile in acid soils where adsorption would be greater.

Su and Lin (1971) had also demonstrated that the herbicidal efficacy of PCP was affected by pH. In the pH range 3 - 8, the lower the pH the less was the efficacy of PCP. Its efficacy also decreased with an increase in organic matter content and surface area of the soil.

Choi and Aomine (1972) investigated the effect of soils on PCP and summarized their results as follows:

"The effect of soils on the activity of pentachlorophenol was investigated by bioassay using wheat seedings and 10 soil samples of various clay minerals. Sodium pentachlorophenate added to soil suspensions disappeared from the soil solution to a greater or lesser extent, owing to adsorption by the soil colloid and also, probably partly due to precipitation in the case of a very low pH. The pH value of soil is of primary importance in the decrease of pentachlorophenol concentration from the soil solution, and the nature and content of colloid is of secondary importance.

"The amount of pentachlorophenol in the soil solution is roughly proportional to the toxicity of the pentachlorophenol-soil suspension to the plant. Absorbed pentachlorophenol, is obviously lower in toxicity than dissolved pentachlorophenol, although it retains some degree of activity.

"Pentachlorophenol is less active and more persistent in acid soils than in neutral ones. Humus-rich soils especially allophanic ones make pentachlorophenol less soluble as compared with mineral soils, resulting in reduction of toxicity of pentachlorophenol."

6.2.2 Diffusion and Volatilization. Letey and Farmer (1974) concluded that the diffusion of pesticides through soils was dependent on a number of soil properties including water content, bulk density, air-filled porosity and temperature, and on certain chemical properties of the pesticide such as solubility, vapor density, and the diffusion coefficient.

However, the NRCC publication on phenoxy herbicides (NRCC, 1978) noted that "The diffusion process does not contribute significantly to the long distance transport of herbicides in the soil medium, but would be important in herbicide dispersion in still water." The same statement would aptly apply to CPs. In addition it was stated that "Diffusion, however, plays an important role in a number of processes, especially where localized dispersion is essential, such as in volatilization, availability, degradation, etc."

Letey and Farmer (1974) in their review stated that: "Diffusion is a primary process controlling volatilization of soil incorporated pesticides. As soon as loss occurs at the soil surface, a concentration gradient is established causing pesticide diffusion to the surface replacing that lost by vaporization". They concluded from research of other workers that volatilization losses from soil were one of the important pathways for the disappearance of pesticides.

In a more general statement, Spencer et al (1973), who had reviewed pesticide volatilization, concluded that:

> "Volatilization is obviously a major pathway for loss of applied pesticides from plant, water, and soil surfaces. Progress is being made on quantitatively evaluating the factors affecting volatilization and developing mathematical models to predict volatilization rates under various conditions of field application. The vapor pressure of the pesticide is the major factor influencing volatilization rate. Many other variables affect volatilization rate, but most of them do so through their effects on vapor pressure, rate of movement away from the evaporating surface, or rate of movement of the pesticide to the surface of the soil."

Howard et al (1978) briefly summarized the factors affecting volatilization:

> "Volatilization of a chemical from soil and water is dependent on a number of environmental factors (temperature, humidity, soil type, soil moisture content, evaporation, mixing, air movement) as well as the vapor pressure of the chemical. Higher rates of volatilization occur with higher temperature (higher vapor pressure), decreased soil organic matter (decrease of adsorption sites), and increased soil moisture."

Kaufman (1976) stated that volatilization was a significant mechanism for loss of some pesticides in soil. He remarked, however, that the significance of the mechanism of volatilization in phenolic pesticides in the soil had not been adequately determined.

Rate of loss of a pesticide from the soil can, in part, be a function of the rate of evaporation of water from the soil, as explained by Hartley (1969). He suggested that:

> "When the pesticide is distributed in the soil, evaporation of water can accelerate that of the water-soluble pesticide; the mechanism lies in capillary flow of solution and not in the evaporation process itself."

6.2.3 Leaching. Leaching is a term used to describe a transport process for the movement of a chemical with water in a soil matrix (Haque and Freed, 1974). Leaching usually takes place in a downward direction, although it can occur in both an upward and lateral direction. Leaching and diffusion are interrelated, as pointed out in the review of Haque and Freed (1974), who stated that where water percolation is rapid, the bulk movement of the chemical will be in the direction of water flow, but as the water percolation slows, diffusion becomes a greater factor. They also stated that the factors

controlling leaching were 1) water solubility of the chemical, 2) adsorption, 3) soil type, and 4) moisture and percolation velocity.

The physical-chemical parameters associated with leachability of a chemical were stated by Howard et al (1978) as follows:

> "Leachability in soil is related to the dissociation constant (thus pH of soil is important), water solubility (highly water-soluble compounds are more mobile), charge distribution (in the case of organic cations), molecular size, and polarity (more polar, more affinity for water - more mobility). However, as with evaporation, soil properties, rainfall, etc., can have considerable impact on a chemical's soil mobility."

Kuwatsuka (1972) noted that although photodecomposition was the major degradation process of PCP in rice paddy fields, a considerable portion of the PCP applied to the flooded paddy fields infiltrated into the soil with percolating water. The Weed Science Society of America (WSSA) Herbicide Handbook (1974) also notes that NaPCP leaches readily in soils.

Kaufman (1976) briefly summarized the interrelationships of adsorption, volatilization, and leaching of phenols in soil as follows:

> " . . . phenols exist as the free acid in acidic soils and in the presence of mineral clays would be strongly adsorbed. In the absence of anionic adsorption sites volatilization of the free acid would readily occur, particularly at elevated temperatures. Although leaching occurs in both acidic clay and sandy soils (87), leaching may occur more readily in alkaline soils."

In a review of depletion and fate of fungicidal wood preservatives in the environment, Stranks (1976) commented on the permanence (i.e. persistence) of PCP in treated wood as follows:

> "In general PCP does not leach, especially if applied by oil carrier. Permanence is related to the low solubility of PCP in water. No extensive study of the fixation of this preservative in wood has been made, and this in itself shows that little leaching occurs. However, Unligil (1968) has shown that PCP was removed from blocks of treated wood during steam sterilization. Similar but long-term leaching can probably be expected for soil and water exposures under outdoor hot humid cyclical weather conditions.

"There is some question concerning the permanence of PCP in wood treated by the Cellon process, in which the carrier is liquefied petroleum gas (LPG). Recovery of the LPG during treatment practice may affect PCP distribution and fixation and result in more leachability.

"The current practice of incorporating water-repellent waxes and oils with carriers to prevent blooming (blooming causes loss of PCP) undoubtedly reduces the rate of water absorption in the treated material. The reduced mechanical working of the wood may contribute to greater permanence of PCP under conditions of service."

6.2.4 Exudation. There has been an apparent lack of information on depletion and/or persistence of preservatives, including PCP, in treated round wood used in freshwater, considering the period of time preservatives have been used (Kelso and Behr, 1977). Most of the literature has been concerned with service tests of marine pilings treated with creosote.

Kelso and Behr (1977) investigated depletion of PCP and creosote from round southern pine in freshwater. The treated green southern pine pole sections had been exposed in freshwater for 5.5 to 13 months followed by a period out of water. Analysis of 0.5 to 1.0 inch zones from outer to inner wood, prior to and following exposure, indicated that overall losses of PCP were small and confined mostly to the outer 0.5 inch zone. The losses that did occur were from exudation of oil-PCP as well as dissolution of PCP by the large quantity of water in contact with the wood surface.

6.2.5 Surface Movement. The greatest opportunity for surface movement of CPs is when they are present in surface run-off waters, both in solution and adsorbed onto soil particles. Chlorinated phenols primarily enter the run-off waters via the processes of leaching from treated material or, of more concern environmentally because of volumes of material, from overflows from wood-treatment plants' wastewater holding ponds. A case history of the latter was detailed by Pierce and Victor (1978). Data obtained in this study on levels and persistence of PCP are presented in Section 5.1.1, 5.1.2 and App. 8, Sect. 8.1, along with other examples.

6.2.6 Atmospheric Movement. Howard and Durkin (1973) noted that although CPs are considered water and soil contaminants, the moderate volatility (Table A1-1) of these compounds would suggest that atmospheric transport may be a significant route. They also pointed out that there had been a general lack of monitoring data.

Haque and Freed (1974) in a general discussion on behaviour of chemicals in the atmosphere noted that a chemical's entry into and transport through the atmosphere will depend on several factors, including:

> "(1) vapor pressure and the heat of vaporization of the chemical, (2) the partition coefficient between the atmosphere and any other phase, and (3) the air flow mass which will transport any chemical dispersed in the atmospheric phase."

Howard et al (1978) stated that once a chemical has entered the atmosphere, either directly or by volatilization, it can be carried long distances by wind currents before eventual deposition. In a summary statement they noted that the mechanisms of atmospheric deposition included: a) adsorption to particulate matter followed by gravitational settling or rain washout, b) washout by being dissolved into rain.

Ifeadi (1975) compiled information on the major air contaminant emissions from the manufacture of PCP. Information was supplied on systems, costs, and air contaminant recoveries. The summary stated that the quality of air contaminant emissions in the manufacture of PCP was significant since large numbers of compounds were emitted at high emission rates. The particulate collection systems were not always adequate.

Measurable quantities of PCP detected in melt from samples of snow collected during the winter of 1977-78 at locations throughout Ontario, demonstrated that aerial transport of PCP occurs year-round (also see Sect. 5.1.1) (Strachan, 1979).

APPENDIX 7

APPENDIX 7

7 GENERATION, DEGRADATION, AND TRANSPORT OF POLYCHLORINATED DIBENZO-p-DIOXINS AND POLYCHLORODIBENZOFURANS IN THE ENVIRONMENT

This appendix contains brief accounts from the published literature on the environmental generation, degradation and transport of PCDDs and PCDFs. The majority of the references are post-1972, when it became possible and feasible to detect, at ppt levels, the impurities, PCDD and PCDF, in environmental samples.

Information on formation of these impurities through photolysis, pyrolysis, and thermal generation is presented. Experimental evidence for degradation of PCDDs and PCDFs via photolysis, and thermal and microbial degradation has been reported in the literature and is summarized in this appendix. Notes on the transport of PCDDs and PCDFs in soil, sediment, water, and air have also been included.

7.1 Environmental Generation of PCDD and PCDF

7.1.1 Photolysis. The photochemical generation of chlorinated dioxins was reviewed by Crosby et al (1973), Helling et al (1973), and again by Crosby and Wong (1976).

Kearney et al (1972) reported that photolysis of 2,4-DCP in water at wavelengths <300 nm had failed to yield dioxins. Similar negative results (i.e. no dioxin production), were reported by Plimmer and Klingebiel (1971) when they investigated irradiation of 2,4-DCP in the presence of a sensitizer, riboflavin, and oxygen, although the phenols were efficiently consumed. The reaction products were tetrachlorophenoxyphenols, and tetrachlorodihydroxybiphenyls (Fig. A7-1). It was noted that despite extensive destruction of the phenols their conversion to dimeric products was less than 5%.

Plimmer et al (1973) went on the report that:

> "By contrast, alkaline aqueous solutions of 2,4-dichlorophenol, 2,4,5-trichlorophenol, and pentachlorophenol rapidly colored when exposed to sunlight. The isolation of several photolysis products predictably derived by hydrolytic and reductive reactions (including tetrachlororesorcinol from pentachlorophenol)

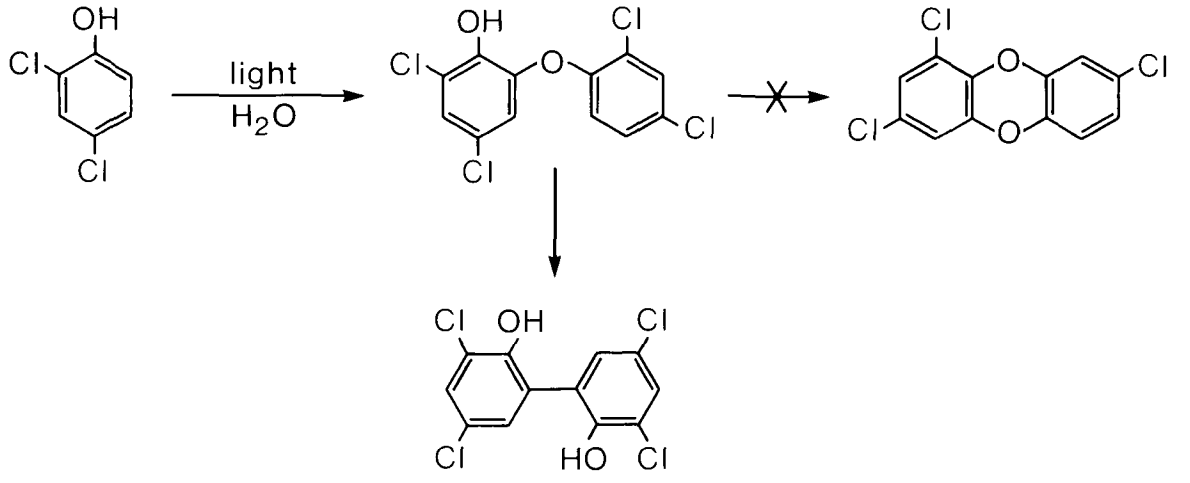


FIGURE A7-1 IRRADIATION OF 2,4-DCP (Plimmer et al, 1973)

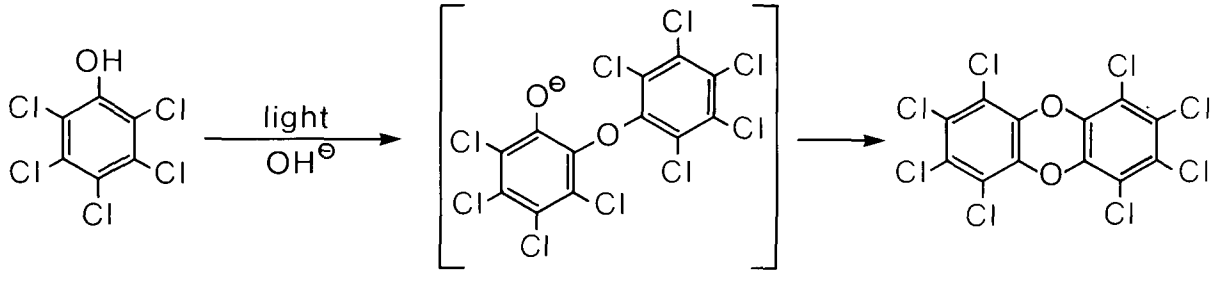


FIGURE A7-2 PHOTOLYSIS OF PCP (Plimmer et al, 1973)

> suggested strongly that a nucleophilic ionic mechanism is operative (Fig. A7-2). Pure dioxin-free PCP, irradiated as a 1000 ppm solution in aqueous sodium hydroxide with light in the 300-350 nm region, gave a neutral benzene-soluble extract which contained octachlorodibenzo-p-dioxin as shown by GLC/MS. Although the yield in repeated experiments was not consistent and the maximum concentration was only 36 ppm in any single irradiation, a smaller amount of an unidentified neutral constituent also was present whose retention time on GLC corresponded to that of a heptachlorodibenzo-p-dioxin."

Concurrently, Stehl et al (1973) had observed that under controlled laboratory conditions the photolytic degradation of NaPCP buffered at pH 8 was very rapid and produced no more than 0.03% OCDD.

Nilsson et al (1974) reported that photolysis of pentachloro-2-phenoxyphenol, which was one of the impurities found in commercial 2,4,6-TCP, gave 1,2,3,8-TTCD, two TCDDs, a DCDD, and a DCDF. Nortsrom et al (1976) found from further investigation that irradiation of another abundant impurity, polychlorinated diphenyl ethers, in commercial polychlorinated phenols resulted in formation of PCDFs (Fig. A7-3). They concluded that:

> "The photochemical formation of the highly toxic PCDFs from the much less toxic diphenyl ethers can be a reaction of environmental significance. . . . It is therefore recommended that the level of chlorinated diphenyl ethers in chlorophenols should be minimized."

Following their review of photochemical generation of CDDs, Crosby and Wong (1976) concluded:

> "The alkaline character of many natural waters suggests that relatively acidic chlorophenols often will occur in ionic form in the environment. although they also are introduced in that form in many instances (e.g., sodium PCP). Operation of the photonucleophilic mechanism would permit photochemical generation of chlorinated dibenzodioxins provided that phenol concentrations were high enough for the bimolecular reaction to proceed efficiently, as apparently has been the case in PCP-treated rice field (Munakata and Kuwahara, 1969). The nature and levels of the resulting dioxins would depend greatly upon the rate of dioxin photolysis, and the detection of hexachloro

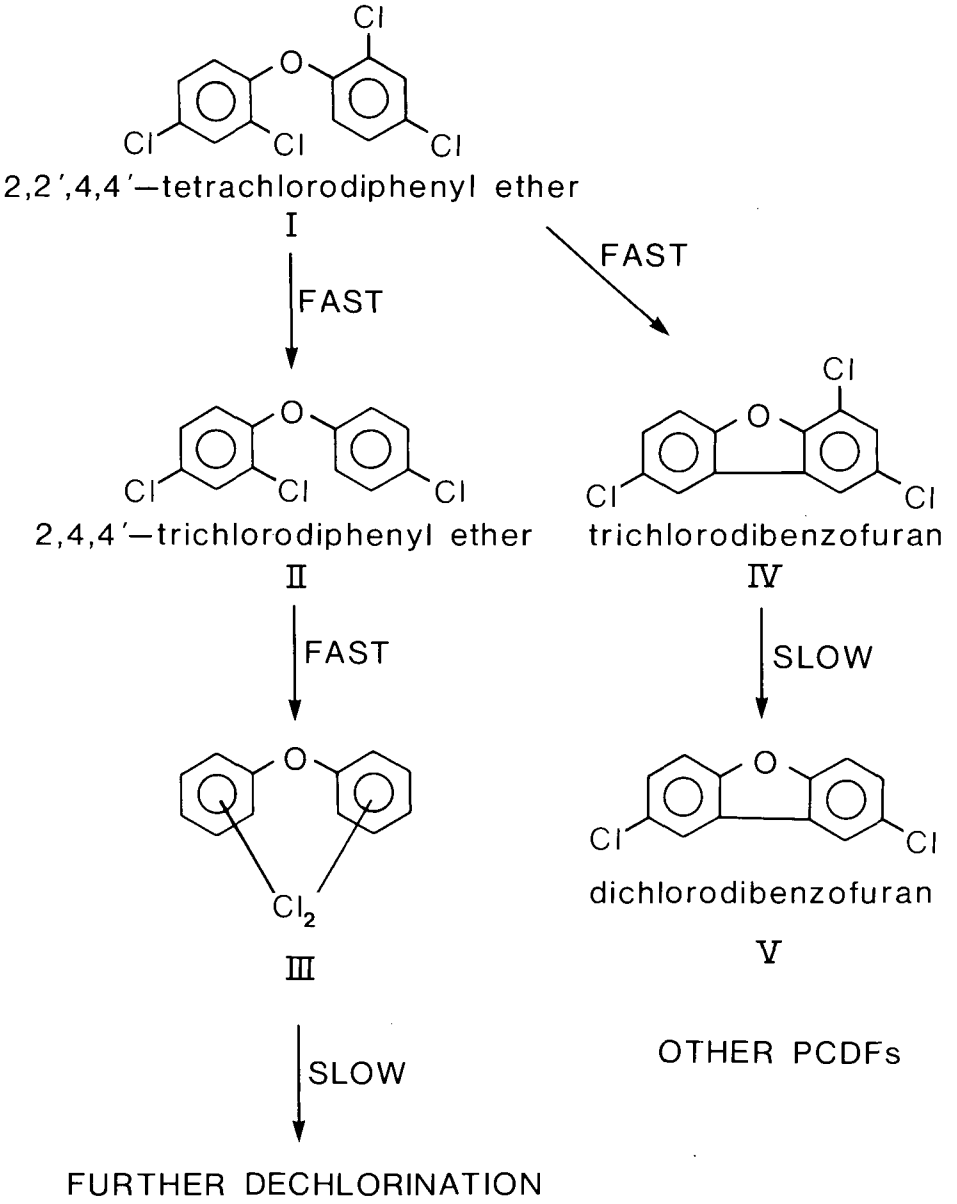


FIGURE A7-3 PHOTOLYSIS OF 2,2',4,4'-tetrachlorodiphenyl ether (Norstrom et al, 1976)

dioxin after irradiation of pure PCP in water raises the intriguing possibility that the dioxins reported (Baughman and Meselson, 1973) to occur in environmental samples might actually arise by photochemical generation rather than by direct introduction. However, at the nanomolar chlorophenol concentrations normally encountered in chemically polluted (Zigler and Phillips, 1967) or chlorine-purified (Burttschell et al, 1959) water, it seems probable that more concentrated or reactive environmental nucleophiles such as hydroxide ions or ammonia usually would participate preferentially in the photonucleophilic displacements of chloride (Crosby et al, 1972) to provide less toxic products."

Akermark (1978) reviewed the literature on photochemical reactions of phenoxy acids and CDDs and commented on the data of Plimmer and Klingebiel (1971). He considered that the formation of the predioxin (Fig. A7-1) from photolysis of 2,4-dichlorophenol was indicative of a phenolic coupling, rather than a photonucleophilic reaction and that the riboflavin served as a dehydrogenating agent rather than as a sensitizer. Following this line of reasoning he suggested that the end result would be a very inefficient route for formation of predioxins in the environment. In addition, the lack of proof of CDD formation (Crosby and Wong, 1973; Plimmer and Klingebiel, 1971), could be due to not only inefficient formation and efficient destruction of CDDs, but could also be attributed to lack of a sufficiently sensitive analytical method. To overcome this, a very sensitive mass fragmentographic method was under development in mid-1978 in Sweden (Akermark, 1978).

To produce easily and safely prepared qualitative reference standards for several PCDDs and PCDFs, Buser (1976) subjected OCDD and OCDF to UV and γ-irradiation to produce lower chlorinated CDDs and CDFs. For example, the major products formed when OCDD was exposed to UV rays for 24-h were HCDDs and heptaCDDs, plus significant amounts of pentaCDDs and trace amounts of TCDDs. γ-Irradiation of OCDD for 4-h primarily produced PnCDDs, HCDDs, and HpCDDs. Dechlorination of OCDF by UV irradiation for 24-h led primarily to HCDFs, plus lesser amounts of Pn- and HpCDFs, and trace amounts of TCDFs.

Buser (1979a) reported on the formation and identification of TCDDs and PnCDDs from "the UV-photolysis of two isomeric, unambiguously assigned hexa-CDDs, namely 1,2,3,6,7,8- and 1,2,3,7,8,9-hexa-CDD." Buser's (1979a) diagram of the main reaction pathways of the photolytic dechlorination of the two HCDDs to five PnCDDs and five TTCDs is shown in Fig. A7-4.

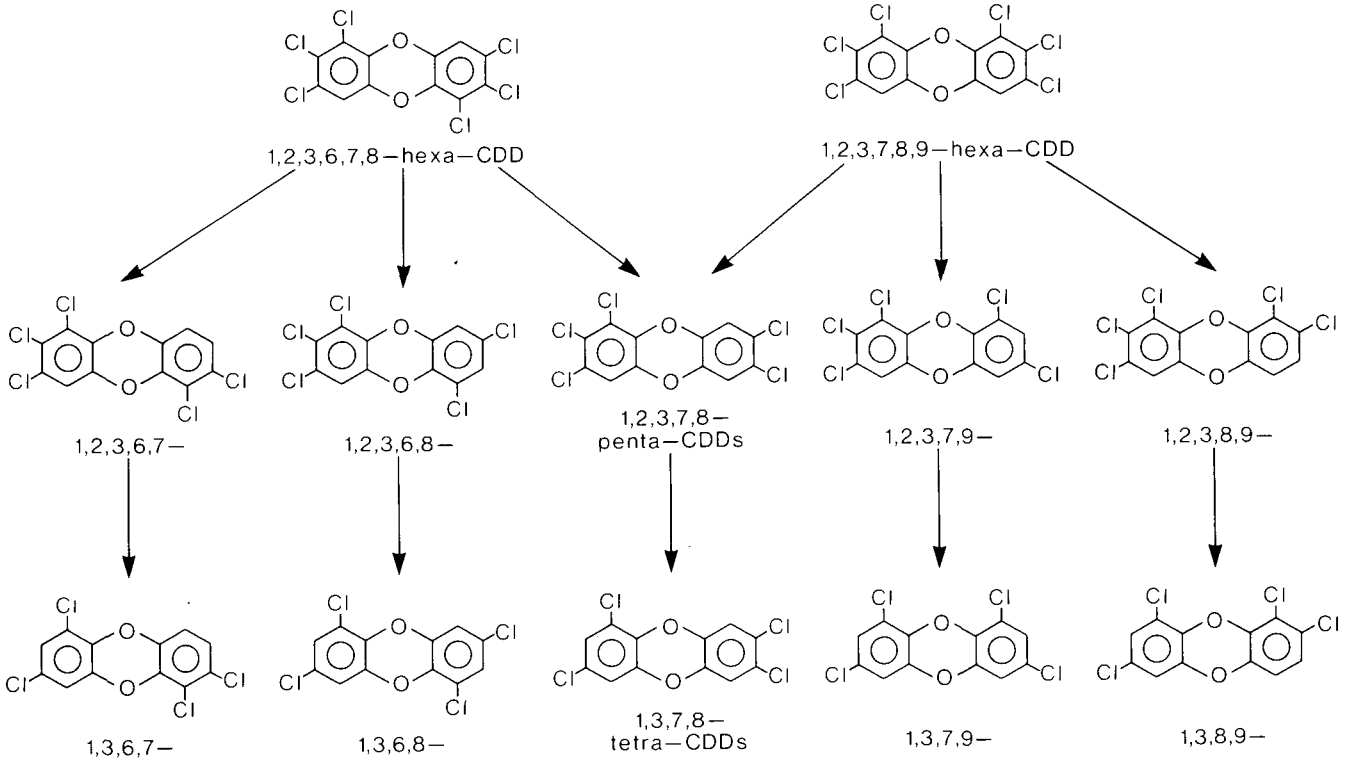


FIGURE A7-4 MAJOR PHOTOLYSIS PATHWAYS LEADING TO TETRA- AND PENTA-CDDs FROM 1,2,3,6,7,8- AND 1,2,3,7,8,9-HEXA -CDD. (Buser, 1979a)

Lamparski et al (1980) in a laboratory study determined that OCDD was formed by photolytic condensation of PCP on a wood substrate. Treatment solutions were made up with technical PCP, Dowicide EC-7 antimicrobial, and purified PCP. In the conclusion to their report, Lamparski et al (1980) state that,

> "The analytical results indicate that the photolytic reactions at the surface of Dowicide EC-7 treated wood with methylene chloride solvent can produce CDDs at concentrations that approach those in technical PCP. If a hydrocarbon oil, such as P-9, is used as a carrier, the OCDD concentration on the wood surface is only about 1% of the amount in typical technical PCP."

7.1.2 Pyrolysis and Thermal Generation. Information on thermal generation of halodibenzo-p-dioxins has been somewhat sporadic. Kulka (1961) reviewed the early literature and reported on a method for the conversion of PCP to OCDD in almost quantitative yield by heating the phenol in the presence of an initiator. Langer et al (1973), of Dow Chemical Co., investigated the thermal properties of several CPs and derivatives by differential thermal analysis and ms (in bulk reactions). They summarized their findings as follows:

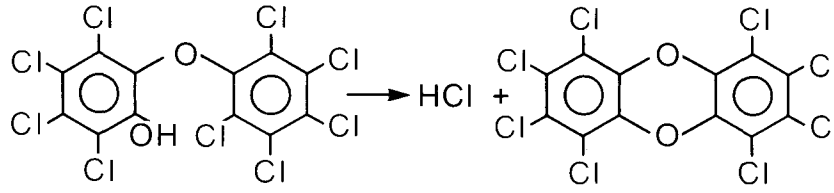
> "In summary thermal decomposition of chlorinated phenols does not generally lead to dioxins. There are, however, several conditions which by themselves or combined would favor dioxin formation. First, of all chlorinated phenols either in bulk or in solution, only pentachlorophenol produced measurable amounts of dioxin. Secondly . . . only sodium salts in solid state reactions produced dioxins in reasonable yields. In contrast, the silver salt of pentachlorophenol . . . undergoes an exothermic decomposition at considerably lower temperatures and produced only higher condensed materials. No dioxin was detected."

Stehl et al (1973), also associated with Dow Chemical Co., reporting on the products derived from burning PCP treated materials, stated that:

> "Analysis of combustion products of wood and paper treated with pentachlorophenol indicated no increase and possibly a decrease in octachlorodibenzo-p-dioxin concentration while paper treated with sodium pentachlorophenate increased slightly in octachlorodibenzo-p-dioxin concentration after combustion."

Crosby et al (1973) demonstrated in a simulated situation that enough concentrated heat was generated in the burning or pyrolysis of PCP-treated scrap plywood or mill wastes to convert the PCP to predioxins (phenoxyphenols) which when volatilized would form CDDs and polyphenyl ethers. Wood which had a pre-pyrolysis content of 1 ng/g of OCDD had approximately twice that amount following pyrolysis. Hepta-and hexa-CDDs were also present.

In 1972, Rappe and Nilsson had demonstrated the presence of an artifact in the gc determination of impurities in PCP. They had accounted for increased OCDD content in gc analyzed PCP by proposing and confirming that the heated injection block of a gc provided suitable conditions for a ring-closure of an impurity 3,4,5,6-tetrachloro-2-(2,3,4,5,6-pentachlorophenoxy) phenol (I) (Fig. A7-5) in the PCP to OCDD (II). Therefore, reported CDD levels detected by gc in samples prior to 1972 may be higher than actual.



I II

FIGURE A7-5 RING-CLOSURE OF 3,4,5,6-tetrachloro-2-(2,3,4,5,6-pentachlorophenoxy)phenol (I) to OCDD (II) (after Rappe and Nilsson, 1972).

Nilsson et al (1974) presented evidence that a DCDD and a TCDD were the only detectable products from the pyrolysis of chloro-2-phenoxyphenols, an impurity in European commercial 2,4,6-TCP. Because of the decomposition of chloro-2-phenoxyphenols to dioxins and furans through either photolysis (App. 7, Sect. 7.1.1) or pyrolysis, Nilsson et al (1974) suggested that the level of chloro-2-phenoxyphenols in commercial CP formulations should be minimized.

Ahling and Johansson (1977) investigated the combustion of PCP in sawmill wastes. In a pilot scale test, under controlled conditions, no formation of OCDD could be proved. In a follow-up full scale test of combustion of PCP and TTCP, with greater variation in temperature of combustion, shorter transit time, increased load factor, and reduced rate of air flow, there was some indication of formation of OCDD in the flue gas. In further tests reported by Jansson et al (1978), technical TCP, TTCP, and PCP products were mixed with wood chips and burned in a pilot-scale incinerator from which the emitted gases and particulate matter were analyzed for presence of PCDD. The abstract to the report stated that:

> "For the tri- and tetrachlorophenol formulations, large formations of PCDD were found at burning temperatures of 500 - 600°C. At higher temperatures the emissions decreased. Insufficient oxygen supply increased the emission of PCDD. Addition of copper salts to the tetrachlorophenol formulation drastically decreased the PCDD emissions. Likewise, an increase in transit time decreased the amount of PCDD in the flue gases. Combustion of a pentachlorophenol formulation resulted in small PCDD emissions except when burned with insufficient oxygen supply when PCDD with four to eight chlorine atoms appeared in the flue gases."

Ahling (1979) studied the destruction of CPs in a cement kiln with final temperature of 1400 - 1450°C. Emission of CPs was <0.1 mg/kg of CP feed. In the series of tests, peaks were observed on the gc that showed the same retention times as HpCDD, OCDD, and OCDF; because concentrations were too low, confirmation by ms was not possible. No PCDDs or PCDFs with 4 or 6 chlorines were detected.

Rappe et al (1978b) had identified and quantified PCDDs formed during the uncontrolled open burning of leaves and wood wool impregnated with commercial and purified chlorophenates. They suggested that there were three reactions which could yield various and different PCDD isomers: a biomolecular reaction, the dimerization of chlorophenates; and two monomolecular reactions, the dechlorination of higher chlorinated PCDDs, and the cyclization of predioxins. They further concluded that the ratio of isomers formed in burning reactions should be dependent on the chlorophenate concentration. Of the many PCDD isomers observed, there were two isomers with the same retention time and chlorination pattern as the highly toxic 2,3,7,8-TCDD and 1,2,3,7,8-PnCDD, although only as minor constituents, and probably formed from impurities present in the commercial formulations. Rappe et al (1978b), in the same

experiments, were unable to identify discrete isomers of PCDFs present, due to a lack of suitable PCDF standards. They did note, however, that although the general trend was for reduction of PCDF levels during burning, a few individual PCDFs did increase, including an unknown major tetra-CDF. Rappe et al (1978b) also compared formation of 2,3,7,8-TCDD in their study with potential formation of 2,3,7,8-TCDD from various 2,4,5-T precursors. They concluded that:

> "The burning of chlorophenates or material like wood shavings, plywood or waste oil containing chlorophenates seems to be a more important source to environmental pollution by PCDDs including 2,3,7,8-tetra-CDD than the accidental burning of 2,4,5-T derivatives or vegetation treated with 2,4,5-T derivatives."

As pointed out by Buser (1979b) the generation of PCDDs and PCDFs can occur not only from the pyrolyses of CPs but also from the pyrolyses of chlorobenzenes (CBs); additionally, CPs were also formed from the pyrolyses of CBs. To improve the yields of PCDDs and PCDFs, Buser's (1979b) model experiments on the gas phase pyrolyses of CBs were carried out at concentrations corresponding to 1 g/L of air. In his discussion Buser (1979b) (Fig. A7-6) noted:

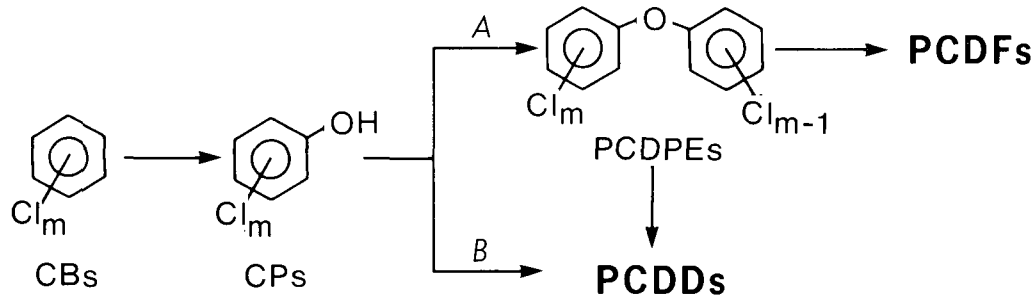


FIGURE A7-6 POSSIBLE ROUTES FOR FORMATION OF POLYCHLORINATED DIBENZO-p-DIOXINS (PCDDs) AND POLYCHLORINATED DIBENZOFURANS (PCDFs) FROM CHLOROBENZENES (CBs) THROUGH CHLOROPHENOLS (CPs) (Buser, 1979b)

"In addition to PCDFs and PCDDs, other chlorinated compounds were also observed from these pyrolyses. All pyrolyzed samples showed the presence of chlorophenols. The degree of chlorination of these phenols was the same and higher than the degree of chlorination of the chlorobenzenes used: tri-, tetra- and pentachlorophenol were observed from trichlorobenzenes, tetra- and pentachlorophenol from tetrachlorobenzenes, and pentachlorophenol also from pentachlorobenzene. These chlorophenols could possibly serve as reaction intermediates in the formation of PCDFs and PCDDs from chlorobenzenes. A reaction of chlorophenol with unreacted chlorobenzene could lead to PCDPEs (route A, below), which are known to form PCDFs (and to a lesser degree PCDDs) upon pyrolysis[6]. However, PCDPEs were not acutally observed in the samples analyzed here. Dimerization of chlorophenols is a further route to PCDDs (route B, below). The former condensation (route A) via PCDPEs may be preferred in these pyrolyses due to the initially much higher concentration of chlorobenzenes present, but for a substantiation of these presumptions and to obtain a more detailed picture of the reactions involved, further work will be required."

Note: [6]Ref. R. Lindahl, C. Rappe and H.R. Buser, in preparation (1979).

A Dow Chemical Co. Chlorinated Dioxin Task Force (1978) concluded from circumstantial evidence that normal combustion processes, such as occur in refuse incinerators, gasoline and diesel engines, fireplaces, charcoal grills, and cigarettes, give rise to particulate matter which contains chlorodioxins. They noted that additional research was planned to answer such questions (e.g. What are the routes by which chlorinated dioxins from these emission sources enter the environment?) (also see Sect. 6.2, 6.3, 6.4, 6.5.2, and 6.6). Because of the current interest in formation of CDDs through normal combustion processes, it is of interest to note that, in 1955, the Battelle Memorial Institute had reported that, for every ton of rubbish incinerated in backyard burners, more than 8 pounds of the emissions were phenols (Anonymous, 1955).

7.1.3 Microbial. Kearney et al (1972) incubated 2,4-DCP and 2,4,5-TCP in Lakeland and Hagerstown soils at 10, 100, and 1000 ppm for 70 days. No evidence was found for in-vivo formation of DCDD or TCDD by microbial condensation reactions. Kearney et al (1972) considered this an important finding:

"... since biosynthetic production from chlorinated phenols or herbicide metabolites would be impossible to regulate under environmental conditions, while the inherent dioxin content in commercial pesticides can be lowered by changes in the manufacturing process."

7.2 Degradation of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans

Kearney et al (1972) investigated the persistence and metabolism of CDDs. Their findings were concisely summarized as follows:

"The persistence of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) was determined in Hagerstown and Lakeland (Md.) soils receiving 1, 10 or 100 ppm TCDD after 20, 40, 80, 160, and 350 days. After one year, 56 and 63% of the originally applied TCDD was recovered in the Hagerstown and Lakeland soils, respectively. Neither 2,7-dichlorodibenzo-p-dioxin (DCDD) nor TCDD could be detected in soils receiving 10, 100 and 1000 ppm of the 2,4-dichlorophenol or 2,4,5-trichlorophenol after 70 days. A polar metabolite of DCDD-^{14}C was detected by thin-layer chromatography in the ethanol soil extract. TCDD is degraded slowly in soils, and TCDD and DCDD are not biosynthesized by microbial condensation reactions."

Helling et al (1973) stated that although persistence is often measured as half life ($t_{1/2}$), since pesticide disappearance from soil typically approximates a first-order reaction, it could be misleading since several physical and biochemical mechanisms simultaneously act on a residue (App. 7, Sect. 7.2.1, 7.2.3, and 7.3.1). Another method used to measure persistence is by the time required for a residue to reach a non-detectable level in the soil. Young et al (1974) who investigated the persistence of TCDD relative to 2,4-D and 2,4,5-T, estimated the $t_{1/2}$ of TCDD at 88 days in alkaline soils, under desert conditions, and in the presence of massive quantities of the herbicides. They concluded that TCDD degraded at a more rapid rate than either 2,4-D or 2,4,5-T.

Botré et al (1979) reported a new method for the decomposition of 2,3,7,8-TCDD, which the authors' suggested may have practical application as a decontamination technique. As noted in the authors' summary, the reaction, which applies not only to 2,3,7,8-TCDD, but also to other substances containing ether bonds together with aromatic rings, requires the use of chloroiodides obtained from different quaternary ammonium salt surfactants and does not need the presence of light.

7.2.1 Photolytic. Crosby et al (1971) investigated the photodecomposition of CDDs and reported that:

> "The toxic herbicide impurity 2,3,7,8-tetrachlorodibenzo-p-dioxin and its homologs decomposed rapidly in alcohol solution under artificial light and natural sunlight, the rate of decomposition depending upon the degree of chlorination. However, photodecomposition was negligible in aqueous suspensions and on wet or dry soil."

Plimmer et al (1973) reviewed the photochemistry of CDDs and stated that:

> "Rate measurements showed that 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is more rapidly photolyzed in methanol than octachlordibenzo-p-dioxin, and subsequent reductive dechlorination is accompanied by ring fission. Pure dibenzo-p-dioxin gave polymeric material and some 2,2'-dihydroxybiphenyl on irradiation."

Similar findings were reported by Stehl et al (1973). The 2,7-DCDD and 2,3,7,8-TCDD were subject to rapid photolytic decomposition under artificial sunlight. The OCDD was considerably more stable towards UV radiation. Under simulated environmental conditions, Gebefugi et al (1977) observed the photodecomposition of TCDD from 100% to 2% within 4 days. Crosby and Wong (1977) also investigated the environmental degradation of 2,3,7,8-TCDD (i.e. they examined the degradation of 2,3,7,8-TCDD in formulated material rather than in a pure form). They stated that:

> "Herbicide formulations containing known amounts of 2,3,7,8-tetrachlorodibenzo-p-dioxin (PCDD) and exposed to natural sunlight on leaves, soil, or glass plates lost most or all of the TCDD during a single day, due principally to photochemical dechlorination. Despite the known persistence of pure TCDD, it is not stable as a contaminant in thin herbicide films exposed to outdoor light."

Crosby and Wong (1977) listed the three essential requirements for significant dioxin breakdown:

1) dissolution in a light transmitting film,
2) the presence of an organic hydrogen-donor such as a solvent or pesticide, and
3) exposure to ultraviolet light.

The practical application of this was their suggestion that TCDD might be removed from contaminated surfaces by treatment with a low volatility solvent and ultraviolet light.

Botré et al (1978) investigated the solubilization and photodecomposition of 2,3,7,8-TCDD in aqueous solutions in cationic, anionic, and nonionic surfactants. They observed that a cationic surfactant, 1-hexadecylpyridinium chloride (CPC), acted as an energy transfer agent in the photodecomposition process, and increased the TCDD decomposition rate. They suggested that a method utilizing CPC would be appropriate and effective for the decontamination of buildings, furniture, and personal belongings from TCDD.

Dobbs and Grant (1979) reported on studies concerned with the photolysis of OCDD, HpCDD, and some HCDD. They demonstrated that the structural isomers which were expected to be the most toxic were most rapidly photodecomposed. Dobbs and Grant (1979) stated:

> "There are two groups of chlorine atoms in OCDD: those bonded to carbon atoms in the 1-position, which are adjacent to an oxygen substituent (1,4,6 and 9 in Fig. A7-7) and those bonded to carbon atoms in the 2-position (2,3,7 and 8 in Fig. A7-7). The half lives (Fig. A7-7 and A7-8) indicate that CDDs with chlorine atoms in the 2-position photlyse more rapidly than those with chlorine atoms in the 1-position."

With reference to photodecomposition of CDFs Hutzinger et al (1973) observed that photolysis of 2,8-DCDF and OCDF in methanol and hexane solutions resulted in rapid dechlorination of the substrates with the eventual accumulation of unidentified resinous polymeric products.

7.2.2 Thermal Degradation. Stability of chlorinated dioxins to combustion was investigated by Stehl et al (1973). They observed that 2,3,7,8-TCDD was somewhat stable up to 700°C with 50% decomposition obtained at that temperature with a 21 s exposure. When the residence time was more than doubled, it resulted in only slightly more decomposition. However, decomposition of 2,3,7,8-TCDD was complete at 800°C.

7.2.3 Microbial. Matsumura and Benezet (1973) conducted studies on the microbial degradation of TCDD. They reported that:

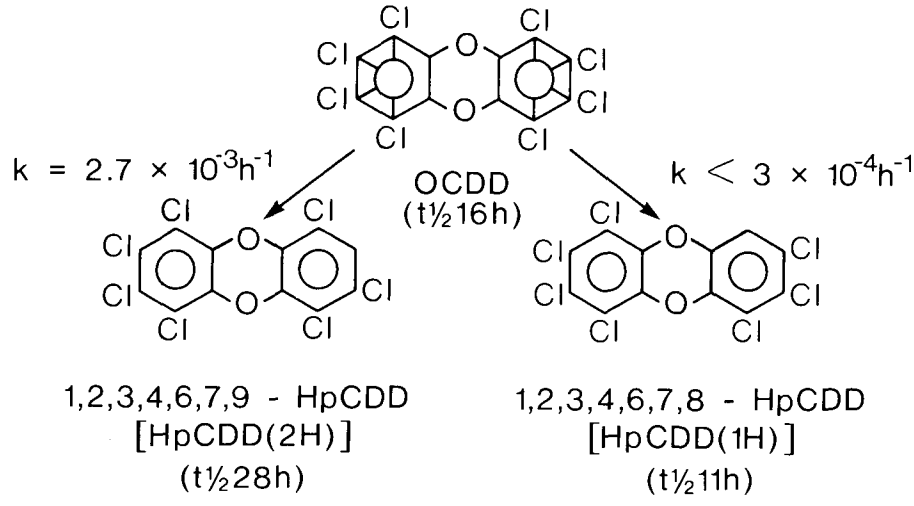


FIGURE A7-7 PHOTOLYSIS OF OCTACHLORODIBENZO-p-DIOXIN IN HEXANE EXPOSED TO SUNLIGHT (Dobbs and Grant, 1979)

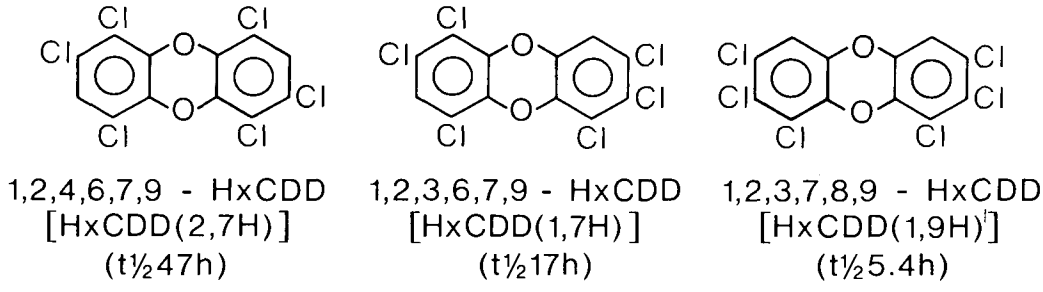


FIGURE A7-8 PHOTOLYTIC HALF LIVES FOR THREE HEXACHLORODIBENZO-p-DIOXINS IN HEXANE EXPOSED TO SUNLIGHT (Dobbs and Grant, 1979)

"As for the microbially mediated degradation of TCDD, our current survey indicates that such capabilities are rather rare in nature. Approximately 100 microbial strains in which the ability to degrade persistent pesticides has been previously demonstrated were screened for this purpose. Among them, only five strains showed some ability to degrade this compound."

Ward and Matsumura (1977) described the fate of ^{14}C-2,3,7,8-TCDD in lake water and sediments by studying its microbial degradation, evaporation and adsorption to sediment. They concluded that "TCDD is mostly bound with the lake sediment where it is stable and not very available for microbial degradation." Metabolism of TCDD did occur in the aqueous phase and was genuine, as judged by the freeze-drying technique employed by the authors, although the extent of the metabolism was very small and confined to anaerobic conditions. Ward and Matsumura (1977) also stated that "The metabolites are degraded faster than TCDD since metabolite production decreased with time indicating an apparent case of intermediary metabolism." Under the conditions imposed in the experiment the authors determined that the half-life of TCDD in the sediment was about 590 days. They also determined that: "Water-mediated evaporation of TCDD apparently occurred. Microbially catalyzed evaporation (gas evolution) of TCDD also provided a possible 'escape route' for TCDD."

7.3 Transport of Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans

7.3.1 In Soil. Transport of CDDs in soil is affected by the same mechanisms that affect movement of CPs, such as adsorption and leaching (App. 6 Sect. 6.2.1 and 6.2.3, respectively) and by the same edaphic factors such as organic matter content, soil pH, etc. Helling et al (1973), in discussing the mobility of CDDs in soils, made the general statement that mobility of individual organic compounds varied widely, though adsorption to a soil was perhaps the best index to leaching behaviour in that soil. In the review article, Helling et al (1973) noted the investigations of the senior author (Helling, 1970) on mobility of 2,7-DCDD and 2,3,7,8-TCDD as evaluated by the thin-layer chromatography technique. In all soils tested, which included Norfolk and Lakeland sandy loams, Hagerstown silty clay loam, Barnes clay loam, and Celeryville muck, both dioxins were immobile (i.e. they would not be leached into the soil by rainfall or irrigation). This did not preclude their lateral transport during surface erosion, as witnessed by Pierce and Victor (1978).

Kearny et al (1973) stated that the relative immobility of the chlorodioxins was expected, based on their very low solubility in water (0.6 μg/L). They concluded that the dioxins would not threaten ground water supplies and would be subject to the surface processes affecting pesticides.

In a laboratory procedure, Matsumura and Benezet (1973) investigated the movement of 2,3,7,8-TCDD from treated sea sand on top of a column of sandy loam subjected to leaching by water at the rate of 2 mL/h. Virtually no TCDD was found to leach out from the column. They concluded that since the mobility of TCDD in soil was relatively low, "...the mode of translocation of TCDD in the environment would be limited to movement of soil particles or dust-carried dispersion and biological transfer (but not plant-mediated transfer), particularly in aquatic environments." This conclusion was subscribed to by Isensee (1978) as follows:

> "Because TCDD is tenaciously adsorbed to soil, it is not easily removed in runoff and is not leached. Neither is it taken up by plants. As a result, the only way in which soil-adsorbed TCDD can reach an aquatic site is by erosion."

Following investigations on uptake of DCP, DCDD, and TCDD from treated Lakeland sandy loam, by oats, Avena sativa L., or soybeans, Glycine max L., Kearney et al (1973) and Helling et al (1973) concluded that plant uptake of TCDD from soils was highly unlikely.

Arsenault (1976) composted PCP-OCDD containing sludges from a wood preservation process with soil and measured the leachates. He concluded from results at the end of a 205 day test that OCDD would not migrate significantly in soil if soil contamination occurred, and that the low level of OCDD in the leachate was attributed to OCDD biodegradability.

7.3.2 In Sediments and Water. A unique 1.6 km^2 area at Elgin Air Force Base, Florida, was used to calibrate and test aerial application equipment for herbicides. The soil in this area, over a 9-yr period, received more than 72,570 kg each of 2,4,5-T and 2,4-D. During that time significant levels (10 - 710 ppt) of TCDD were detected in the top 15 cm of soil (Young et al, 1976). Erosion of soil occurred into a pond in the test area and into a stream immediately adjacent to the area. In silt of the aquatic system there were 10 - 35 ppt of TCDD, but only at the point where eroded soil entered the water. No additional data on the transport of PCDDs in sediment and/or water could be located.

7.3.3 In Air. Although there have been few investigations on the transport of PCDDs in the air, it has generally been assumed that any movement in the air would largely occur via air-borne particles (Ramel, 1978). However, recent investigations in the Netherlands (Olie et al, 1977) and in Switzerland (Buser et al, 1978) have identified PCDDs and PCDFs in fly ash and flue gas from municipal incinerators and in one case in Switzerland an industrial heating facility burning a large proportion of used industrial oils. Olie et al (1977) had suggested that extensive monitoring of flue gases and fly ash from incinerator plants would be advisable because of the known toxicity of the PCDD and PCDF compounds coupled with the usual location of plants near centers of populations which would be subjected to fall-out from the plumes.

APPENDIX 8

APPENDIX 8

8 BIOCONCENTRATION AND ENVIRONMENTAL MODELLING OF CHLOROPHENOLS, CHLORODIBENZO-p-DIOXINS, AND CHLORODIBENZOFURANS

Two major areas of interest regarding a biologically active chemical in the environment are, first, whether it can accumulate or concentrate in specific biota in the food chain and second, whether the bioaccumulation or bioconcentration factor can be predicted. This appendix contains a brief account of the literature published since 1972 on bioconcentration of the chlorophenols, their impurities, and some of their derivatives, in aquatic, terrestrial and intermedia situations. Model ecosystem studies, such as those referenced in this appendix, have provided useful, but limited, information on the transport and bioconcentration of chlorophenols and their impurities in the environment.

8.1 Bioconcentration of Chlorophenols

The two terms which are used to adequately describe the uptake and accumulation of chemicals in tissues of organisms are bioconcentration and bioconcentration factor. They will be used in the following sense as defined by Kenaga (1972), but are not limited to pesticides:

> "Bioconcentration is defined here as the amount of a pesticide residue accumulated by an organism by adsorption, and by absorption via oral or other route of entry, which results in an increased concentration of the pesticide by the organism or specific tissues. Bioconcentration is thus defined broadly because residues of compounds accumulate on the outside as well as on the inside of organisms whether they are plants or animals. Residues on the outside of the organisms are rarely differentiated from the total residues in and on organisms in publications reporting analytical results.
>
> "The bioconcentration factor is the ratio of the measured residue compared to the residue of the pesticide in the ambient air, water, or soil environment of the organism and/or the various species of food organisms consumed, as specified. Such a definition emphasizes the difficulty of putting a name to a phenomenon which has so many variable inputs, interpretations, and lack of definition in the literature."

Research on bioconcentration of the CPs and their PCDD and PCDF impurities is in its infancy and has been limited primarily to aquatic situations.

Aquatic

Preliminary results from an investigation by Fox and Hodson (1978) on the fate of PCP in a model ecosystem have indicated an accumulation of PCP in fish, snails and crustaceans of >100X the concentration of PCP in the water (whole animal, wet-weight basis).

Schimmel et al (1978) exposed several estuarine animals to NaPCP in flow-through toxicity tests. In addition to the basic toxicity data as reported in App. 4, Sect. 4.1.2 (Invertebrate) and App. 4, Sect. 4.1.3 (Fish), they developed bioconcentration data for the eastern oyster, Crassostrea virginica, as follows:

> "Eastern oysters exposed to Na-PCP concentrations of 25.0 and 2.5 µg/l accumulated the chemical in their tissues an average of 41 and 78 times, respectively. After Na-PCP delivery was discontinued, however, the oysters purged themselves of the pesticide within four days."

Holmberg et al, 1972, observed that eel, Anguilla anguilla L., exposed to 0.1 ppm PCP in sea water for 8 days contained 33.4 ppm of PCP in the liver, 9.4 ppm in the muscle, and 4.4 ppm in the blood. Following an 8 day recovery period in untreated sea water, levels of PCP were 11.9, 3.6, and 2.1 ppm in the liver, muscle, and blood, respectively. In a comparable freshwater test, eel exposed to 0.1 ppm PCP for 4 days had 8.8 ppm in the liver, 0.81 ppm in the muscle, and 1.7 ppm in the blood. A recovery period of 55 days elapsed before levels of PCP in the liver and muscle dropped to 1.3 and 0.08 ppm, respectively; and 38 days were required for the PCP blood level to recede to 0.31 ppm.

Kopperman et al (1976) had investigated the bioaccumulation potential of chlorinated compounds found in waste-treatment effluents. (For a brief discussion on aqueous chlorination see Sect. 4.2.3). As a result of both their research, which demonstrated the presence of the higher CPs (TTCPs and PCP) in fish exposed to chlorinated effluent, and of a review of related literature they stated that:

> "Reports appear to be conclusive in support of the argument that even the most gentle chlorination conditions will cause chlorine to be incorporated into organic molecules. The incorporation of chlorine into an organic molecule

increases its lipophilic character and at the same time normally causes an increase in the observed toxicity or bioaccumulation, or both.

"The persistence of these compounds is now becoming a concern. Not all organochlorine compounds bioaccumulate to high levels. The data suggests that polar compounds are more easily biodegraded, and the nonpolar (highly lipophilic) compounds accumulate."

Kopperman et al (1976) also noted that:

"Some investigators have been able to demonstrate positive correlation between the η-octanol/water partition coefficients for given compounds and their ability to bioaccumulate in various species of fish."

Although CPs were not included in their study, Neely et al (1974) had demonstrated that bioconcentration of several chemicals in trout muscle was found to follow a straight line relationship with a partition coefficient.

To fill an information gap, Chiou et al (1977), who had noted that partition coefficients of many compounds of environmental significance were not always available, developed an empirical equation to relate the experimental n-octanol/water partition coefficients to the aqueous solubilities of a wide variety of chemicals including organochlorine pesticides.

Landner et al (1977) in their review of the literature noted the work of Leach and Thakore (1975) in British Columbia who had identified five compounds in western Canada kraft pulpmill effluents that were toxic to juvenile rainbow trout, Salmo gairdneri. Two of these compounds were 3,4,5-tri-, and 3,4,5,6-tetra-chloroguaiacol. Landner et al (1977) also referred to the investigations of Lindstrom and Nordin (1976) as follows:

"The first characterization of the acid-phenolic fraction of European, pine-wood pulp bleachery effluents, with respect to low-molecular chlorinated compounds was carried out by LINDSTROM and NORDIN (1976). They identified trichlorophenol, isomeric trichlorocatechols and tetrachlorocatechol in the effluents from the first chlorination step and trichlorophenol, isomeric trichloroguaiacols and tetrachloroguaiacol in the effluents from the first alkaline extraction step. However, the neutral fraction of these effluents still remains to be characterized for low-molecular chlorinated compounds."

Following the identification of these compounds, Landner et al (1977) then demonstrated that 2,4,6-TCP and tri-, and tetra- chloroguaiacol bioconcentrated in liver of rainbow trout after exposure to low concentrations of these compounds in effluents from Swedish kraft pulp bleacheries. They observed that:

> "These preliminary data indicate that the three chlorinated phenols here studied are rapidly taken up from water to fish, resulting in a steady state concentration in the liver fat already after a couple of weeks. They also indicate that clearance from the fish tissues is relatively rapid after discontinuation of exposure and that at least tetrachloroguaiacol may be metabolized by the fish before excretion."

Landner et al (1977) pointed out that although they had identified a few CPs occurring in sulphate pulp mill bleachery effluent, and that these chemicals were bioconcentrated in fish, their research was not designed to indicate any possible detrimental effects in fish or other parts of the aquatic ecosystem. To fill this void research was underway to determine possible chronic effects on the fish of the compounds after concentration to high levels.

In a laboratory project, Pruitt et al (1977) studied the accumulation and elimination of PCP by the bluegill, Lepomis macrochirus. In an abstract of the report on the investigation, they stated:

> "Fish exposed to sublethal concentrations (0.1 mg/liter) accumulated PCP in various tissues from 10 to 350 times the ambient concentration. The liver had the greatest concentrations followed by the digestive tract, gills, and muscle. Upon removal from PCP-containing water the contaminated fish rapidly eliminated PCP. Residues ranging from 0.03 to 0.6 ppm were still detectable, however, 16 days after fish were placed into a clean environment."

Faas and Moore (1979) developed an improved method for measuring PCP in tissue samples from biota in the estuarine environment. Their application of the method to organisms exposed to various concentrations of PCP in a flow-through system indicated that PCP accumulated in mullet (Mugil cephalus), shrimp (Palaemonetes pugio), and oyster (Crassostrea virginica) (Table A8-1).

Ernst (1979) examined factors affecting bioconcentration of PCP in the common mussel, Mytilus edulis, and the polychaete, Lanice conchilega. He determined that species differences and lipid content of the animals seriously affected bioconcentra-

TABLE A8-1 MEASURED RESIDUES OF PCP IN FISH, SHRIMP, AND OYSTERS EXPOSED TO SEVERAL MEASURED CONCENTRATIONS OF PCP IN FLOWING SEA WATER FOR 96 h (Faas and Moore, 1979)

Species	Conc. in water (ppb)	Tissue residues (ppm)
FISH (Mugil cephalus)	46.0	0.29
	85.0	6.7
	157.0	8.8
SHRIMP (Palaemonetes pugio)	32.0	0.050
	54.0	0.10
	76.0	0.23
	249.0	0.43
OYSTER (Crassostrea virginica)	2.8	0.18
	26.0	0.86

tion factors when measured at a steady-state concentration in a static system. Factors which showed no remarkable effect on concentration of PCP in the test animals included variation in temperature of sea water, 5 - 15°C, and metabolic activity, as measured by conjugation of PCP in the mussels. Predicted and measured values (ng/g wet-weight) for concentration of PCP in Lanice were 120 and 160, respectively, and for PCP in Mytilus were 10 and 3 - 10, respectively; which demonstrated the closeness of fit between experimentally derived concentration values and field values in animals from a coastal area. Concentration factors for PCP, as measured by Ernst (1979) in Mytilus and Lanice were 390 and 3830, respectively. The animals were exposed to PCP at an initial concentration of 2 - 5 µg PCP/L of sea water at 33% salinity for Mytilus and 27% salinity for Lanice.

In one of the few available reports on residue levels of PCP in fish as a result of exposure to PCP in a freshwater ecosystem, Pierce et al (1977) observed that fish contained concentrations of PCP for a period of at least six months after initial exposure following the accidental release of untreated wastes from a pole-treatment plant (Sect. 5.1.1). PCP concentrations in small fish dropped to near background levels within 10

months (Table A8-2). It is of interest to note the comment of Pierce et al (1977) regarding the effect of sampling procedure on conclusions from the study:

> "Unfortunately, the collection of fish by seining along the shore resulted in capturing small individuals that feed near the first consumer level. Therefore, these fish reflect the low PCP concentration in the water column and do not provide information regarding biological magnification via the benthic food chain."

TABLE A8-2 PCP CONTENT IN FISH FROM A FRESH WATER ECOSYSTEM (Adapted from Pierce et al, 1977)

Sample date	PCP concentration ng PCP/g dry-weight sample (ppb)[a]
2-27-75	2500 ± 200
4-24-75	1380 ± 20
6-23-75	130 ± 70
10-11-75	Trace
12-6-75	651 ± 650
2-10-76	87 ± 22
5-3-76	Trace

[a]Average of replicate samples ± one-half the range

Following these initial investigations on the fate of PCP in an aquatic ecosystem, Pierce and Victor (1978) reported on results obtained during the second year of monitoring the residues of PCP and PCP-degradation products in fish (Table A8-3). The obviously higher concentrations of PCP and its degradation products observed in fish tissue in January 1977 were the result of an addition of PCP to the ecosystem in December 1976. The authors' comments on the PCP residues in fish were as follows:

> "Fish contained only background levels of PCP in October, 1976 (Table A8-3) but rapidly accumulated very high concentrations in January, 1977 immediately after the spill. Concentrations decreased somewhat by April, 1977 but were still well above background levels. Fish collected shortly after

TABLE A8-3 PCP AND PCP-DEGRADATION PRODUCTS IN FISH (Adapted from Pierce and Victor, 1978)

Date	Sample	Concentrations - ng/g wet weight (ppb)					
		Muscle			Liver		
		PCP	PCP-OCH_3	2,3,5,6-TTCP	PCP	PCP-OCH_3	2,3,5,6-TTCP
10-11-76							
	sunfish-1	5	4	< 1[a]	26	10	30
	sunfish-2	4	2	< 1[a]	150	12	50
1-6-77							
	sunfish-1	9,400	94	95	130,000	530	950
	sunfish-2	6,400	32	60	-[b]	-[b]	-[b]
	bass-1	7,000	-[b]	-[b]	200,000	-[b]	-[b]
	bass-2	17,000	250	300	140,000	500	1,600
	bass-3	16,000	90	130	325,000	700	8,200
	catfish-1	19,000	164	219	214,000	1,200	8,500
4-27-77							
	sunfish-1	900	30	27	14,600	190	250
	sunfish-2	1,000	28	22	14,900	115	150
	catfish-1	8,200	100	82	50,600	140	1,400
	catfish-2	1,500	177	41	20,200	575	940

[a] lower limit of detection

-[b] not analyzed

the spill in January, 1977 contained PCP in concentrations of 200,000 ppb in liver tissue, 40,000 ppb in gills, and 12,000 ppb in muscle. These values represent concentration factors over the PCP content in the water of 500 for muscle, 1,500 for gills, and 8,000 for liver. These data are similar to the results obtained by exposing fish to 0.1 ppm PCP in water under controlled laboratory conditions (Pruitt et al., 1977)."

Concentrations of TTCP detected in fish tissue (Table A8-3) through the study period indicated that fish rapidly accumulated TTCP from the water immediately after the spill and retained it in a manner similar to PCP. The authors noted that although only a few analyses for 2,3,4,5-TTCP were accomplished, sufficient evidence indicated a similar, yet somewhat lower concentration than the 2,3,5,6-TTCP isomers in the same samples. Pierce and Victor (1978) had the following comments on concentration of PCP-OCH_3 in fish:

"Fish muscle and liver tissue contained about the same concentration of PCP-OCH_3 as PCP in October, 1976 (Table A8-3). The concentration increased in January, 1977 immediately after the spill and remained relatively high through April. The high concentration of PCP-OCH_3 in fish obtained from water containing very low concentrations suggests an extremely high partitioning of PCP-OCH_3 from water to fish. It is also possible that the PCP-OCH_3 in fish was obtained from food or from PCP converted to PCP-OCH_3 by flora in the fish gut."

In a summary statement on the impact of PCP on a freshwater ecosystem, Pierce (1978) stated:

"In general, the results indicate that PCP released into the aquatic ecosystem was not rapidly assimilated by photo- or microbial-degradation. Acute toxicity to fish occurred by rapid uptake of the water soluble phenate anion, whereas chronic exposure occurred by leaching of PCP from the contaminated water shed into the lake and by incorporation of PCP into the benthic food chain. Thus, once contaminated with PCP, the sediment and water shed area provided a source for chronic PCP pollution of the aquatic ecosystem."

Terrestrial

There was little information available on movement of PCP in plants. Isensee and Jones (1971) studied the uptake of ^{14}C-labelled 2,4-DCP by seedling oats and

soybeans from nutrient solution and from treated soils, as part of an investigation on the uptake of 2,3,7,8-TCDD by agronomic crops. In spite of experimental errors, it was demonstrated that the concentration of 2,4-DCP from nutrient solution maximized in the seedlings within 24 h and then remained constant or declined. Concentrations of 2,4-DCP were much lower in the tops than in the roots, indicating little translocation. In the studies on uptake of 2,4-DCP from treated soils, there was no evidence of a concentration of 2,4-DCP in the seeds of oats and soybeans.

Kaufman (1976) in reviewing the translocation of PCP in plants noted the results of Miller and Aboul-Ela (1969) who had observed that in cotton sprayed with PCP there could be some translocation of PCP or possible metabolites within the plants and that PCP residues definitely existed in seed from bolls which were closed at the time of treatment.

Intermedia (Aquatic - Terrestrial)

Lu et al (1978) investigated the environmental fate of ^{14}C-PCP in three model ecosystems: aquatic, terrestrial-aquatic, and terrestrial. They found that:

> "The principal degradative products appeared to be tetrachlorohydroquinone, pentachlorophenyl acetate, and conjugales. In the terrestrial-aquatic model ecosystem, the bioaccumulation factors for PCP were: alga 5, daphnia 205, snail 21, mosquito 26, and fish 132. The parent PCP constituted 11.1% of the total ^{14}C in alga, 12.2% in snail, 33.3% in mosquito, 55.5% in daphnia, and 51.2% in fish. Tetrachlorohydroquinone constituted 5.4% of the total extractable ^{14}C in alga and 10.5% in snail. None was detected in other organisms.
>
> "In the terrestrial model ecosystem, the vole at the top of the food chain contained 0.5% of the total dosage applied to the corn-soil interface, and 6.4% of this was intact PCP."

8.2 Bioconcentration of Chlorodibenzo-p-dioxins and Chlorodibenzofurans

Matsumura and Benezet (1973) devised a model ecosystem to study bioconcentration of TCDD in three organisms, brine shrimp, mosquito larvae (Aedes aegypti), and northern brook silverside (Laludesthes sicculus sicculus). The model ecosystem which best reflected a natural mechanism, resulted from deposition of TCDD on sand, which was then added to the test aquarium. Only that portion of TCDD which was soluble was present in the water at any time. In this experiment the mosquito larvae, which were bottom feeders, were the best concentrators of TCDD. The concentration factor for dioxin in

mosquito larvae was 9,222; while brine shrimp had a concentration factor for TCDD of 1,570. The concentration factor for dioxin in fish was not calculated except for a 2-step bioconcentration when mosquito larvae were available for fish food, which resulted in a concentration factor in fish of 54. Three limiting factors in bioaccumulation of TCDD were a) its low solubility in water and lipids, b) its low partition coefficient in lipids, and c) species-specific factors.

In the second of the two known studies in which bioaccumulation of TCDD has been measured, Isensee (1978) summarized the work of Isensee and Jones (1975), as follows:

> "Several aquatic model ecosystems were used to estimate the bioaccumulation potential of 2,3,7,8-tetrachlorodibenzo-para-dioxin (TCDD). Five species of aquatic organisms (algae, snails, daphnids, mosquito fish and catfish) were exposed to TCDD in water at concentrations of 0.05-1330 ppt (parts per trillion = 10^{-12}) for up to 32 days. The various organisms accumulated TCDD to an average of 2-7000 times the concentration in the water. Total amounts accumulated were directly related to water concentrations, and equilibrium concentrations were reached in tissues in 7-15 days. TCDD bioaccumulation ratios were about the same as those reported for many of the chlorinated hydrocarbon insecticides."

Norstrum et al (1976) studied the elimination of CDFs associated with PCBs fed to mallard ducks (Anas platyrhynchos) for nearly a year. The analytical method developed for the study allowed CDFs to be extracted from lipid, and separated from interfering PCBs, with greater than 90% recovery of tetra- to hexa-CDFs. It was concluded from the results of the feeding study that less than 3% of the dosed CDFs were accumulated by the mallards and that it was unlikely that CDFs were sufficiently persistent in avian species to enable their detection in environmental samples. It was noted that starting with 40g of lipid, the detection limit for individual CDFs in lipid was of the order of 0.01 µg/kg.

8.3 Modelling

Modelling studies to elucidate the fate of CPs and their impurities in the environment have been reported by just a few researchers, including Lu et al (1978), Lu and Metcalf (1975), Isensee and Jones (1975), Matsumura and Benezet (1973), and Fox and

Hodson (1978) (App. 8, Sect. 8.1 and 8.2). The studies had been limited to microcosm studies simulating aquatic, terrestrial-aquatic, and terrestrial ecosystems.

The "model ecosystems" had been devised by Metcalf et al (1971) to study the biodegradability of pesticides but they considered them adaptable to industrial chemicals as well. As noted by Metcalf et al (1975) the methodology developed yielded useful information about 1) degradation pathways of radiolabelled material, 2) the toxic effects of the compounds and their degradation products, 3) their comparative biomagnification and food chain concentration, and 4) their comparative biodegradability; all in organisms of five phyla linked in several food chains.

Although "model ecosystem" studies have produced tangible results, such as the bioaccumulation factors for PCP in algae, daphnia, snails, mosquitoes, and fish, as reported by Lu et al (1978) (App. 8, Sect. 8.1), they have only provided part of the answers. The reasons for this were expressed by Witherspoon et al (1976), in their review on modelling and environmental transport of toxic materials, as follows:

> "Review of laboratory microcosm studies revealed that microcosms have served as useful tools in the study of movement of nutrients, toxic substances, and energy. Although experimental microcosms have employed a wide variety of species of organisms and levels of complexity, all of them omit large-scale processes such as alluvial deposition or long distance migration. Microcosms of reduced complexity are suitable for measuring one or a few rate processes over a short time range. The more complex microcosms, while being more like natural systems, present difficulties in measurement of some rate processes due to the importance of mutualistic and competing processes.
>
> "Three critical problems arise in using microcosm results for predicting actual environmental results. There is the question of applying results from an isolated process to a more complex system. There is not yet enough field verification of microcosm results to assume that this interpolation is valid. Secondly, the comparability of similar, but not identical, model systems has not been evaluated. And, perhaps most important, there is a general lack of estimates of confidence limits on parameter measurements made in microcosm studies. It is unusual to find replicated experiments in the literature except for the simplest microcosms.
>
> "No standardized microcosm was found to exist, although the seven species system devised by Metcalf has been used to test a number of toxicants. This

model terrestrial-aquatic ecosystem, however, still excludes some functionally important groups such as soil and benthic organisms.

"It is proposed that, until research on microcosms progresses and until there is more field validation of microcosm results, simple food chain tests should be used to determine whether toxicants are likely to bioaccumulate or biomagnify. The food chain tests which were selected (one for terrestrial transfers, two for aquatic freshwater transfers, and one for marine transfers) offer a maximum chance for a chemical to bioaccumulate. Moreover, enough trophic levels are included to give a reasonable prediction of the extent of biomagnification."

Gebefugi et al (1979) used modelling experiments to demonstrate and quantify the amounts of PCP that could be transported from treated wood surfaces to untreated surfaces in an enclosed area. The project was undertaken following an incident in West Germany in which residents of an apartment showed continuing symptoms of PCP poisoning following treatment of wood surfaces in a living room 6 years previously with a protectant compound containing 6% PCP. Gebefugi et al (1979) concluded that the initial concentration of PCP in the top layer of the freshly painted wood surface was 4000 - 6000 mg PCP/kg of treated wood, with reduction to 50% in 6 yr. After 4 yr of exposure to contaminated air (the probable concentration of PCP in the air was calculated at 1 - 160 µg PCP/m^3) the surface of untreated wood had levels of 5 - 25 mg PCP/kg of wood. Comparable concentrations of PCP and TTCP were noted in wallpaper, plaster, carpet, upholstery, curtains, books, etc. As a result of these findings Gebefugi et al (1979) noted that the German press reported that use of PCP in inhabited rooms should be limited or completely avoided. Although Gebefugi et al (1979) were not able to fully explain the mechanisms, they did confirm the transport of the CPs and chlorinated impurities through the air to untreated materials and that levels of CPs in the air could fluctuate widely.

APPENDIX 9

APPENDIX 9

9 WASTE MANAGEMENT OF CHLOROPHENOLS

This appendix contains information on the sources and management of CP contaminated wastes from both CP production processes and industries utilizing CPs. In addition, ways and means for rehabilitation of CP contaminated ground water at a wood preserving plant are presented, and a case history is cited.

Chlorophenol Wastes from Production Processes

During production of CPs and subsequent derivatives, specific amounts of liquid wastes, consisting of reaction waters and washing waters, will be generated for any given amount of product. Actual volumes will depend, in part, on process chemistry, in-plant house-keeping, and on amounts of contaminated residual solutions (effected by process equipment design). In addition to wastes from these sources there are also those wastes from spills, etc., carried in storm-water run-off from plant sites. Wastes from all of these sources have to be managed so that contaminants present in the waste will have a minimum or negligible effect on the environment.

In Canada, liquid effluent from the CP derivative production facility at Clover Bar, Alberta, is disposed of by either pumping raw effluent into deep-wells, or by treating the effluent in lagoon systems prior to its discharge into the North Saskatchewan River. The effluent from the lagoons is monitored for BOD, COD, and other water quality characteristics, and occasionally for CPs.

Chlorophenol Wastes from Wood Preservation

In addition to CP contaminated wastes which originate from production processes, a significant volume of wastes arise during treatment of wood with preservatives containing TTCP or PCP. For a description of the treatment processes and the waste volumes which result, refer to Sect. 4.1, and to Richardson (1978).

Four documents completed since 1976, which are particularly relevant to the management of CP-containing wastes from Canadian wood preservation plants, include the following:

1) "The control of preservative wastes from wood treatment", (Shields, 1976). The information for this review was acquired by the Ontario Research Foundation under

contract to the Eastern Forest Products Laboratory and included material from the world literature and from interviews with Canadian wood preservers.

2) "Literature review of wastewater characteristics and abatement technology in the wood and timber processing industry", (Thurlow and Assoc., 1977). This literature review, which was done under contract for the Water Pollution Control Directorate of the Environmental Protection Service, drew heavily on two Development Documents of the United States Environmental Protection Agency, from 1973 and 1974.

3) "The Proceedings of the Technology Transfer Seminar on the Timber Processing Industry, March 10 - 11, 1977, Toronto, Ontario", were published in June 1978. This collection of eight papers from both industry and government, represent a Canadian state-of-the-art review on the wood preservation industry and its impact on the environment.

4) "Hydrogeological control and clean-up of soil and groundwater contaminants at Northern Wood Preservers, Limited." (Thompson et al, 1978). Paper presented at the 25th Ontario Industrial Waste Conference, June 18-21, 1978, Toronto, Ontario. A report on a waste management case study conducted by W.L. Wardrop and Associates Ltd., Winnipeg, Man., for Abitibi-Price Northern Wood Preservers, Limited.

The 24 commercial wood preservation plants that were in operation in 1979, and which utilized PCP in an oil-borne formulation, are listed in Table A9-1. The number and size of the treatment cylinders and tanks are also included. Not included are 11 pressure treatment plants, which use water-borne salts exclusively (Table 10), and several non-pressure plants, where wastewater volumes were considered negligible (Shields, 1976).

Table A9-2 to A9-5 inclusive (Shields 1976) contain additional information on the waste treatment and disposal practices at each of the 24 pressure treating plants, grouped according to method of stock conditioning: air drying (Table A9-2), Boultonizing (Table A9-3), open steaming or a combination of open steaming plus Boultonizing (Table A9-4), and closed steaming or a combination of closed steaming and Boultonizing (Table A9-5). One apparent characteristic, common to all the Tables, is the amount of information not available, including data in the categories of volume of effluent, sludge disposal methods, and effluent analysis.

TABLE A9-1 LIST OF CANADIAN WOOD PRESERVING PLANTS[1] (1979)
(data provided by: Eastern Forest Products Laboratory, Environment Canada, Ottawa)

Managing Company	Headquarters	Plant Location	Wood Preservative[2]	Cylinders		
				Number	Diameter (Inches)	Length (Feet)
Commercial Pressure Plants						
Bay Wood Processing Ltd.	Thunder Bay, Ont.	Thunder Bay, Ont.	OWF	1	72	74
				1	72	60
Cote Wood Industries	Kamsack, Sask.	Cote Indian Reserve, Sask.	O	1	60	21
Domtar Chemicals Ltd. Wood Preserving Div.	Montreal, Que.	New Westminster, B.C.	COW	1	84	135
				1	72	125
				2	84	166
		Prince George, B.C.	O	1	84	98
				1	84	104
		Cochrane, Alta.	CO	1	84	164
				1	84	132
		Edmonton, Alta.	COW	2	84	150
				1	84	75
		Trenton, Ont.	COMC	2	84	134
		Delson, Que.	COW	2	84	150
				1	84	75
				1	84	50
		Newcastle, N.B.	COW	2	84	150
		Truro, N.S.	COW	1	84	150
				1	84	120
				1	72	55
Goodfellow Lumber Ltd. Wood Treating Division	St. Andrews East, Que.	St. Andrews East, Que.	O	1	48	20
			WF	1	72	42
Kirkland Wood Treatment Ltd.	Kirkland Lake, Ont.	Dobie, Ont.	COW	1	72	50
Koppers International (Canada) Ltd.	Richmond, B.C.	Burnaby, B.C.	COWF	1	90	108
				1	90	165
				2	78	46

[1] List includes only those plants where pentachlorophenol has been used in an oil-borne formulation

[2] Types of preservatives used: C - creosote, O - oil-borne, W - water-borne, F - fire retardant, MC - methylene chloride

[3] Portable plant

[4] Vacuum plant

TABLE A9-1 (Cont'd) LIST OF CANADIAN WOOD PRESERVING PLANTS[1] (1979)
(data provided by: Eastern Forest Products Laboratory, Environment Canada, Ottawa)

Managing Company	Headquarters	Plant Location	Wood Preservative[2]	Cylinders Number	Cylinders Diameter (Inches)	Cylinders Length (Feet)
Koppers International (Canada) Ltd.	Richmond, B.C.	Camrose, Alta.	OW	1	84	72
				1	84	84
L & M Wood Products Ltd.	Glaslyn, Sask.		O	1	84	45
Lehner Wood Preservers	Prince Albert, Sask.	Prince Albert, Sask.	O	1	72	38
Natal Forest Products Ltd.	Coleman, Alta.	Coleman, Alta.	O	1	72	50
Newfoundland Hardwoods Ltd.	Clarenville, Nfld.	Clarenville, Nfld.	CO	1	84	80
North American Wood Preserving Ltd.	Rosedale, B.C.	Rosedale, B.C.[3]	OWF	2	36	60
Northern Wood Preservers Ltd.	Thunder Bay, Ont.	Thunder Bay, Ont.	COWF	3	84	132
Peerless Wood Preservers Ltd.	Cayley, Alta.	Cayley, Alta.	O	1	72	52
Rocky Wood Preservers Ltd.	Rocky Mtn. House, Alta.	Rocky Mtn. House Alta.	OW	1	60	72
Wood Preservation Industries Ltd.	Montreal, Que.	Tracy, Que.	COWF	1	72	72
				2	72	50
Private Pressure Plants						
Eastern Forest Products Laboratory Forintec.	Ottawa, Ont.	Ottawa, Ont.	COWF	1	60	10

				Tank Size (Feet) Number	Length	Width	Depth
Commercial Non-pressure Plants							
Canada Cedar Pole Preservers Ltd.	Galloway, B.C.	Galloway, B.C.	CO	3	10	(Diam.)	12
				1	26	12	9
				1	82	12	9
				1	47	10	10
				1	52	12	12
Falconbridge Nickel Mines	Falconbridge, Ont.	Falconbridge, Ont.	OW[4]	1	28	5	4

[1] List includes only those plants where pentachlorophenol has been used in an oil-borne formulation

[2] Types of preservatives used: C - creosote, O - oil-borne, W - water-borne, F - fire retardant, MC - methylene chloride

[3] Portable plant

[4] Vacuum plant

TABLE A9-2 WASTE TREATMENT AND DISPOSAL BY PRESSURE TREATING PLANTS IN CANADA EMPLOYING AIR DRYING AS METHOD OF CONDITIONING STOCK (Shields, 1976)

Plant Number	Annual Production (1974)	Type of Plant	Volume of Effluent (gal/day)	Primary Treatment		Secondary Treatment	Sludge Disposal Method	Data on Effluent Analysis
				Bulk Oil Separation	Flocculation Settling Filtration			
1	1 million posts	single retort (P)*	50 - 100 ($0.23-0.46m^3$)	Gravity Separation	None	Containment and Evaporation in open Pit	Not known	Not available
2	200,000 posts	single retort (P) surface condenser	300 - 400 ($1.36-1.82m^3$)	Gravity Separation Tank	None	Containment and Evaporation in open Pit	Not known	Not available
3	250,000 posts	single retort (P) surface condenser	<30 ($<0.14m^3$)	Gravity Separation in Tank	None	Containment and Evaporation in open Pit	Sludge trucked by commercial disposal company	Not available
4	750,000 posts	single retort (P)	None	Not known	Not known	Not known	Sludge Burail	Not available
5	50,000 cu. ft. lumber	single retort (P)	Not available	Gravity Separation in Sump	None	Containment and Evaporation in open Pit	Not known	Not available
6	Not available	single retort (P)	Not available	Gravity Separation in Tank	None	Containment in Septic Tank	Trucked by commercial disposal company	Not available

*(P) - Pentachlorophenol-petroleum

TABLE A9-3 WASTE TREATMENT AND DISPOSAL BY PRESSURE TREATING PLANTS IN CANADA CONDITIONING BY BOULTONIZING (Shields, 1976)

Plant Number	Annual Production (1974)	Type of plant	Volume of Effluent (gal/day)	Primary Treatment: Bulk Oil Separation	Primary Treatment: Flocculation Settling Filtration	Secondary Treatment	Sludge Disposal Method	Data on Effluent Analysis
1	660,000 ties 300,000 poles 130,000 posts	single retort (P)*	Not available	Gravity separation in tanks	None	Mixed with bunker fuel oil and incinerated	sludge burial	Not available
2	2-1/2 million cu. ft. lumber	Two retorts (P)(C)*	Not available	Gravity separation in tanks	None	Evaporation in tank by steam coils followed by incineration mixed with bunker fuel oil	sludge trucked to town garbage	Not available
3	2-1/2 million cu. ft. lumber	Four retorts (1 for (W) only) (P) (C)(W)* Barometric condenser	2500-3000 (11.4-13.6m^3) before bulk oil separation	Gravity separation in tanks (separate tank for each preservative)	None	Incineration mixed with bunker fuel oil (96% efficiency)	sludge disposed of by commercial disposal company	Not available
4	Not available	As above except 2 retorts for (W) & surface condenser	4000 (18.2 m^3) after bulk oil separation	Gravity separation in tanks	None	Spray evaporation on shavings and then incineration. No scrubbing of stock gas. Plan to use cylinder condensate for scrubbing	sludge disposed of by commercial disposal company	Not available

*(P) - Pentachlorophenol-petroleum, (C) - Creosote (W) - Water-borne preservative

TABLE A9-4 WASTE TREATMENT AND DISPOSAL BY TREATING PLANTS IN CANADA CONDITIONING BY OPEN STEAMING ALONE OR IN CONJUNCTION WITH BOULTONIZING (Shields, 1976)

Plant Number	Annual Production (1974)	Type of Plant	Volume Effluent (gal/day)	Primary Treatment		Secondary Treatment	Sludge Disposal Method	Data on Effluent Analysis
				Bulk Oil Separation	Flocculation Settling Filtration			
1	3 million cu. ft. lumber	Two retorts (one for (W) only) (P) (W)* open steaming barometric condenser	200 (0.9 m^3)	Gravity Separation in tank	Sand filtration	Filtrate for make up of water-borne preservative solution	Sludge trucked by commercial disposal company	Not available
2	5 million board ft.	(one retort (P) (C) open steaming surface condenser	Not available	Gravity Separation in tank	None	Containment and Evaporation in a pit	Not available	Not available
3	2-1/2 million cu. ft. lumber	Three retorts (one for (W) only) (P)(C) (W) Boultonizing and open steaming	Not available	Gravity Separation in tanks & API separation	Hay filtration	Lagooning in ditch and discharge. Plan-flocculation, polyurethane filtration and activated carbon adsorption	Not available	Not available
4	Not available	As above	4000-7000 (18.2-31.8m^3)	Gravity Separation in tanks & API separation	None	Same as above	Not available	Based on waste water vol. of 7000 gpd, API separator effluent carries 370 ppm total phenol and 1900 ppm oil to ditch
5	400,000-500,000 posts	Single retort (P) Boultonizing & open steaming barometric condenser	Not available	Gravity separation in tanks	None	Containment and Evaporation in gravel pit	Sludge mixed with shavings and sold	Not available

*(P) - Pentachlorophenol-Petroleum, (C)-Creosote, (W) - Water-borne preservative
**(PMC) - Pentachlorophenol in Methylene Chloride

TABLE A9-4 (Cont'd) WASTE TREATMENT AND DISPOSAL BY TREATING PLANTS IN CANADA CONDITIONING BY OPEN STEAMING ALONE OR IN CONJUNCTION WITH BOULTONIZING (Shields, 1976)

Plant Number	Annual Production (1974)	Type of Plant	Volume Effluent (gal/day)	Primary Treatment: Bulk Oil Separation	Primary Treatment: Flocculation Settling Filtration	Secondary Treatment	Sludge Disposal Method	Data on Effluent Analysis
6	700,000 cu. ft. lumber	Two retorts (P) (C) boultonizing & open steaming	Not available	Gravity separation in tanks	None	Containment and Evaporation in a pond	Sludge mixed with shavings and burned	Not available
7	14 million board ft.	Two retorts (P) boultonizing and open steaming	Not available	Gravity separation in tanks	None	Containment and evaporation in a gravel field	Not available	Not available
8	Not available	Two retorts (C) (PMC)** boultonizing and open steaming	Not available	Segragation of two effluents: a) creosote/ creosote-oil. Gravity Separation in heated tank b) Penta-methylene chloride gravity separation in heated tanks	None	After gravity separation. a) incinerated or trucked by commercial disposal company b) trucked by commercial disposal company	Sludge trucked by commercial disposal company	Not available

*(P) - Pentachlorophenol-Petroleum, (C)-Creosote, (W) - Water-borne preservative
**(PMC) - Pentachlorophenol in Methylene Chloride

TABLE A9-5 WASTE TREATMENT AND PRESSURE-TREATING PLANTS IN CANADA CONDITIONING BY CLOSED STEAMING ALONE OR IN CONJUNCTION WITH BOULTONIZING

Plant Number	Annual Production (1974)	Type of Plant	Volume of Effluent (gal/day)	Primary Treatment: Bulk Oil Separation	Primary Treatment: Flocculation Settling Filtration	Secondary Treatment	Sludge disposal Method	Data on Effluent Analysis
1	Not available	Single retort (P)(C)* closed steaming surface condenser	Not available	Gravity separation in tank	None	Containment and evaporation in heated tank	Sludge trucked for land filling	Not available
2	1.2 million posts	Single retort (P) closed steaming surface condenser	Not available	Gravity separation in tank	None	Containment and evaporation in pit	Not available	Not available
3	640,000 cu. ft. lumber	Single retort (P) closed steaming	15-20 ($0.07-0.09 m^3$)	Gravity separation in tank	Hay filtration	Containment and evaporation in a pond containing wood shavings	Sludge burned	Not available
4	9 million board ft.	Three retorts (P)(C)(W) (1 retort for (W) only) (C) boultonizing (P) closed steaming	Not available	Gravity separation in tank	None	Boultonizing condensate by soil irrigation, steaming cond. by containment and evaporation in summer and by burial in winter	Not available	Not available
5	Not available	Three retorts (P)(C)(W) (1 retort for (W) only) Boultonizing and closed steaming surface condenser	5000 ($23 m^3$)	Gravity Separation in tank	Not available	Activated sludge biological treatment and then discharge	Sludge trucked for land filling	Untreated eff. Total phenol 1600 ppm Treated eff. Total phenol 0.11-0.4 ppm
6	Not available	Two retorts (P) (C) boultonizing closed steaming	2000-2500 Max. 4000 ($9-12 m^3$) max. $18 m^3$)	Gravity Separation in tanks and API separator	Sand/polyurethane foam filtration	Activated carbon adsorption treatment and then dishcarge	Not available	From pilot plant study: anal. of untreated eff. before prefiltration: 500 ppm COD, 50-1100 ppm oil, 40 ppm "phenolic" equiv. and 3-60 ppm penta After carb. adsorp. "anal. limit" residuals of oils, phenols PCP.

*P - pentachlorophenol-petroleum
C - creosote
W - water-borne preservative

Although there has been some upgrading of waste treatment facilities since 1974 (not shown in the Tables) the comments of Shields (1976) are probably still valid; that is, of 34 plants surveyed (Table A9-6), 30 claimed

> ". . . to have no discharge and either do not generate any waste water or produce small volumes of waste water that are easily disposed of by incineration or containment and evaporation. The adequacy of such waste disposal systems is unknown since pertinent data on water pollution from seepage, leaching, run-off, and on air pollution associated with incineration are unavailable."

No data were available on volumes or disposal of NaPCP contaminated wastes which originate from NaPCP dip-tank treatment of green lumber destined for export. No information was available on the total number of these dip-tank treatment facilities in Canada or the volume of lumber so-treated with NaPCP. In British Columbia alone, there are at least 20 sawmills and three lumber export terminals which use this method of treatment (Ito, personal communication, 1978).

Allard (1978) outlined the role of the Environmental Protection Service, Environment Canada, in establishing effluent controls for the wood preservation industry, and noted that an Environmental Protection Service Discussion Paper was in preparation.

Two innovations for treatment of wood preserving effluents are a) use of activated carbon in physicochemical treatment systems (Richardson, 1978), as utilized in a plant in Nova Scotia since 1973 (Shields, 1976); and b) use of an activated sludge system. This latter system has been under investigation by Guo (1978) and Guo et al (1979) in cooperation with Northern Wood Preservers Ltd., Thunder Bay, Ontario, which is the only Canadian plant using this system. The plant, presently operating at near capacity, reduces the levels of phenols from 800 mg/L to approximately 1 mg/L (Thompson et al, 1978). Guo et al (1979) determined that the range of PCP loading in the Northern Wood Preservers Ltd. plant's wastewater prior to treatment, and during the period November 1977 to May 1978, varied between 0.30 to 14.9 mg PCP/L. The authors stated: "The wastewater discharged to the treatment plant is the condensate generated during the steam conditioning. Because wood conditioning and treatment are carried out in the same retort, the condensate is highly contaminated." The volume of wastewater averaged 13 m^3/day. Following an extended aeration-activated sludge treatment PCP levels during two monitoring periods were reduced to 5.5 and 3.6 mg PCP/L. If this process were followed by granular activated carbon adsorption, the PCP level could be

TABLE A9-6 DISPOSAL OF WASTEWATER BY PRESSURE TREATING PLANTS IN CANADA - 1978 (Adapted from Shields, (1976) and updated).

Disposal method		Number of plants
1.	INCINERATION - No discharge	
	Separation of bulk oil in gravity separation tanks, evaporation; no evaporation, incineration mixed with bunker fuel oil.	3
2.	CONTAINMENT AND EVAPORATION - No discharge	
	(a) Separation of bulk oil in gravity separation tanks, spray evaporation; evaporation by heating and stored in tank; open pit, and sludge trucked by commercial disposal company	16
	(b) Same as in (a), but wood shavings, etc., dumped in open pit and sludge incinerated; sludge disposed of by burial, land fill or cattle bedding; sludge disposal by commercial disposal company.	
3.	LAGOONING - Discharge	
	Separation of bulk oil in gravity separation tanks, flocculation with coagulant and settling; API separation and hay filtration; API separation and air flotation; lagooning in ditch and discharge	2
4.	SECONDARY BIOLOGICAL TREATMENT - Discharge	
	Separation of bulk oil in gravity separation tanks, and activated sludge biological treatment.	1
5.	ACTIVATED CARBON PHYSICOCHEMICAL TREATMENT - Discharge	
	Separation of bulk oil in gravity separation tanks, filtration through sand; polyurethane, and adsorption on activated carbon.	1
6.	NO WASTEWATER	
	From plants exclusively on waterborne preservative.	10
7.	OTHER - No discharge	
	Separation of bulk oil in gravity separation tanks, filtration through sand and effluent reuse for makeup water-borne preservative solution	1
		34

reduced to 0.03 mg PCP/L. When an activated carbon system was used to treat the wastewater directly, then the average PCP level in the effluent was reduced to 0.02 mg PCP/L of effluent. (Static bioassay tests conducted on rainbow trout indicated a nominal 96-h LC_{50} of 0.13 mg PCP/L (Guo et al, 1979) (Table A4-3).

A summary of available technology for physical-chemical treatment of wood preserving wastewaters from both oil-borne wood preserving processes and water-borne processes has been published by Averill et al (1978).

The use of bioassays as an integral part of wood preserving plant effluent treatment systems has been described by Westlake (1978). He also suggested that bioassay should play an important part in the modification of processes for effluent control.

Shields (1976) has enumerated six in-plant process changes which can help reduce wastewater volume and its accompanying pollution load to less than 2,000 gal. (9 m^3)/day for a 4-cylinder plant. The suggested practices are as follows:

1. Segregation of various effluents -- (a) separation of clean water (e.g. single-pass indirect cooling water, indirect steam condensate, and storm water) from process wastewater; (b) separation of creosote wastewater from pentachlorophenol wastewater to minimize emulsion problems; and (c) segregation of process wastewaters containing water-borne preservatives.
2. Stock conditioning by closed steaming instead of conventional open steaming: (a) use of closed steam coils for conditioning, with a separate holding tank for the contaminated water from the treatment, and reuse of the wastewater to generate steam in the retort; (b) use of separate water recycle systems for creosote and pentachlorophenol-oil treatments to minimize emulsion problems. Advantages of closed steaming over open steaming are: (a) reduction of wastewater volume, (b) reduction of COD in the retort effluent, (c) increase in oil recovery by alleviating the emulsion problem, and (d) reduction of the cost of wastewater treatment.
3. Maximum reuse of cooling water in plants equipped with barometric condensers, or replacement of barometric condensers with surface condensers.
4. Reduction in steam conditioning by using kiln dried wood.
5. Recovery of free oils by either American Petroleum Institute gravity separation procedures or by air flotation and flocculation using polyelectrolytes such as either lime or alum.

6. Plant sanitation by elimination or reduction of the incidence of water contamination with preservatives resulting from run-off by storm water, equipment (pumps, etc.) leaks, preservative loss from retorts, and spillage of preservatives.

Rehabilitation: Groundwater Contamination

Ground water contamination problems that arose at a wood treatment plant and the solutions to the problems were detailed by Thompson et al (1978) when they reported on a study commissioned by Abitibi-Price Northern Wood Preservers Ltd., Thunder Bay, Ontario, as the first step in a site rehabilitation process. In the introduction to the report Thompson et al (1978) stated:

> "Although the particular problem is interesting in itself, the principles used in its solution are much more significant. At the plant, wood products are treated with creosote, chromated copper arsenate and pentachlorophenol in an autoclave under heat and pressure. As part of the day to day operation, small amounts of preservative, mainly creosote, reach the ground adjacent to the plant. Some of these spilled materials have been washed directly into the Lake by surface runoff. But the major portion has infiltrated into the landfill underlying the site and then moved laterally into the Lake. The resulting high phenol content in the water and the accumulation of creosote on the bottom have seriously degraded the lake environment adjacent to the plant."

As an immediate remedy, the contaminated wastes which had already seeped into the lake were dredged and removed from the lake. To prevent continued migration of creosote and contaminated ground water into Lake Superior, it was recommended that a series of pump well points be established between the spill site and the lake. Thompson et al (1978) outlined the principles upon which the recommendation was based:

> "A brief outline of some basic principles of groundwater flow will serve as a background. Groundwater is part of a continually moving cycle wherein rainwater percolates downward to the water table, moves laterally and reappears in a lake or stream (Figure A9-1). This is a highly simplified version of the groundwater portion of the hydrologic cycle. Groundwater movement in the near surface materials is controlled by the configuration of the water table wherein groundwater flows in the direction of the water table slope. In deeper zones, the direction of groundwater flow is determined by the hydraulic head in that the groundwater moves from points of high hydraulic head to

points of low hydraulic head. Here we are interested primarily in the near surface zone and will restrict ourselves to the water table situation.

"Figure A9-2 depicts the movement of a contaminant from a spill site to a lake by means of the groundwater flow system. In order to control the movement of shallow groundwater, and thus control the movement of a contaminant, one need only manipulate the water table slope. This can be accomplished by pumping water out of the ground or pumping water into the ground. For example, in the case of Figure A9-3, a pumping well is used to intercept the contaminant and actually remove it. Figure A9-4 depicts a well into which water is injected, raising the water table in the surrounding formation and causing the contaminant to move in the opposite direction."

The seepage and migration of creosote and other wood preservation materials into the landfill occupied by Northern Wood Preservers Ltd. was illustrated by Thompson et al (1978) (Fig. A9-5). He further described the site as follows:

"The landfill consists primarily of fine sands which extend to a depth of 3 -5 metres and are underlain by a very low permeability clay. The water table in the fine sands is within one metre of surface, and the water table gradient is 0.004 to 0.002 adjacent to the Lake. The gradient is affected to a large extent by fluctuations in Lake level. The permeability of the landfill materials as indicated by the pumping test and slug tests varies considerably, but the average is estimated to be 10 metres/day at a flow gradient of 1. Based on the permeability of the sand and the gradient of the water table, the rate of migration of the creosote-water mixture to the Lake is estimated to range from 2 - 10 cm/day. This may seem slow but the distance from the spillage point to the lake is less than 12 metres so that within three months the creosote could reach the Lake."

As noted by Thompson et al (1978) the well system will prevent further contamination of the lake, but the approximately 100,000 L/day of ground water removed from the wells will require treatment.

Three processes considered for treatment of well effluent included biological oxidation, activated carbon treatment, and chemical oxidation, each to be preceded by an oil-solid-water separation step.

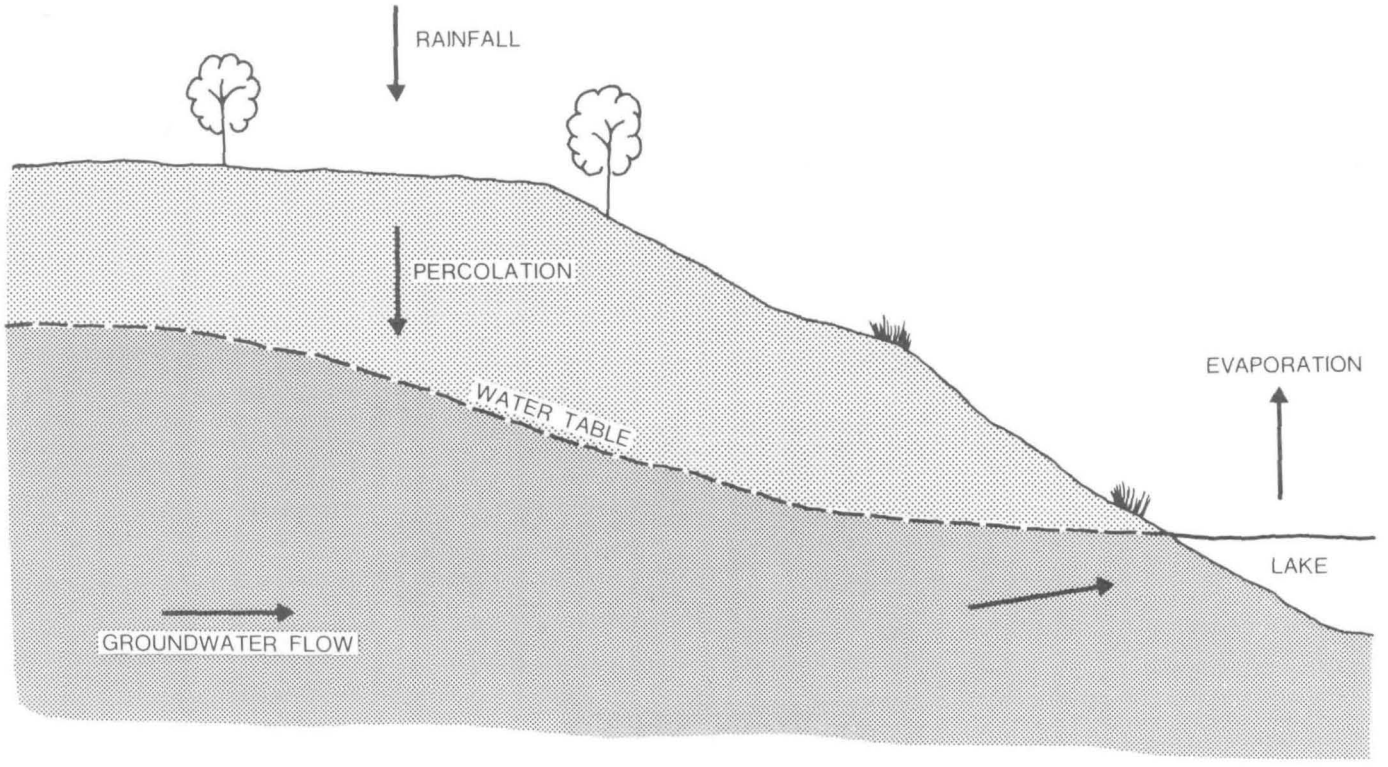


FIGURE A9-1 GENERAL GROUNDWATER SYSTEM (Thompson et al, 1978)

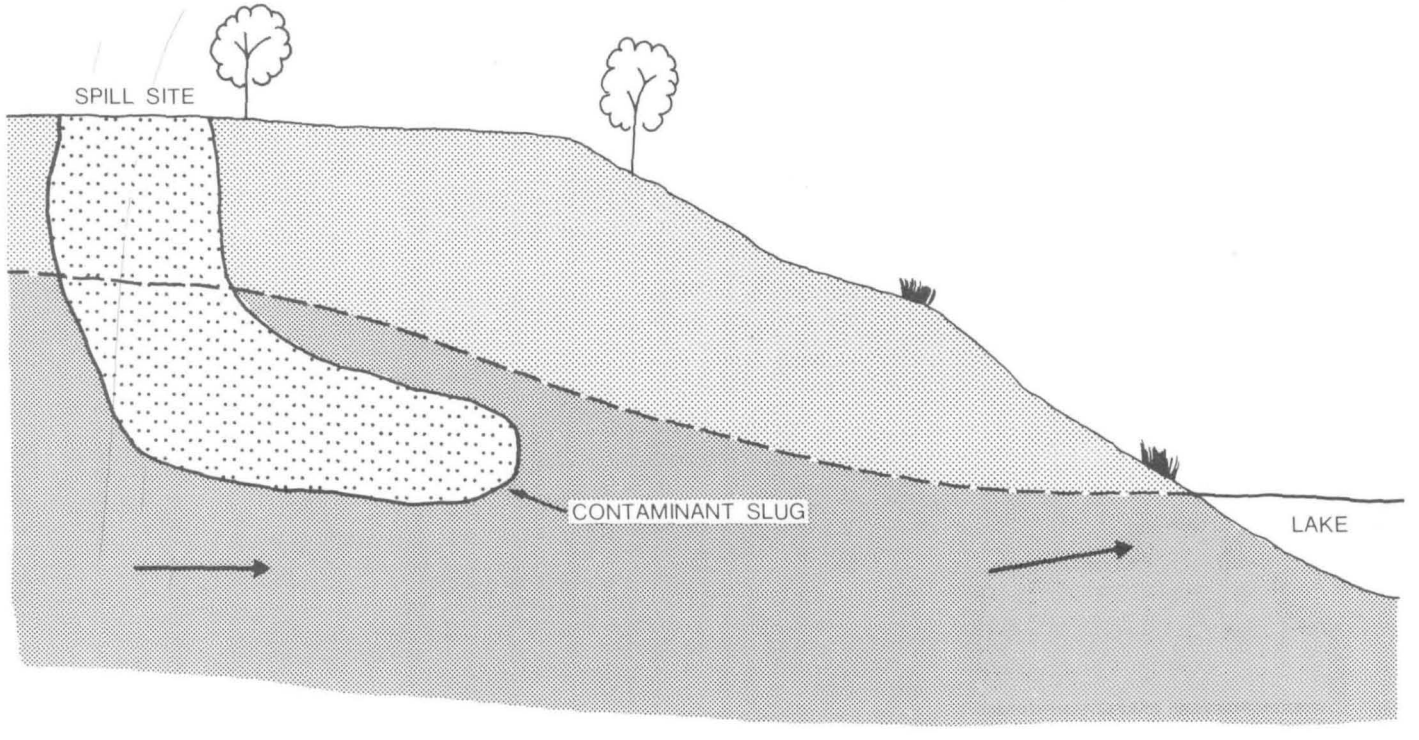


FIGURE A9-2 CONTAMINANT MOVING IN GROUND WATER FLOW SYSTEM (Thompson et al, 1978)

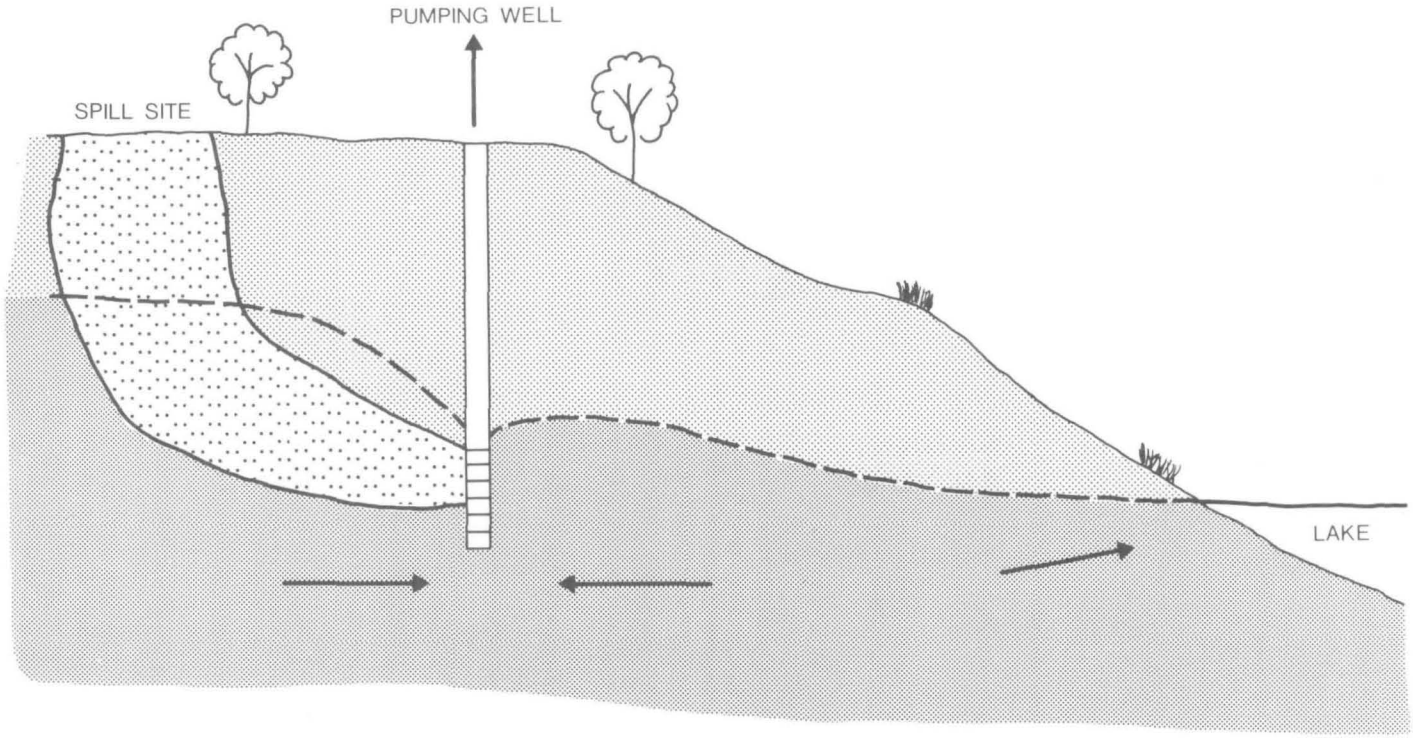


FIGURE A9-3 CONTROL AND CLEAN-UP OF CONTAMINANT USING PUMPING WELL (Thompson et al, 1978).

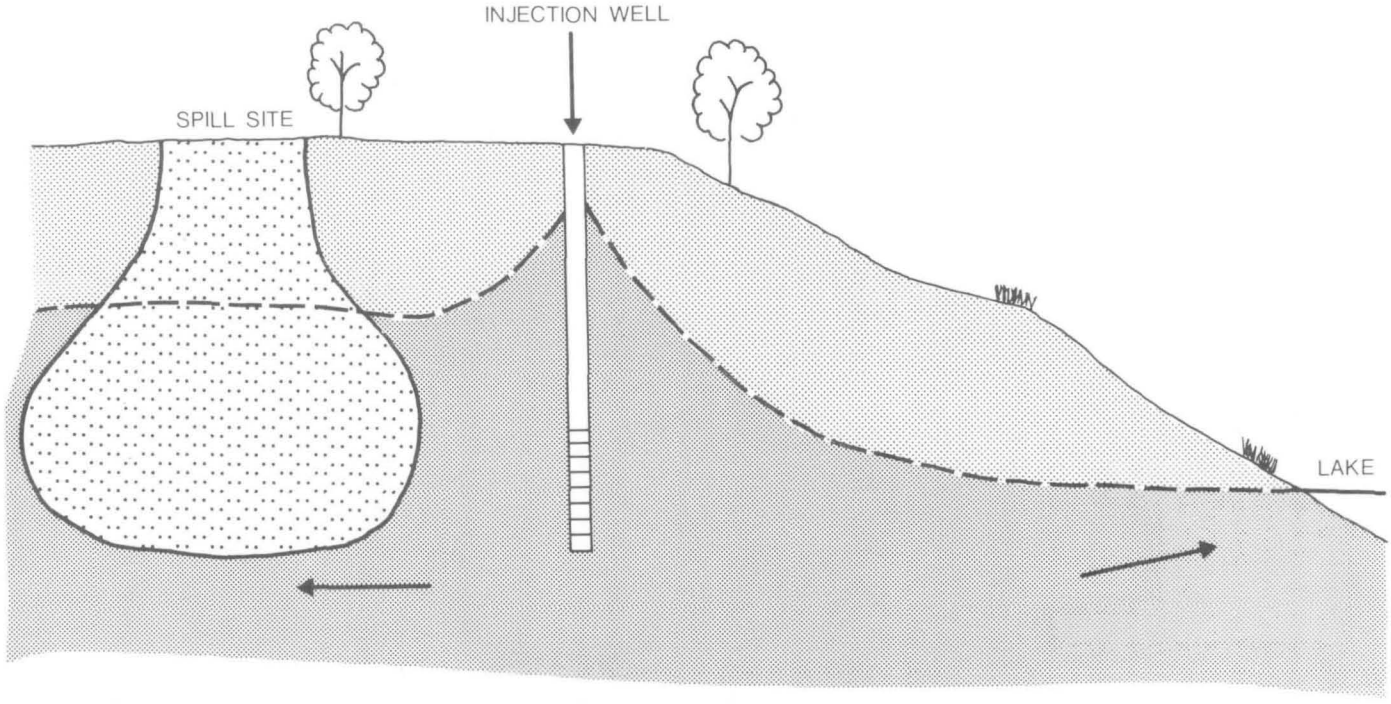


FIGURE A9-4 CONTROL OF CONTAMINANT USING INJECTION WELL (Thompson et al, 1978).

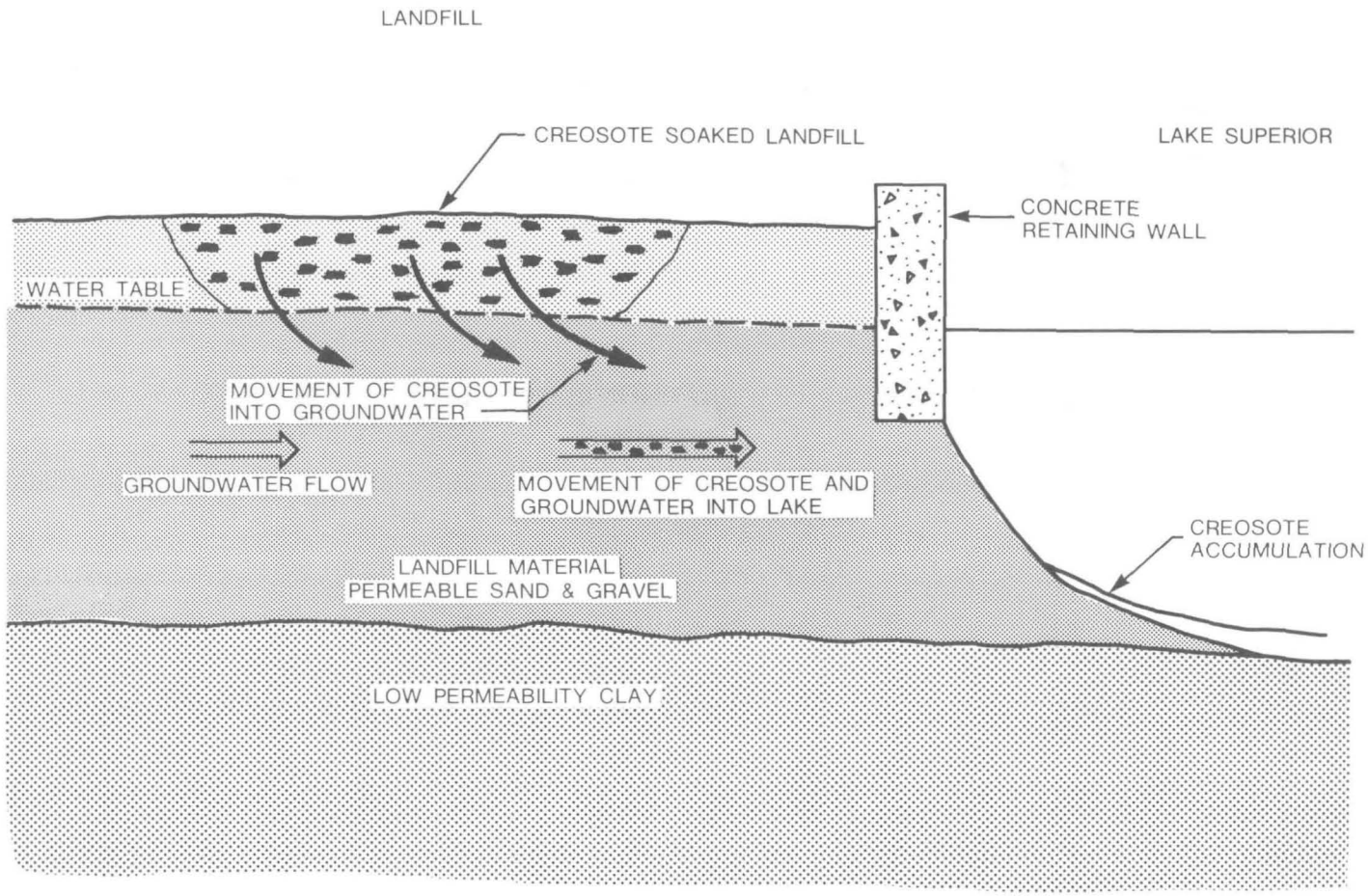


FIGURE A9-5 SCHEMATIC CROSS-SECTION THROUGH NORTHERN WOOD PRESERVERS SITE SHOWING MOVEMENT OF CREOSOTE TO LAKE SUPERIOR (Thompson et al, 1978).

Biological oxidation

In a biological oxidation pilot plant operating under stable conditions and using the contaminated ground water, fortified with additional carbonaceous material, good reductions were obtained in BOD, 400 mg/L reduced to 50 mg/L; phenols, 25 mg/L reduced to 1.1 mg/L; ss, 300 mg/L reduced to 50 mg/L; and oil, 75 mg/L reduced to 15 mg/L. Unfortunately, the concentration of PCP was reduced very little, from 3.35 mg/L to 2.5 mg/L, with the PCP still posing a significant environmental hazard (Fig. A9-6) (Thompson et al, 1978).

Thompson et al (1978) stated that:

> "On the basis of some relatively long-term tests extending over a period of approximately four months, it was concluded that very little could be done with the biological oxidation process to improve its performance in PCP removal. As a result, despite the success in phenol removal, it was decided that biological oxidation is not a viable process for treating the well point effluent at Northern Wood."

Activated carbon

Following several pilot plant runs, Thompson et al (1978) observed that the use of an activated carbon system was technically feasible and a very desirable process with respect to removal of COD, phenol, and PCP (Fig. A9-7). They did note that 0.3 m^3 of activated carbon would be required to treat each day's production from the well point system, and the carbon filters would then either require disposal or regeneration.

Similar technology to that described by Thompson et al (1978), ie., interception, pumping, and activated carbon treatment of CP contaminated ground water, was used in 1978-79 to curtail possible contamination of the Okanagan River in British Columbia, by TTCP and PCP, which had leaked from a newly installed lumber dip tank.

Chemical oxidation

Thompson et al (1978) concluded from pilot plant studies that removal of phenols and PCP from the well point effluent by ozonation was very good (Fig. A9-8). In an economic evaluation of the system, it was noted that the ozonation system did not require an auxilliary treatment process since the problem organics, phenols and PCP, were destroyed within the system.

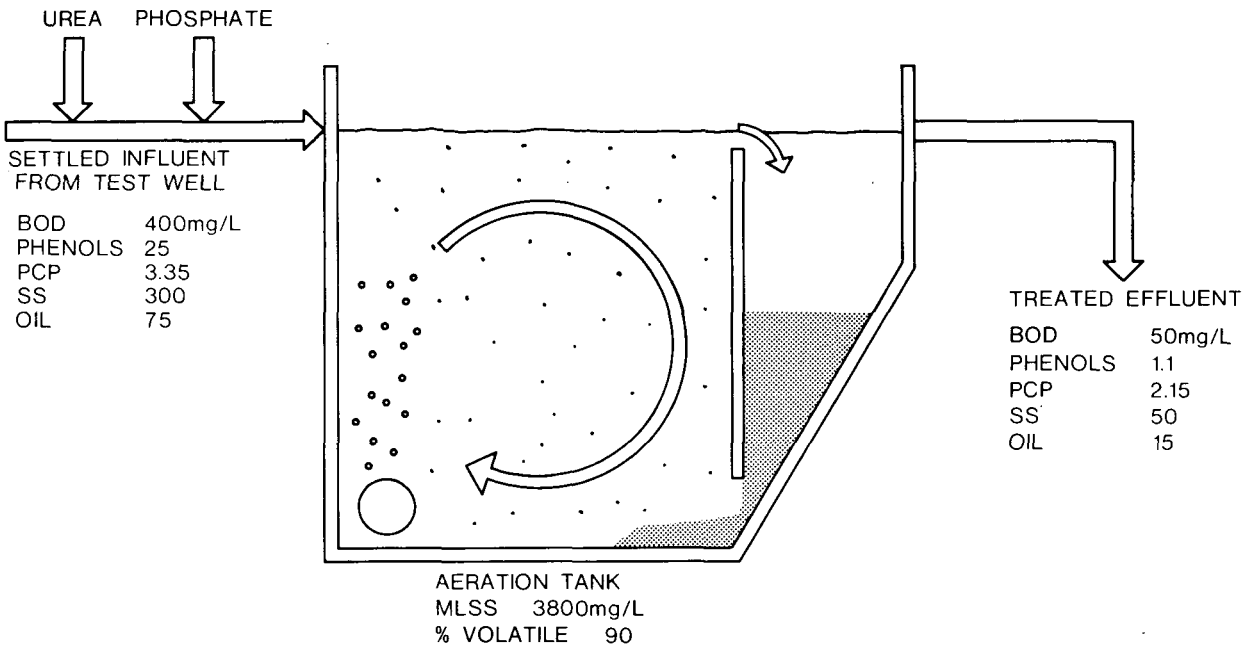


FIGURE A9-6 BIOLOGICAL OXIDATION (Thompson et al, 1978)

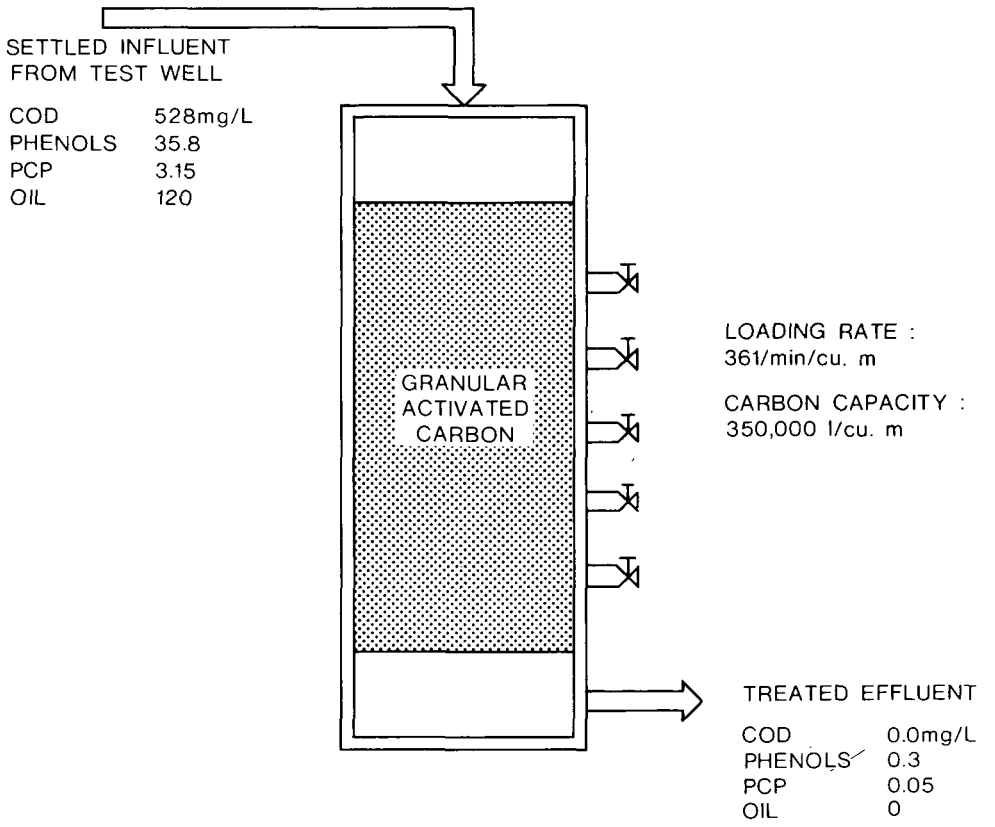


FIGURE A9-7 ACTIVATED CARBON TREATMENT (Thompson et al, 1978)

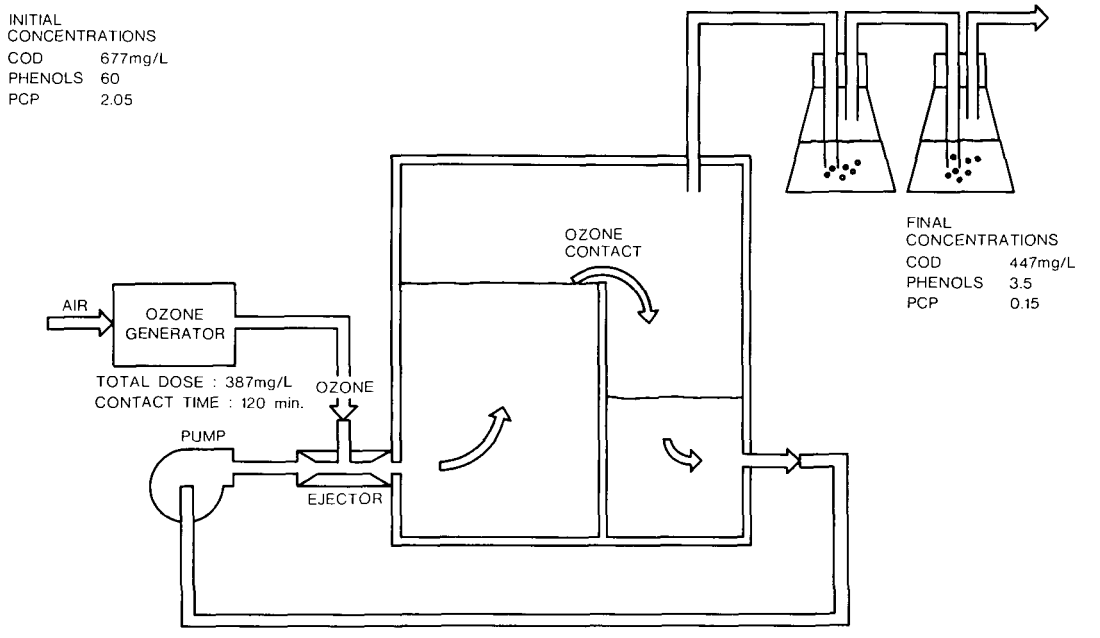


FIGURE A9-8 OZONATION (Thompson et al, 1978)

Disposal of Wood Preservation Wastes by Incineration

There has been some concern over disposal by incineration of CP contaminated wastes from wood preserving plants. Arsenault (1976) examined the effect of heating PCP-oil solution at the Boulton process temperature of 220°F at times of, from 40 to 50 h. and observed decreases in OCDD levels in both used solution and cylinder sludge, when compared to a fresh mix. He also noted that at temperatures above 300°C, PCP melts and vaporizes and affords little opportunity for the formation of OCDD. Incinerator temperatures would probably be over 300°C since wood does not generally burn at temperatures below 300°C. Arsenault (1976) did observe that when NaPCP is burned at temperatures of 550° - 800°C, OCDD was formed; however, NaPCP is not impregnated into wood, but is used for a surface treatment. No information has been obtained on the temperatures required to burn PCP-oil sludge or effluent, or sludge from NaPCP dip-tanks.

The Disposal of Chlorinated Phenol Contaminated Wastes Generated during Processing of Hides and Leathers

The use of CP products during the processing of hides and leathers has resulted in a waste disposal problem. Traditionally, hide fleshings (or scrapings) containing CPs have been utilized by the feed industry in both Canada and the U.S. to increase protein and fat content in poultry feed. The presence of toxic impurities (e.g. CDDs) in the CP contaminated wastes has led to infrequent occurrences of chick edema disease (App. 3, Sect. 3.3).

In Canada in 1978, there was a partially successful attempt made within the hide processing industry to curtail or decrease the quantities of process chemicals containing CPs. The actual quantities of NaPCP used during processing of hides and leathers in Canada has been difficult to define, although an estimate is made in Table 8.

Disposal of Chlorophenol Contaminated Wastes at Oil, Gas and Sulphur Operations in the U.S.

Outer Continental Shelf.

A waste disposal problem exists where drilling muds in oil and gas operations, particularly in marine situations, are contaminated with CPs (Sect. 4.2.2). In the United States, in order to control the problem, a ban was proposed by the U.S. Geological Survey, Department of the Interior, on the use of halogenated phenols in oil, gas, and sulphur

operations in the Outer Continental Shelf (U.S. Geological Survey, 1979a). Other less toxic bactericides are available and are being used by the U.S. petroleum industry to prevent microbial degradation and to suppress the formation of hydrogen sulfide by sulfate reducing bacteria. The U.S. Geological Survey announced in the Federal Reg. (U.S. Geological Survey, 1979b) that effective Oct. 1, 1979, the use of halogenated phenols as additives in drilling muds, completion fluids, saltwater disposal and water injection systems, workover fluids, surface production equipment, and other Outer Continental Shelf oil and gas operational systems was prohibited.

Cleaning of Metal Drums and Tanks Which Have Been Exposed to PCDD Contaminated Chemicals.

Erk et al (1979) at Wright State University, Dayton, Ohio, developed a gc-ms method for the detection and quantitation of 2,3,7,8-TCDD residues on metal surfaces. Forty samples of metal scrapings from the interiors of large herbicide holding tanks had levels of 2,3,7,8-TCDD ranging from zero (a minimum detectable concentration of 5 ng/g) to values as high as 355 ng/g. These data were used, in part, to evaluate the effectiveness of several tank cleaning procedures. Erk et al (1979) stated that the best cleaning method, known as "butterworthing", reduced the level of TCDD contamination by as much as 96%.

APPENDIX 10

APPENDIX 10

10 REGULATION OF CHLOROPHENOLS IN CANADA

Since all CPs used in Canada have pesticidal properties, commercial or domestic products marketed in Canada which contain CPs are registered under the Pest Control Products Act (PCP Act) (Bill C-50, 1968-69), administered by Agriculture Canada.

As the result of a railway car of livestock feed grains being contaminated with PCP in 1977-78, PCP has been uniquely listed as a Dangerous Good in the Transportation of Dangerous Goods Act (Bill C-17, Nov. 9, 1978). This Act is administered by Transport Canada.

An apparent anomalous situation can be related regarding product registration. Although the sole manufacturer of PCP in Canada is Uniroyal, their product does not require registration under the PCP Act but does fall under the Act's jurisdiction (Cedar, F. 1979. Personal communication).

The Control Products Section, Plant Products Division, Agriculture Canada, reevaluated the uses of the CPs in 1980. A memorandum, R-1-77, dated Sept. 24, 1979, outlined to registrants proposed deletions (suspensions) of uses, new limitations and new cautionary statements for labels of products containing CPs (Annex 1). Following the registrant's response to this memorandum, a notice, T-1-229, dated Nov. 28, 1980, noted changes in the regulatory status of the CPs effective Jan. 1, 1981 (Annex 2). Details of these new revised use patterns for the CPs are contained in Table A10-1. It should be noted that Agriculture Canada will continue the review process as additional scientific data on CPs become available.

Agriculture Canada compiled information on CPs registered under the PCP Act, for inclusion in the 1978 Wood Preservative volume of the Compendium of Pest Control Products Registered in Canada. Agriculture Canada information on CPs is excerpted and reproduced in this appendix as follows:

1) Introduction to the Wood Preservatives volume by the coordinator, Dr. F.J. Cedar.
2) Definition of terms used in the Wood Preservatives volume
3) Wood treatment terminology
4) Use claims acceptable (as of Jan. 1, 1981) for registration in Canada for products containing CPs (Table A10-1)
5) Current registrations (as of April 1, 1980) of products containing CPs (Table A10-2)

Coded information used in Tables A10-1 and A10-2 is further listed and expanded in Appendix 10, Sect. 10.1 to 10.4, inclusive, as follows: 10.1, CP products Registrants; 10.2, CP products active ingredients; 10.3, Registrant's Canadian Agents; and 10.4, CP products formulations.

FOOD PRODUCTION AND MARKETING BRANCH PLANT PRODUCTS DIVISION OTTAWA, ONTRAIO	DIRECTION DE LA PRODUCTION ET DE LA COMMERCIALISATION DES ALIMENTS DIVISION DES PRODUITS VÉGÉTAUX OTTAWA (ONTARIO)	DATE September 24, 1979	R-1-79
		SECTION PESTICIDES	

RE OBJET

MEMORANDUM TO REGISTRANTS

RE: RE-EVALUATION OF THE CHLOROPHENOLS

The purpose of this Memorandum, the second with respect to the re-evaluation of the chlorophenols, is to outline to registrants proposed deletions (suspensions) of uses, new limitations and new cautionary statements for labels of products containing chlorophenols. Any comments on the proposed regulatory actions outlined below should be addressed to Dr. F.J. Cedar, Pesticides Section, Plant Products and Quarantine Division, Agriculture Canada, Ottawa, Ontario, K1A OC6 by December 17, 1979.

In January of 1979, the following regulatory actions were taken:

1) suspension of pentachlorophenol for use as a wood preservative for use in the interiors of poultry houses;

2) suspension of pentachlorophenol for use as a disinfectant and insecticide to kill chicken mites in poultry houses;

3) limitation of the use of pentachlorophenol and sodium pentachlorophenate in leather tanning operations. Limitation: Do not use in the pretanning operations of curing, liming, soaking, and pickling from which by-product fats result.

The above actions, as well as some proposed actions, were a result of several recent incidents in Canada of poor feed conversions, increased flock mortality and undefined disease syndromes associated with intensive poultry operations as well as a "musty" taint problem in chicken meat, all linked to various uses of the chlorophenols.

Information available in the literature suggests that many of the registered uses of chlorophenol products may entail undue occupational, bystander and animal health hazards. In addition, chlorophenols and their by-products have been shown to have a variety of irreversible deleterious effects in laboratory animals. Many of these effects have been ascribed to the dibenzodioxins, dibenzofurans, and other by-products present in technical chlorophenols.

It has been known for some time that the dibenzo-p-dioxin and dibenzofuran contaminants can be separated from technical tetrachlorophenol and pentachlorophenol by presently available industrial technology. However, the problem is one of safe handling and safe disposal of the separated highly toxic waste. The technology and/or methods are not available to dispose of the chlorinated contaminants with the confidence that they will be completely destroyed. We, in Canada, therefore propose to allow the continued use of the present grades of technical chlorophenols available in the Canadian marketplace but with numerous reductions in the uses of such products where undue risk to animal and human health results.

In view of these concerns, it would seem prudent to limit human exposure to the chlorophenols to a minimum and to reduce input of chlorophenol residues into feed operations and the food chain. We therefore propose the following revisions in the regulatory status of these compounds:

1) Suspension of all products labelled for incorporation into cellulosic materials, fabrics, feathers, fibres, leather, plastics, rubber, vinyls and other polymers. This suspension covers dehydroabietylamine pentachlorophenate (DAP), fatty acid (C_6-C_{20}) esters of pentachlorophenol (PCF) and sodium pentachlorophenate (SPC) as material preservatives. End-use of treated materials may result in prolonged direct contact with the skin; e.g., in backpacks, camping gear, cots, pillows and foot wear. Information available to us is insufficient to properly evaluate human health hazards associated with such uses.

2) Suspension of all products labelled for use as herbicides and soil sterilants.

3) Suspension of products containing pentachlorophenol (PCP) and carrying label instructions for use as a wood preservative on the interior woodwork of farm buildings e.g. above ground, dry locations such as walls, floors, bins, feed troughs, silos, stalls, chicken roosts, etc. It is recommended that pentachlorophenol - 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.

4) Suspension of products containing pentachlorophenol for use as a wood preservative on wooden food containers and on horticultural lumber, e.g. seed flats, stakes, greenhouse lumber, etc.

5) Suspension of products containing sodium pentachlorophenate (SPC) for use as a fungicide in mushrooms houses and on tools for mushroom culture.

6) Suspension of products containing the chlorophenols and their sodium salts for use as slimicides in pulp and paper mill operations.

7) Suspension of all chlorophenol products carrying label instructions for use as wood preservatives and/or wood stains for INTERIOR home use.

8) Suspension of all chlorophenol products carrying label instructions for use as microbiocides for the curing of hides.

9) Suspension of all chlorophenol products and their salts for commercial application as wood preservatives and sapstain inhibitors by SPRAY methods. Spraying results in inadequate treatment coverage of wood and waste of material and poses environmental and health hazards.

Draft use standards, summarizing the label instructions which will be acceptable for full registration for 1981, are attached. Please note that the directions for use are given in metric terminology.

Additional changes in the regulatory status of the chlorophenols may take effect when suitable alternative chemicals for remaining uses become available. A prime candidate use for consideration would be that of sodium tetrachlorophenate and sodium pentachlorophenate formulations for sapstain and mold control on freshly-cut lumber. Agriculture Canada proposes that this use be phased out over the next three to five years as newer treatments of less mammalian and fish toxicity are proved effective against sapstain and are registered for use in Canada. Chlorophenol and dioxin residues have been confirmed to be present in wood chips/shavings used for animal bedding and poultry litter, are thus an animal health hazard and should be reduced and/or eliminated. Suspension of the chlorophenol sapstain use is one possible solution.

S.W. Ormrod
A/Chief
Pesticides Section

This replaces memorandum R-1-79 dated August 7, 1979

ANNEX 2

Agriculture Canada

FOOD PRODUCTION AND INSPECTION BRANCH	DIRECTION GÉNÉRALE, PRODUCTION ET INSPECTION DES ALIMENTS	DATE November 28, 1980	T-1-229
		SECTION PESTICIDES	

RE OBJET

CHANGES IN THE REGULATORY STATUS OF THE CHLOROPHENOLS

Products containing trichlorophenol, tetrachlorophenol and pentachlorophenol and their salts have been registered for use in Canada since 1949. As part of an on-going review of older pesticides, Memorandum R-1-79, dated August 7, 1979, notified registrants that the chlorophenols were to undergo a re-evaluation. Memorandum R-1-79, dated September 24, 1979, requested the comments of registrants on proposed revisions in the regulatory status of these compounds.

Information available in the literature suggests that potential occupational, bystander, human and animal health hazards may be associated with certain registered uses of chlorophenol products. Hazards have been ascribed to the dibenzodioxins, dibenzofurans and other by- products present in technical chlorophenols as microcontaminants.

In view of these concerns, Agriculture Canada announces the following revisions to the use standards, effective January 1, 1981:

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 mushroom culture;

3) Suspension of products containing pentachlorophenol 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 pentachlorophenol (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 pentachlorophenol-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;

.../2

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 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 use standards for the chlorophenols will be subject to re-review as additional scientific data becomes available. Your attention is directed to new FIRST AID statements, limitations and cautions outlined in the use standards.

Two use areas (material preservation and sapstain inhibition) will receive on-going evaluation during the next registration period. For those chlorophenols and their derivatives used as additives to textiles, the following limitation is to be added to product labels:

> "Do not incorporate into materials of which end use will result in prolonged direct skin contact e.g. life jackets, sleeping bags, sports equipment."

This notice is issued under the authority of the Pest Control Products Act and Section 20 of the Regulations. Registrants should take note of Section 22 of the Regulations under the Pest Control Products Act for an understanding of the regulatory effects of a notice of suspension.

Use patterns for the chlorophenols summarizing all of the label instructions acceptable for registration under the Pest Control Products Act may be obtained from the Chief, Technical Services Unit, Pesticides Section, Agriculture Canada, K.W. Neatby Building, Ottawa, K1A 0C6.

Registrants may amend their registration by submitting applications for amendment, including draft labels, in compliance with this memorandum. Applications for new registrations should include draft labels in compliance with this memorandum.

S.W. Ormrod
Associate Director (Pesticides)
Plant Products and Quarantine Division

This replaces Memorandum R-1-79 dated September 24, 1979.

Distribution: Registrants of chlorophenols; PPD-2; PCP-3; PCP-7 PCP-10; PCP-12.

Introduction to the Wood Preservatives Volume of the Compendium of Pest Control Products Registered in Canada

Dr. F.J. Cedar

1. SCOPE: This volume includes summaries of label instructions and cautions for wood preservatives accepted for registration under the Pest Control Products Act. As used here, the expression "wood preservative" includes products for control of wood-attacking fungi (responsible for decay, mold, sap stain and soft rot) and insects (termites, powder post beetles, carpenter ants, woodworms, etc.).

   Most instructions outlined will be found on labels of pesticides currently registered under the Pest Control Products Act. There are a few instructions listed which do not relate to any registered label because products or instructions have been discontinued by manufacturers, but are still acceptable for reregistration.

   Other instructions which do not appear on labels have been listed as acceptable for registration on the basis of data submitted by provincial and federal agencies concerned with pest control.

   Please note that the instructions and cautions presented here do not correspond exactly with those on product labels. Product labels are usually more detailed and more specific than these outlines. The registered product label is the authority in setting forth how a product may be used in the field.

2. LAYOUT: This volume is organized on an active ingredient basis, and each use pattern sets forth the accepted uses for an active ingredient, and for mixtures containing that active ingredient. Each active ingredient is represented by a three-letter code; a list of these three-letter codes follows the introduction. The three-letter code occurs as part of each page number for each use pattern.

3. COMPENDIUM VOLUME CODES: Each volume of the Compendium has been assigned a two-letter code. These codes are used for cross-references among volumes. The volume codes are as follows:

   HS disinfectants for environmental hard surfaces

IN acaricide, insecticide, insect repellent, nematocide
MP material preservatives
PD plant disease control products
RP list of registered products
SL slimicides
SW swimming pool chemicals
WD herbicides
WP wood preservatives

4. PESTICIDES NOMENCLATURE: The common names used in this compilation are as defined in the Canadian Standards Association Standard Z143, Common Names for Pest Control Chemicals, or a trivial name.
5. MARKETING TYPES: The rate of application line for each use carries a designation of DOM, COM, RES, or several of these codes. These represent the marketing classifications accepted by Control Products Section. It should be noted that, in some instances, provincial legislation provides a more stringent classification than that assigned by the federal agency.
6. CAUTIONS: Caution statements included in the use patterns do not necessarily apply to every formulation, package size, or type of use. Some judgement must be used in identifying the warnings which are appropriate to a particular situation.
7. RATES OF APPLICATION: All rates of application are expressed in terms of active ingredient.
8. MIXTURES: Mixtures with other ingredients are entered in the use pattern in alphabetical order by ingredient code, with the additional restriction that two-component mixtures are entered first, then three-component, four-component, five-component mixtures, etc.
9. UPDATE: A use pattern can be assumed to include all uses which have been accepted up to the end of the month entered at the top of the first page. For information about subsequent registration, check the Compendium Supplements - WOOD PRESERVATIVES.
10. LIST OF REGISTERED PRODUCTS: The lists of registered products are available and are published as a separate volume of the Compendium, under code RP. A list of registered wood preservatives is printed semi-annually and is available on request from the Control Products Section.

March 6, 1978

DEFINITIONS OF TERMS

EXTERIOR STRUCTURAL LUMBER - includes shingles, roofs, troughs, siding, sills, boardwalks, patios, bridges, dams, fences, foundations, docks, boats, boat houses, outdoor furniture, truckbeds (for non-food use).

FIELD CUTS - site cuttings, end-cuts of foundation lumber.

HORTICULTURAL LUMBER - includes greenhouse flats, field crates, lug boxes, pallets, stakes (e.g. for grapes, tomatoes).

INTERIOR STRUCTURAL LUMBER - includes beams, subfloors, joists, floors, greenhouse lumber, farm buildings, ice-houses.

LUMBER - solid wood product, less than 4 inches (100 mm) in width.

POLES - timber greater than twelve feet in length.

POSTS - timber twelve feet or less in length.

STANDING POSTS AND POLES - inground posts and poles (referring mainly to a secondary treatment).

STRUCTURAL MATERIALS - includes plywood, particleboard, pressed board, flaked board (these are not considered lumber).

TIMBER - solid wood product, four inches (100 mm) or more in thickness and four inches (100 mm) or over in width.

WOOD TREATMENT TERMINOLOGY

In determining a treatment's effectiveness, the method by which preservatives are impregnated into the wood is as important as the type of preservative used. The desired characteristic of a good treatment is an ability to secure a deep and reasonably uniform penetration with a preservative retention appropriate to the ultimate end use of the product. For some purposes, adequate preservation can be accomplished through the use of relatively cheap, quick and easy processes such as dipping, steeping, brushing and spraying. These are non-pressure processes, the effectiveness of which depends on the species being treated, the moisture content, the properties of the wood, the preservative used and the thoroughness with which the procedures are carried out. Diffusion is the primary driving force of non-pressure application.

For most commercial and industrial purposes where maximum protection and durability is desired, the wood must be pressure processed to force the preservatives into the wood for greater penetration and retention.

A brief outline of the types of treatment follows:

1. PRESSURE TREATMENT. This is the best method for treating wood, particularly wood to be used under conditions of severe decay potential. It is an industrial process, performed at commercial pressure treatment plants. Wood to be treated must first be kiln dried or well seasoned to remove water, then enclosed in a sealed cylinder or retort, submersed in a liquid preservative and subjected to pressure. There are two pressure treatment techniques. The full-cell process retains the liquid in the wood by filling the wood cells. The empty-cell process achieves the same depth of penetration but less retention of the liquid by coating the walls of the cells. Neither process actually leaves all the cells completely empty or full.
2. BRUSH OR SPRAY. These are applications to the surface of the wood and are useful for certain exterior woodwork. Such treatments may add one to three years to the life of a wood product. They are not recommended for preservation of fence posts or other wood in severe decay situations.
3. DIPPING. Dipping for a few seconds to 15 minutes in an oil-type preservative, commonly penta, is adequate for wood where the decay potential is low. Its primary use is for pre-cut sash, frames and millwork. Although penetration across the grain is negligible, penetration into the cut ends may be appreciable. Wood should be trimmed before dipping.

4. PASTE AND/OR BANDAGE. Used for groundline treatments of standing poles and other "in place" applications.
5. SOAKING. A practical and economical method for treating fence post that can result in 20 or more years service life for the posts. Hot-and-cold open tank treatments have shown over 35 years life for fence posts (test conducted by Eastern Forest Products Laboratory, Department of Fisheries and Environment, Ottawa). Soaking utilizes an oil-borne solution of a preservative. Hot-and-cold tank process uses mainly creosote. The mechanism of absorption is by capillary movement and will vary depending on the species of posts and type of oil solvent used. Posts should be peeled, air-dried and the ends trimmed before treatment.
6. STEEPING. Steeping involves submerging seasoned or green timbers in a solution of a water-soluble preservative salt for periods ranging from several days to several weeks. Penetration of the chemical will depend on the extent of seasoning of the wood and on the duration of the steeping bath. Seasoned posts generally require about one week and green posts about two weeks. Preservative uptake could be enhanced by heating the solution.

TABLE A10-1 SUMMARIES OF USES FOR CHLOROPHENOL COMPOUNDS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT.

Information from Pesticides Section, Agriculture Canada, November 1980.

Table Contents

TCH-TC January 1981 (P2)

2,4,5-TRICHLOROPHENOL

Common Name: 2,4,5-trichlorophenol

Chemical Name: 2,4,5-trichlorophenol

Other Names:

Category: material preservative

Guarantee in Terms of: 2,4,5-trichlorophenol

Marketing Types: COM commercial

Formulations: SO solid

SN solution

Cautions: Keep out of reach of children. Avoid contact with skin, eyes, and clothing. Causes burns of eyes and skin. Wear goggles and rubber gloves when handling. Avoid breathing dusts. Wash thoroughly after handling. Harmful if swallowed. Keep away from animals. Do not contaminate foods or feeds. Keep out of potable water supplies. Toxic to fish; keep out of lakes, ponds and streams. Do not contaminate water by cleaning of equipment or disposal of wastes.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient to the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information: Any effects on users will be largely due to the acidic nature of the product. After thorough washing, treat as per acid burns.

Decontamination and Disposal: Do not reuse empty containers. Return to drum reconditioner or destroy by perforating or crushing. Bury with waste away from water supplies.

LIMITATIONS:

1. Products containing this chemical are not to be used in food packaging materials or in areas where food is stored, handled, or processed.

2. Do not collect for further use run-off water from treated roof.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

ADHESIVES
(POLYVINYL ACETATE
EMULSIONS)

resists fungi — COM 95% product #SO
ADDITIVE: Dissolve in oil phase of emulsion to give 0.4% by weight of total adhesive solution.
Limitation (1)

AUTOMOTIVE
RUBBER GASKETS

resists fungi

COM 95% product #SO
ADDITIVE: Mix into molten rubber to give 0.5 to 1.0% by weight.

TEXTILES (RAYON)
resists fungi

COM 95% product #SO
ADDITIVE: To preserve emulsions used for rayon spinning, dissolve in oil phase to give 0.1% by weight of emulsion.
Limitation (1)

MIXTURES CONTAINING 2,4,5-TRICHLOROPHENOL

1. with pentachlorophenol plus related chlorophenols (PCP)

ROOFS, WALKS, WALLS
(BRICK, CONCRETE, WOOD)
kills moss

COM TCH: 6.7% PCP: 19% product #SN
SPRINKLE OR BRUSH-ON: Dilute the concentrate with water in a ratio of 1:12. For heavy moss deposits, apply 2.7 L of diluted solution per 10 m^2 of wall, roof or walk area. For light moss deposits, apply 1.6 L of diluted solution per 10 m^2 of wall, roof, or walk area. Apply enough to wet the roots, but just short of run-off. Do not apply when moss is wet with rain water as wet moss will not absorb the product. Avoid stepping on treated areas while wet. Heavy deposits of dead moss can be raked off, if desired, one week after treatment. Chemical control is most effective when rain is not likely for several days.
Houses with no gutters: Exercise special care. Do not allow solution to run directly off edges onto plants. Cover plants with plastic sheets to assure safety. Do not apply in rain.
Limitation (2)

STD-TC January 1981 (P2)

SODIUM 2,4,5-TRICHLOROPHENATE

Common Name: sodium trichlorophenate

Chemical Name: sodium 2,4,5-trichlorophenate

Other Names:

Categorization: materials preservative, slimicide, wood preservative

Guarantee in Terms of: sodium 2,4,5-trichlorophenate

Marketing Types: commercial

Formulations: SN solution

Cautions: Keep out of reach of children. Causes skin irritation. Harmful if swallowed. Do not get in eyes, or on skin, or on clothing. Wear rubber gloves for handling. Wash thoroughly after handling. Toxic to fish and wildlife. Keep out of lakes, streams, and ponds. Do not contaminate water by cleaning of equipment or disposal of wastes or containers.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient to the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information: Ingestion may cause immediate burning in the mouth and rapidly developing confusion and weakness with labored breathing. Gastric lavage should be carried out promptly, followed by oxygen and artificial respiration to support breathing.

Decontamination and Disposal: Do not reuse empty containers. Return to drum reconditioner, or discard by perforating or crushing and burying in a safe place.

Limitations:

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

MIXTURES CONTAINING SODIUM 2,4,5-TRICHLOROPHENATE

1. with sodium pentachlorophenate plus related chlorophenates (SPC)

See SPC use pattern.

2. with isopropyl alcohol (IAL), nabam (NAB), and sodium pentachlorophenate plus related chlorophenates (SPC)

See SPC use pattern.

3. with isopropyl alcohol (IAL), n-alkyl (50% C_{14}, 40% C_{12}, 10% C_{16}) dimethyl benzyl ammonium chloride (QAC), sodium dimethyldithiocarbamate (SDD), and sodium pentachlorophenate plus related chlorophenates (SPC)

See SPC use pattern.

TCP-WP January 1981 (P3)

TETRACHLOROPHENOL PLUS RELATED CHLOROPHENOLS

Common Name: tetrachlorophenol

Chemical Name: 2,3,4,6-tetrachlorophenol

Other Names:

Categorization: wood preservative

Guarantee in Terms of: tetrachlorophenol; related chlorophenols

Note: In this summary, products formulated with Dowicide EC-7 (i.e. pentachlorophenol 88%, tetrachlorophenol 12%) are not included. Refer to the PCP use standard.

Marketing Types: commercial

Formulations: EC emulsifiable concentrate
FL flake
GR granular
SN solution

Cautions: Keep out of reach of children. Harmful if swallowed or absorbed through the skin. May cause skin irritation. Avoid contact with skin, eyes, and clothing. Wash thoroughly with soap and warm water after using. Keep away from heat, flames, and sparks. Avoid breathing vapor or mist. Use with adequate ventilation. In closed quarters, wear a respirator and goggles. Wear synthetic rubber gloves to handle freshly treated wood. Product is toxic to vegetation; keep it away from desirable plants. Toxic to fish. Treated effluent should not be discharged where it will drain into lakes, streams, ponds, or public water. Do not contaminate water by cleaning of equipment or disposal of wastes.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient to the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information:

Decontamination and Disposal:

Limitations:

1. Dip tanks and storage areas for freshly dipped lumber should not be located adjacent to water bodies.
2. Dip tanks should be covered to prevent overflow during periods of precipitation.
3. Freshly dipped lumber should be allowed to "drip dry" on the apron of the dip tank before it is removed to storage areas.
4. Products treated with or containing this chemical are not to be used in food packaging materials or in areas where food is processed, handled or stored.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

MIXTURES CONTAINING TETRACHLOROPHENOL PLUS RELATED CHLOROPHENOLS

1.	with copper-8-quinolinolate (CUQ)
FRESHLY CUT LUMBER AND TIMBER resists mold, resists sapstain	TCP: 20.0% CUQ: 5.0% #EC SPRAY, DIP OR FLOWCOAT: Apply within 24 hours after cutting. For rough cut dilute one part concentrate with 300 parts water. For thicker than 50 mm lumber, tightly bundled lumber, surfaced wood or for prolonged exposure to wet conditions dilute one part concentrate with 125 to 150 parts water. For applications to plywood veneers to prevent mold prior to lay up and pressing dilute one part concentrate with 500 parts water. Consult company representatives for exact rates, dilutions and application method appropriate to various operating and exposure conditions. RATES AS ACTIVE: TCP: 12.7 CUQ 3.2 to TCP: 51 to CUQ 12.7 ml per 1000 board feet. Limitations (1)(2)(3)

STC-WP January 1981 (P3)

SODIUM TETRACHLOROPHENATE PLUS RELATED CHLOROPHENATES

Common Name: sodium tetrachlorophenate

Chemical Name: sodium tetrachlorophenate

Other Names:

Categorization: wood preservative

Guarantee in Terms of: sodium tetrachlorophenate; related chlorophenates

Marketing Types: commercial

Formulations: SN solution
SU suspension

Cautions: Keep out of reach of children. Causes skin irritation. Injurious to the eyes. May be harmful or fatal if swallowed or absorbed through the skin. Avoid contact with skin, eyes, and clothing. Do not breathe fumes. Keep from freezing. Wear goggles and rubber gloves when handling. Wash contaminated clothing before reuse. Do not apply to surfaces that may contact food or animal feed. Do not use on horticultural lumber. Toxic to fish, wildlife, farm and domestic animals. Do not contaminate water by cleaning of equipment or disposal of wastes.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient to the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information:

Decontamination and Disposal: Clean equipment by thoroughly rinsing with water and detergent followed by flushing with clean water until free of all traces of detergent. Clean empty containers by thoroughly rinsing with water. Dispose of rinsings from equipment and empty containers by burying them in a non-crop, non-graze area away from water supplies. Crush, break or puncture empty containers and bury them with the rinsings or deliver them to a sanitary land fill dump in accordance with municipal requirements (See Cautions). For additional details on disposal of empty containers and rinsings and for information about the appropriate means of disposing of unused, unwanted product contact the regional office of the Environmental Protection Service, Environment Canada. Dispose of empty containers with household garbage.

Limitations:

1. Dip tanks and storage areas for freshly dipped lumber should not be located adjacent to water bodies.
2. Dip tanks should be covered to prevent overflow during periods of precipitation.
3. Freshly dipped lumber should be allowed to "drip dry" on the apron of the dip tank before it is removed to storage areas.
4. Allow 2 to 3 weeks drying time at room temperature or higher after treating items which may come into contact with plants.

5. Do not collect for further use, runoff water from freshly treated roofs.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

FRESHLY CUT LUMBER
resists mold, sapstain

COM SPC: 24.2% #SN
DIP OR SPRAY: Apply within 24 hours of cutting. For rough lumber, dilute 1 part concentrate with 80 parts water. For surfaced lumber, dilute 1 part concentrate with 33 parts water. Protect freshly treated lumber from rain.
Limitations (1)(2)(3)

COM STC: 6.9 or 14.2% #SU
SPRAY: To be used with a Timberpellor (or similar spray apparatus) at manufacturer's recommended rates.

MIXTURES CONTAINING SODIUM TETRACHLOROPHENATE PLUS RELATED CHLOROPHENATES

1. with bis(tri-n-butyltin)oxide (BTO)

FRESHLY CUT LUMBER
resists sapstain

COM STC: 24% BTO: 1.3% #SN
DIP OR SPRAY: Apply within 24 hours after cutting. Dilute 1 part concentrate with 100 parts water. Protect vats and freshly treated lumber from the rain. For detailed information, consult company representatives.
Limitations (1)(2)(3)(4)

2. with sodium metaborate octahydrate (SMM)

LOGS, LUMBER
resists mold, sapstain

COM STC: 22.82% SMM: 13.23% #SN
DIP OR SPRAY: Apply for thorough and complete coverage of lumber or timbers. Dilute one part concentrate in 60 parts water. Certain molds may not be controlled e.g. Cephaleascus sp. Protect freshly treated lumber from the rain. Follow approved stacking practices.
Limitations (1)(2)(3)

3. with sodium pentachlorophenate plus related chlorophenates (SPC)

EXTERIOR STRUCTURAL LUMBER
(SHAKES, SHINGLES)
kills moss, resists decay

DOM STC: 7.7% SPC: 16.3% #SN
BRUSH, ROLLER, OR MOP: Dilute 1 part concentrate with 5 parts water. Heavy moss growth may require a second application. Scrape or brush all dead moss from surface and carry out preservation treatment to prevent further growth of moss.
PRESERVATION: Apply in dry weather when wind is absent or low. Protect all vegetation with polyethylene sheets. During application, downpipes should be drained into pails to prevent run-off into ground or sewers.
Limitation (5)

4. chlorophenates (SPC)	with borax (8NS) and sodium pentachlorophenate plus	109 110 111 112
FRESHLY CUT LUMBER resists mold, sapstain	COM STC: 16.32% BNS: 2.0% SPC: 7.68% #SN DIP OR SPRAY: Dilute 12.5 L of concentrate with 1000 L of water. Treat within 24 hours after cutting. Protect freshly treated lumber from the rain. Allow adequate space between boards and stacks. Do not use green timber or infected wood for stackers. Limitations (1)(2)(3)(4)	113 114 115 116 117 118 119
5. octahydrate (SMM)	with phenylmercuric lactate (PML) and sodium metaborate	120 121 122
LOGS, LUMBER resists mold, sapstain	COM STC: 22.82% PML: 0.4% SMM: 13.23% #SN DIP OR SPRAY: Apply within 24 hours after cutting. Use one part concentrate to 100 parts water. If a severe problem is expected, increase concentration to one part concentrate to 75 parts water. Protect freshly treated lumber from rain. Follow approved stacking practices. Limitations (1)(2)(3)	123 124 125 126 127 128 129 130 131

PCP-TC January 1981 (P3)

PENTACHLOROPHENOL PLUS RELATED CHLOROPHENOLS

Common Name: pentachlorophenol

Chemical Name: pentachlorophenol

Other Names:

Category: fungicide, herbicide, insecticide, materials preservative, wood preservative

Guarantee in Terms of: pentachlorophenol; related chlorophenols

NOTE: In this summary, the code PCP is used to designate technical pentachlorophenol. The content of active ingredients in 100% technical pentachlorophenol from various sources is as follows:

1. pentachlorophenol 84%;other chlorophenols 12%
2. pentachlorophenol 85%;cther chlorophenols 10%
3. pentachlorophenol 86%;other chlorophenols 10%
4. pentachlorophenol 88%;tetrachlorophenol 12%.

Marketing Types:
DOM domestic
COM commercial

Formulations:
EC emulsifiable concentrate
PA paste
PP pressurized product
GR granular
SN solution
SO solid
SU suspension

Cautions: Keep out of reach of children. Harmful if swallowed or absorbed through the skin. May cause skin irritation. Avoid contact with skin, eyes, and clothing. Wash thoroughly with soap and warm water after using. Keep away from heat, flames and sparks. Avoid breathing vapour or mist. Use with adequate ventilation. In closed quarters, wear a respirator and goggles. Wear synthetic rubber gloves to handle freshly treated wood. Product is toxic to vegetation; keep it away from desirable plants. Do not apply to surfaces that may contact food or animal feed. Do not use on horticultural lumber. Toxic to fish, wildlife, farm and domestic animals. Keep from freezing.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient tc the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information:

Decontamination and Disposal: Clean equipment by thoroughly rinsing with water and detergent followed by flushing with clean water until free of all traces of detergent. Clean empty

containers by thoroughly rinsing with water. Dispose of rinsings from equipment and empty containers by burying them in a non-crop, non-graze area away from water supplies. Crush, break or puncture empty containers and bury them with the rinsings or deliver them to a sanitary land fill dump in accordance with municipal requirements (See Cautions). For additional details on disposal of empty containers and rinsings and for information about the appropriate means of disposing of unused, unwanted product contact the regional office of the Environmental Protection Service, Environment Canada. DOM: Dispose of empty containers with household garbage.

Limitations:

1. Do not use on picnic tables, children's playground structures, or wood in contact with growing plants.
2. Do not collect for further use, run-off water from treated roof.
3. Do not use treated wood in farm buildings in areas contacted by farm animals or in structures to contain animal feed or agricultural produce unless the wood is covered with an impermeable barrier.
4. Products treated with or containing this chemical are not to be used in food packaging materials or in areas where food is processed, handled or stored.
5. Dip tanks and storage areas for freshly dipped lumber should not be located adjacent to water bodies.
6. Dip tanks should be covered to prevent overflow during periods of precipitation.
7. Freshly dipped lumber should be allowed to "drip dry" on the apron of the dip tank before it is removed to storage areas.
8. Do not use in vans employed for the transport of food.
9. This product is for contract performance use only and is not for resale.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

EXTERIOR STRUCTURAL LUMBER AND MATERIALS	
kills powder post beetles, resists insects, resists decay, resists sapstain	COM PCP: 22.2% TCP: 3.0% product #SN BRUSH ON, DIP, OR SPRAY: Dilute one part concentrate with 5 parts mineral spirits and agitate well to insure uniformity. Dip for 3 minutes or longer, and allow to dry for at least 24 hours before painting or varnishing. If dipping is not possible, brush or spray on in 2 coats, waiting at least 1 hour between applications. Limitation (1)
STRUCTURAL LUMBER (EXTERIOR) resists decay, resists mildew, resists mold	DOM 2.85% product #SN BRUSH-ON: Surface should be dry and clean. One coat is generally sufficient but a second coat may be applied if needed. Limitation (1)
STRUCTURAL LUMBER (EXTERIOR) resists decay, resists mildew, resists mold	DOM 3.1% to 3.85% product #SN BRUSH-ON OR DIP (WOOD STAIN): Apply to clean, dry wood. Two liberal coats are recommended. Limitation (1)
STANDING POLES, POSTS AND PILING	

arrests decay, resists insect attack	COM 5% product #SN IN-PLACE TREATMENT: Consult company representatives for details regarding equipment and application techniques. Limitation (9)	109 110 111 112 113
BUILDINGS-OUTDOOP SURFACES		114
resists termites	DOM COM 5% to 96% product #SN SPRAY: Dilute concentrates to form a 5% solution using kerosene, diesel oil or stove oil. Spray ground under buildings with 10 litres per 0.5 m². Replace porch items, sills, or other wood in contact with the ground with treated wood. If construction permits, dig a trench 0.5 to 0.75 metres deep around all foundations, and apply 10 litres per 0.5 m² of trench. RATES AS ACTIVE: 0.5 litre per 0.5 m²	115 116 117 118 119 120 121 122 123
SHINGLES		124
kills moss	COM 5% to 46% product #SN SPRINKLE, SPRAY OR PAINT: Dilute concentrates to a 5% solution with kerosene, diesel oil or stove oil. Five (5) litres of solution will cover 8 to 10 m². After the moss has dried, it will be blown away or can be swept off. Prevent drift to plants around the building. RATES AS ACTIVE: 50 to 60 mls per 10 m² Limitation (2)	125 126 127 128 129 130 131 132 133
POLES, POSTS, STRUCTURAL LUMBER (EXTERIOP)		134 135
resists decay, resists mildew, resists mold, kills powder post beetles, kills carpenter ants, kills termites	DOM COM 4.25% to 100% product #SN, SO BRUSH ON OR DIP: Wood should be well-seasoned before treatment. Soak 5 minutes per inch of thickness. For wood to be in contact with soil or water, soak 30 minutes to several hours per inch of thickness. Allow to dry thoroughly before use. If wood is trimmed or boxed after treatment, brush cut sections with at least two coats, allowing first coat to dry before adding the second. Do not paint until surface is thoroughly dry. Limitation (1) (3)	136 137 138 139 140 141 142 143 144 145
	COM 5% to 100% product #SN SO DOM 3.1 to 5% product #SN BRUSH ON: Dilute concentrates to a 5% solution with kerosene, diesel oil, or stove oil. Wood should be well seasoned before treatment. If wood has been painted or sealed, remove sealing materials before application. Brush over the surface, using 5 litres per 10 to 25 m². Apply at least 2 coats. Allow plenty of time between coats per product to soak into the wood. Do not paint until surface is thoroughly dry. RATES AS ACTIVE: 100 to 250 ml per 10 m² Limitation (1) (3)	146 147 148 149 150 151 152 153 154 155 156 157
	COM 5% to 100% product #SN SO DOM 3.1 to 5% product #SN Dilute concentrates to a 5% solution with kerosene, diesel oil, or	158 159 160

	stove oil. SPRAY-GUN APPLICATION: Wood should be well seasoned before treatment. If wood has been painted or sealed, remove sealing materials before application. Use a low-pressure sprayer equipped with a coarse nozzle. Hold the nozzle about 75 mm from the surface to be sprayed. Apply at least 2 heavy coats. Allow plenty of time between coats for product to soak into the wood. Do not paint until surface is thoroughly dry.	161 162 163 164 165 166 167
		168
POLES, POSTS, RAILWAY TIES, STRUCTURAL LUMBER, WOOD (CONSTRUCTION MATERIAL, FENCE POSTS, HEAVY TIMBERS, POLES)		169 170 171 172 173
resists decay, resists insect attack	COM 5% to 100% product #GR SO PRESSURE TREATMENT: Dilute concentrates to a 5% with kerosene, diesel oil or stove oil. Apply by accepted pressure process. For details, consult "Manual of Recommended Practices of the American Wood Preservers' Association, or Company representatives. Limitation (3)	174 175 176 177 178 179
		180
STANDING POLES (ROOF AREAS)		181 182
arrests decay	COM 10% product #SN POUR-ON TREATMENT: After reaching working position near pole top, open container and pour contents uniformly over pole roof. Then, install a protective pad.	183 184 185 186
		187
STANDING POLES, POSTS AND PILINGS		188 189
arrests decay, resists insect attack	COM 10% product #PA BANDAGE TREATMENT: Excavate soil away from poles to a depth of 50 to 75 cm, brush off adhering soil, and scrape away any surface decay. Wrap with treated bandage or apply a 6 mm layer to a depth of 100 mm below the last evidence of decay and 150 mm above ground line and wrap in polyethylene-coated kraft paper. If the presence of livestock or wildlife is of concern, preservative should not be left exposed above finished ground level.	190 191 192 193 194 195 196 197
		198
CONSTRUCTION MATERIALS (ASBESTOS SHINGLES, BRICK WALLS, CONCRETE BLOCKS, TILE ROOFS, OTHER INERTS)		199 200 201 202 203
resists mold	COM 100% product #GR, SO Incorporate in treating materials such as whitewash, calcimine, and cement paints to give 0.5 to 5.0% by weight. Limitation (4)	204 205 206 207
		208
LEATHER (SHOES)		209
resists mold	COM 100% product #GR, SO ADDITIVE: To impart temporary mold resistance to upper leather for shoes, add at a concentration of 2.0 to 3.5% by weight formulation.	210 211 212

TEXTILE FIBRES (BAILER TWINE, BURLAP, CABLE COVERINGS, ROPE, RUBBERIZED CANVAS BELTING		
resists mold and decay	COM 100% product #GR, SO ADDITIVE: For rope, bailer twine and burlap, apply in cordage oil to impart 0.4 to 1.0% by weight of twine or fabric. For cable coverings, melt into liquid asphalt to impart 1.0% by weight of asphalt and jute. For rubberized canvas belting, melt into liquid rubber at 0.5 to 1.0% by weight. Limitation (4)	
MIXTURES CONTAINING PENTACHLOROPHENOL PLUS RELATED CHLOROPHENOLS		
1.	with borax anhydrous (BNA)	
STANDING POLES AND POSTS		
arrests decay, resists termites	COM PCP: 10% BNA: 15.5% product #PA BANDAGE TREATMENT: Excavate soil away from poles to a depth of 50 to 75 cm, brush off adhering soil, and scrape away any surface decay. Apply a 6 mm layer to a depth of 100 mm below the last evidence of decay and 150 mm above ground line. Wrap in polyethylene-coated kraft paper. If livestock or wildlife may be exposed, place a barrier of hardware cloth over the completed bandage treatment.	
2.	with copper-8-quinolinolate (CUQ)	
FRESHLY CUT LUMBER AND TIMBER		
resists mold, resists sapstain	COM PCP: 17.6% CUQ: 5.0% TCP: 2.4% product #EC SPRAY, DIP OR FLOWCOAT: Apply within 24 hours after cutting. For rough-cut wood dilute one part concentrate with 300 parts water. For thicker than 50 mm lumber, tightly bundled lumber, surfaced wood or for prolonged exposure to wet conditions dilute one part concentrate with 125 to 150 parts water. For applications to plywood veneers to prevent mold prior to lay up and pressing dilute one part concentrate with 500 parts water. Consult company representatives for exact rates, dilutions and application methods appropriate to various operating and exposure conditions. RATES AS ACTIVE: PCP: 11.2 to 44.8 CUQ: 3.2 to 12.7 TCP: 1.5 to 6.1 ml per 1000 board feet. Limitations (5) (6) (7)	
3.	with o-phenylphenol (OPP)	
PAINTS		
resists bacteria, resists fungi	COM PCP: 50% OPP: 50% product #SO ADDITIVE: For shelf preservation of protein-based latex paints, disperse in the paint during manufacturing process	

a minimum concentration of 0.5% by weight of paint formulation. Concentration will vary with the type of formulator facility and the nature of the formulation.
Limitation (4)

4. with fatty acid (C_6-C_{20}) esters of pentachlorophenol (PCF)

CELLULOSIC MATERIALS (LOOSE), FIRE HOSE, YARNS
resists bacteria, resists fungi

COM PCP: 0.5% PCF: 25% product #EC
DIP, SPRAY, PAD OR BRUSH: Dilute as necessary with warm or cold water. Apply at levels of 4 to 8 per cent, according to the degree of protection required, or to meet the requirements of the British Standard 2087:1971. Apply to fire hose and textile piece goods by conventional impregnation techniques. Apply to yarns during winding or doubling by means of a furnishing roller. Loose fibre stock, wood fibre, and similar materials may be sprayed. Do not use concurrently with or before a shower-proofing or water-repellent treatment.
RATES AS ACTIVE: 4 to 8% of fibre weight

5. with 2,4,5-trichlorophenol (TCH)

ROOFS, WALKS, WALLS (BRICK, CONCRETE, WOOD)
kills moss

COM PCP: 19% TCH: 6.7% #SN
SPRINKLE OR BRUSH-ON: Dilute the concentrate with water in a ratio of 1:12. For heavy moss deposits, apply 2.7 L of diluted solution per 10 m^2 of wall, roof or walk area. For light moss deposits, apply 1.6 L of diluted solution per 10 m^2 of wall, roof, or walk area. Apply enough to wet the roots, but just short of run-off. Do not apply when moss is wet with rain water as wet moss will not absorb the product. Avoid stepping on treated areas while wet. Heavy deposits of dead moss can be raked off, if desired, one week after treatment. Chemical control is most effective when rain is not likely for several days.
Houses with no gutters: Exercise special care. Do not allow solution to run directly off edges onto plants. Cover plants with plastic sheets to assure safety. Do not apply in rain.
Limitation (2)

6. with 2-(thiocyanomethylthio)benzothiazole (TCM)

FRESHLY CUT LUMBER AND TIMBER
resists mold, resists sapstain

COM PCP: 10% TCM: 15% product #EC
DIP OR SPRAY: Apply within 24 hours after cutting. For lumber 50 mm or less in thickness, dilute 1.0 to 1.5 parts concentrate with 400 parts water. For timbers or lumber over 50 mm in thickness, dilute 2 to 3 parts concentrate with 400 parts water. Protect freshly treated lumber from the rain. Protect dip tanks from over-dilution. Rake sawdust from the dip tank daily. Treated wood is not to be used for construction of food-holding containers, food storage facilities,

	or food-growing buildings (such as mushroom houses).	317
	Limitations (5) (6) (7)	318
		319
7.	with zinc present as zinc naphthenate (ZNN)	320
		321
STRUCTURAL LUMBER AND TIMBER		322
resists decay, resists	DOM COM PCP: 2.0% ZNN: 1.8% product #SN	323
woodworms	BRUSH ON, DIP, OR SOAK: Apply liberally to clean, dry wood surfaces	324
	which are free of bark, paint, or polish. SMALL TIMBERS: Dip for 3	325
	minutes or brush all surfaces liberally and apply a second coat	326
	before the first is quite dry. OUTDOOR OR LARGE TIMBERS: Dip for 10	327
	minutes or brush with 3 coats.	328
	TIMBERS IN CONTACT WITH THE GROUND: Soak for at least 1 hour.	329
		330
8.	with borax anhydrous (BNA) and creosote (CRT)	331
		332
STANDING POLES AND POSTS		333
arrests decay,	COM PCP: 10% BNA: 15-15.5% CRT: 15-15.5% product #PA	334
resists termites	BANDAGE TREATMENT: Excavate soil away from poles to a depth of 50	335
	to 75 mm, brush off adhering soil, and scrape away any surface	336
	decay. Apply a 6 mm layer to a depth of 100 mm below the last	337
	evidence of decay and 150 mm above ground line. Wrap in	338
	polyethylene-coated kraft paper. If livestock or wildlife	339
	may be exposed, place a barrier of hardware cloth over the	340
resists termites	completed bandage treatment.	341
		342
9.	with dichofluanid (DCA) and lindane (LIN)	343
		344
WOODWORK (EXTERIOR)		345
resists decay,	DOM PCP: 4.8% DCA: 0.6% LIN: 0.38% product #SU	346
resists mold,		347
resists insect attack	DECORATIVE AND PRESERVATIVE STAIN: Apply by brush to wood sur-	348
	faces. For exterior use only, apply at least 2 coats: on wood	349
	subject to severe weathering conditions, apply 3 coats. Covering	350
	rate is approximately 200 g/m² in two coats. Do not treat	351
	beehives. Do not treat woodwork inside greenhouses or mushroom	352
	houses. Do not apply to surfaces which may contact food.	353
	RATES AS ACTIVE: PCP: 9.6 g DCA: 1.2 g LIN: 4.8 g per m²	354
	Limitation (4)	355
		356
10.	with creosote (CRT), sodium fluoride (SFL)	357
		358
STANDING POLES, POSTS AND PILINGS		359
		360
arrests decay,	COM PCP: 10% CRT: 15.0% SFL: 15.0% product #PA	361
resists termites	BANDAGE TREATMENT: Excavate soil away from poles to a depth of 50	362
	to 75 cm, brush off adhering soil, and scrape away any surface	363
	decay. Apply a 6 mm layer to a depth of 100 mm below the last	364
	evidence of decay and 150 mm above ground line. Wrap in	365
	polyethylene-coated kraft paper. If livestock or wildlife	366
	may be exposed, place a barrier of hardware cloth over the	367
	completed bandage treatment.	368

		369
	COM PCP: 10% CRT: 15.0% SFL: 20% product #PA	370
	BANDAGE TREATMENT: Excavate soil from pole. Position one end of	371
	bandage on pole so that it is approximately 75 mm above and 500	372
	mm below final ground-line level. Bring other end around to meet	373
	it. Secure above-ground overlap by staple, roofing nail, or date	374
	tag nail. Backfill pole.	375
		376
11.	with n-octyl bicycloheptene dicarboximide (MGK), technical	377
piperonyl butoxide (PBU), and	pyrethrins (PYR)	378
		379
VANS		380
inhibits growth of:	COM PCP: 0.10% MGK: 1.67% PBU: 1.00% PYR: 0.50% product #PP	381
bacteria, mildew,	SPACE SPRAY: Place unit upright on the floor of the empty van.	382
mold; kills: cockroaches,	Depress and lock the valve. Exit from van. Be sure that all doors	383
fleas, flies, mosquitoes,	etc. are closed to contain the fog within the van. Leave closed	384
silverfish, wasps	for 15 to 30 minutes.	385
	Limitation (8)	386
		387
12.	with creosote (CRT), dinitrophenol (DNP), potassium dichromate (KDC),	388
and sodium fluoride (SFL)		389
		390
STANDING POLES, POSTS AND PILING		391
		392
arrests decay,	COM PCP: 2.21% CRT: 20.0% DNP: 2.0% KDC: 3.1% SFL: 93.7% product #PA	393
resists termites	BANDAGE TREATMENT: Area to be treated must be free from bark and all	394
	evidence of decay. Apply a 1.6 mm (minimum) layer evenly using an	395
	ordinary paint or window brush. Where treated area comes in contact	396
	with the ground, wrap with a water-proof bandage.	397

SPC-TC January 1981 (P3)

SODIUM PENTACHLOROPHENATE PLUS RELATED CHLOROPHENATES

Common Name:

Chemical Name: sodium pentachlorophenate

Definition of code: SPC = sodium pentachlorophenate plus related chlorophenates

Other Names:

Category: crop fungicide, materials preservative, slimicide, wood preservative

Guarantee in Terms of: sodium pentachlorophenate; related chlorophenates

Marketing Types: commercial

Formulations: PE pellets
SG soluble granules
SN solution
TA tablets

Cautions: Keep out of reach of children. Causes skin irritation. Injurious to eyes. May be harmful or fatal if swallowed or absorbed through the skin. Avoid contact with skin, eyes, and clothing. Do not breathe dust. Wash thoroughly after handling. Wear chemical workers goggles when handling concentrate. Wash contaminated clothing before reuse. Toxic to fish, wildlife, farm and domestic animals. Do not apply to surfaces that may contact food or animal feed. Do not use on horticultural lumber. Do not contaminate water by cleaning of equipment or disposal of wastes.

Symptoms of Poisoning:

First Aid: A. Products Containing Petroleum Distillate: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, DO NOT INDUCE VOMITING, but rush the patient to the nearest hospital or doctor's office, taking the pesticide container with you.

B. Non-Petroleum Distillate Formulations: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information: This product is a metabolic stimulant, and causes hyperthermia. Treat symptomatically.

Decontamination and Disposal: Clean equipment by thoroughly rinsing with water and detergent followed by a flushing with clean water until free of all traces of detergent. Clean empty containers by thoroughly rinsing with water. Dispose of rinsings from equipment and empty containers by burying them in a non-crop, non-graze area away from water supplies. Crush, break or puncture empty containers and bury them with the rinsings or deliver them to a sanitary land fill dump in accordance with municipal requirements (See Cautions). For additional details on disposal of empty containers and rinsings and for information about the appropriate means of disposing of unused, unwanted product contact the regional office of the Environmental Protection Service, Environment Canada. DOM: Dispose of empty containers with household garbage.

Limitations:

1. Products treated with or containing this chemical are not to be used in food packaging materials or in areas where food is processed, handled, or stored.

2. Do not use in the pretanning operations of curing, liming, soaking, and pickling from which by-product fats result.
3. This product is not to be used in the production of paper or paperboard that comes in contact with food.
4. Do not incorporate into materials whose end use will result in prolonged direct skin contact e.g. life jackets, sleeping bags, sports equipment.
5. Dip tanks and storage areas for freshly dipped lumber should not be located adjacent to water bodies.
6. Dip tanks should be covered to prevent overflow during periods of precipitation.
7. Freshly dipped lumber should be allowed to "drip dry" on the apron of the dip tank before it is removed to storage areas.
8. Allow 2 to 3 weeks drying time at room temperature or higher after treating items which may come into contact with plants.
9. Do not collect for further, use runoff water from treated roof.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

ADHESIVES	
resists bacteria, resists fungi	COM 90% product #SG ADDITIVE: For adhesives based on starch, vegetable protein, and animal protein, add as concentrated aqueous solution to result in 0.25 to 1.0% by weight of material treated. Limitations (1)
CONSTRUCTION MATERIALS (ASBESTOS SHINGLES, BRICK WALLS, CONCRETE BLOCKS, TILE ROOFS, PIPE SEALING COMPOUND, WALLBOARD)	
resists mold	COM 90% product #SG Rinse previously cleaned inert surface with a 1% aqueous solution. ADDITIVE: Incorporate into material on a 0.5 to 3.0% by weight of product basis.
LEATHER	
resists bacteria, resists fungi	COM 90% product #SG To prevent deterioration of hides and treating solutions during tanning, dyeing, lubrication and finishing steps, add as a concentrated aqueous solution to result in 0.06 to 10.0% by weight in water or 0.5 to 3.0% by weight of product in which incorporated. Limitations (2)
LEATHER, PAPER, TEXTILES	
bacteriostasis	COM 10 to 20% product #SN Deposit 0.5 to 2.5% based on the dry weight of material being treated. Use conventional methods of application. Limitations (1) (2) (3) (4)
PAINT	
resists bacteria,	COM 90% product #SG

resists mold	ADDITIVE: To preserve solutions or dispersions of decomposable raw materials which are to be stored before addition to the paint formulation, add a minimum of 0.6% by weight as a concentrated aqueous solution.	109 110 111 112
		113
PETROLEUM (ENHANCED OIL RECOVERY)		114 115
resists bacteria	COM 90% product #SG To prevent bacterial growth in drilling muds, gypsum muds, packer fluids and underground strata, add as a concentrated aqueous solution at a rate of 125 to 250 g per 160 L of mud or packer fluid or 15 to 40 ppm in underground flooding water.	116 117 118 119 120
		121
PHOTOGRAPHIC SOLUTIONS		122 123
resists fungi, resists slime	COM 90% product #SG ADDITIVE: Add as a concentrated aqueous solution to make up 0.05 to 0.2% by weight of photographic solutions.	124 125 126
		127
PULP AND PAPER		128
resists mildew, resists mold	COM 90% product #SG To preserve processing materials and paper machine felts, and to protect finished paper and fiberboard products, add as a concentrated aqueous solution to give 0.1 to 1.0% by weight of paper or processing material. Limitations (3)	129 130 131 132 133 134
		135
TEXTILES		136
resists mold, resists fungi	COM 90% product #SG ADDITIVE: To preserve processing materials (Warp sizings, gray goods, printing pastes, finishing solutions), add directly or apply as a concentrated aqueous solution to result in 0.1 to 0.75% by weight of material treated. Limitations (1) (4)	137 138 139 140 141 142
		143
WATER COOLING SYSTEMS (RECIRCULATING)		144 145
slime (algal, bacterial, fungal)	COM 25-150 ppm #PE SG SN TA INITIAL: Apply at the rate of 100-150 parts of active ingredient per million of water. Add to the cooling tower basin, cold water sump, or other point which gives good mixing. Repeat at predetermined intervals until control is achieved. MAINTENANCE: Add at the rate of 25 to 50 parts per million at intervals of 2 to 5 days, depending on the amount of bleed-off, or as needed to maintain control. Consult company representatives for detailed instructions.	146 147 148 149 150 151 152 153 154 155
		156
MIXTURES CONTAINING SODIUM PENTACHLOROPHENATE PLUS RELATED CHLOROPHENATES		157
		158
1.	with borax (BNS)	159
		160

FRESHLY CUT LUMBER		161
resists mold, sapstain	COM SPC: 450 g BNS: 700 g to COM SPC: 0.8 kg BNS: 1.25 kg lb in 100	162
	litres of water #SG	163
	DIP: Apply within 24 hours after cutting. Use the lower	164
	concentration for lumber 50 mm or less in thickness, the higher	165
	concentration for thicker lumber or bundled lath or shook.	166
	A dip of 15 seconds or more is sufficient. Protect dipping tank	167
	from the rain. Do not leave lumber piles unprotected in heavy	168
	rains, especially just after dipping. Do not use green lumber	169
	for stackers.	170
	Limitations (5) (6) (7) (8)	171
		172
2.	with sodium o-phenylphenate (SOP)	173
		174
ADHESIVES		175
resists bacteria,	COM SPC: 50% SOP: 50% product #SG	176
resists fungi	ADDITIVE: For additives based on starch, vegetable protein, and	177
	animal protein, add as a concentrated aqueous solution to result in	178
	0.25 to 1.0% by weight of material treated.	179
	Limitations (1)	180
		181
GRAPHITE		182
resists bacteria,	COM SPC: 50% SOP: 50% product #SG	183
resists mold	ADDITIVE: To preserve protein colloidal graphite formulations,	184
	add as concentrated aqueous solution to result in 1.0% by	185
	weight of graphite.	186
		187
PAINT		188
resists bacteria	COM SPC: 50% SOP: 50% product #SG	189
resists mold	ADDITIVE: To preserve solutions or dispersions of decomposable	190
	raw materials which are to be stored before addition to the	191
	paint formulation , add a minimum of 0.6% by weight as a	192
	concentrated 50:50 aqueous solution.	193
		194
	ADDITIVE: For shelf preservation of protein-based latex paints,	195
	disperse in the paint during manufacturing process at minimum	196
	concentration of 0.6% by weight of paint formulation. Concentration	197
	will vary with the type of formulator facility and the nature of	198
	the formulation.	199
		200
3.	with sodium tetrachlorophenate plus related chlorophenates (STC)	201
		202
EXTERIOR STRUCTURAL LUMBER		203
(SHAKES, SHINGLES)		204
kills moss	DOM SPC: 16.3% STC: 7.7% product #SN	205
	BRUSH, ROLLER, OR MOP: Dilute 1 part concentrate with 5 parts water.	206
	Heavy moss growth may require a second application. Scrape or brush	207
	all dead moss from surface and carry out preservation treatment to	208
	prevent further growth of moss. PRESERVATION: Apply in dry weather	209
	when wind is absent or low.	210
	Protect all vegetation with polyethylene sheets. During	211
	application, downpipes should be drained into pails to prevent	212

	run-off into ground or sewers.	213
	Limitations (9)	214
		215
4.	with sodium 2,4,5-trichlorophenate (STD)	216
		217
WATER COOLING SYSTEMS (RECIRCULATING)		218
		219
slime (algal, bacterial)	COM SPC: 32% STD: 8% product #SN	220
	Initial shock dose of 50 to 150 ppm is recommended. After control is	221
	evident, reduce shock dosages to 50 to 75 ppm, or apply less often.	222
	See Product Bulletin for additional information.	223
	RATES AS ACTIVE: SPC 16-24-48 ppm STD 3-6-12 ppm.	224
		225
FRESHLY CUT LUMBER		226
resists mold, sapstain	COM SPC: 200 g STD: 550 g to COM SPC: 400 g STD: 1.10 kg in 1000 L #SN	227
	DIP: Treat within 24 hours after cutting. Use the lower rate on	228
	rough-cut lumber, the higher rate on dressed and surfaced lumber.	229
	Protect freshly treated lumber from the rain.	230
	Limitations (5) (6) (7) (8)	231
		232
	COM SPC: 300 g STD: 825 g to COM SPC: 600 g STD: 1.65 kg in 1000 L #SN	233
	SPRAY: Treat within 24 hours after cutting. Spray to completely	234
	wet lumber. Use the lower rate on rough-cut lumber, the higher	235
	rate on dressed and surfaced lumber. Protect freshly treated lumber	236
	from the rain.	237
	Limitations (5) (6) (7) (8)	238
		239
FRESHLY CUT		240
LUMBER AND TIMBER		241
resists mold, sapstain	COM SPC: 25% STD: 3.0% product #SN	242
	DIP: Treat freshly cut lumber or timber for 15 seconds or more.	243
	For lumber 50 mm or less in thickness, dilute one part concentrate	244
	with 100 parts water. For thicker lumber or timbers or bundled lath	245
	or shook, or under unusually severe conditions, dilute one part	246
	concentrate with 30 to 50 parts water.	247
	Limitations (5) (6) (7) (8)	248
		249
5.	with borax (BNS) and sodium tetrachlorophenate plus related	250
chlorophenates (STC)		251
		252
FRESHLY CUT LUMBER		253
resists mold, sapstain	COM SPC: 7.68% BNS: 2.0% STC: 16.32% product #SN	254
	DIP OR SPRAY: Dilute 12.5 L of concentrate with 1000 L of water.	255
	Treat within 24 hours after cutting. Protect dipping tanks from the	256
	rain. Protect freshly treated lumber from the rain. Allow adequate	257
	space between boards and stacks. Do not use green timber or infected	258
	wood for stackers.	259
	Limitations (5) (6) (7) (8)	260
		261
6.	with isopropyl alcohol (IAL), nabam (NAB), and sodium	262
2,4,5-trichlorophenate (STD)		263
		264

HIDES, LEATHER		265
resists bacteria, resists fungi	COM SPC: 14.1% IAL: 10% NAE: 7.45% STD: 17.3% product #SN For use in hide curing and leather manufacturing. See Product Data Sheet for further details. Limitations (2)	266 267 268 269
		270
WATER COOLING SYSTEMS		271
slime (algal, bacterial, fungal)	COM SPC: 14.1% IAL: 10% NAE: 7.45% STD: 17.3% product #SN Slug feed when slime is noted, using 20 to 200 ppm. Or, continuous or daily additicns of 20 to 50 ppm may be used. For further details see Prcduct Data Sheet. RATES AS ACTIVE: SPC 2.82-7.05-28.2 ppm IAL 2-5-20 ppm NAB 1.49-3.725-14.9 ppm STD 3.46-8.65-34.6 ppm	272 273 274 275 276 277
		278
7.	with isopropyl alcohol (IAL), n-alkyl (50% C_{14}, 40% C_{12}, 10% C_{16})	279
dimethyl benzyl ammonium chloride (QAC), sodium dimethyldithiocarbamate (SDD), and sodium 2,4,5-trichlorophenate (STD)		280 281
		282
WATER COOLING SYSTEMS (RECIRCULATING)		283 284
slime (algal, bacterial, fungal)	COM SPC: 27.6% IAL: 10% QAC: 5% SDD: 4% STD: 9.1% product #SN Feed intermittently daily, or as needed, at a rate of 20 to 100 g/kL of water in the system. Heavily fouled systems should be cleaned before treatment. Technical advice regarding specific site problems is available from company representatives. RATES AS ACTIVE: SPC 5.52-33.12 ppm IAL 2-12 ppm QAC 1.5-6 ppm SDD 0.8-4.8 ppm STD 1.82-10.92 ppm.	285 286 287 288 289 290 291

DAP-MP January 1981 (P2)

DEHYDROABIETYLAMINE PENTACHLOROPHENATE

Common Name: none approved

Chemical Name: dehydroabietylamine pentachlorophenate

Other Names:

Category: materials preservative

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

Products containing dehydroabietylamine pentachlorophenate are no longer acceptable for registration on a precedent basis. Any application for registration are to be referred to the Evaluation Unit for re-establishement of standards.

PCF-MP January 1981 (P2)

FATTY ACID (C_6-C_{20}) ESTERS OF PENTACHLOROPHENOL

Common Name: none approved

Chemical Name: fatty acid (C_6-C_{20}) esters of pentachlorophenol

Other Names: pentachlorophenyl laurate
lauryl pentachlorophenate

Category: materials preservative

Guarantee in Terms of: fatty acid (C_6-C_{20}) esters of pentachlorophenol

Marketing Types: commercial

Formulations: EC emulsifiable concentrate
SN solution

Cautions: Keep out of reach of children. Harmful if swallowed. Do not allow in direct contact with skin. Do not allow material to be splashed in eyes. Avoid contact with clothing.

Symptoms of Poisoning:

First Aid: In case of poisoning, call a physician immediately. IF ON SKIN, remove contaminated clothing and wash skin thoroughly with soap and water. IF IN EYES, flush with water for 5-10 minutes and obtain medical attention. IF SWALLOWED, give the patient one to two glasses of water, and cause vomiting by giving one dose (15 ml) of syrup of ipecac. If the patient does not vomit within 20 minutes, give a second dose. If syrup of ipecac is not available, give the patient one to two glasses of water and cause vomiting by inserting a finger down the throat. Repeat with water until vomit fluid is clear. The patient should be lying down with the head below the level of the feet. DO NOT TRY TO CAUSE VOMITING IF THE PATIENT IS UNCONSCIOUS OR IN A CONVULSIVE STATE.

Toxicological Information:

Decontamination and Disposal: Do not reuse empty drums. Return to drum reconditioners or destroy by crushing and burying in a safe place.

Limitations:

1. Do not incorporate into materials whose end use will result in prolonged direct skin contact e.g. life jackets, sleeping bags, sports equipment.

USE CLAIMS ACCEPTABLE FOR REGISTRATION IN CANADA

TEXTILES

resists decay, resists mildew, resists mold

COM 40% product #EC, SN

DIP, SPRAY, PAD OR BRUSH: Dilute, apply with conventional padding equipment, and dry fabric on cans or on a frame. Where exposure to weather or severe leaching is a factor, incorporate 5% of the finished weight of the fabric. For materials to be used under indoor conditions or in conjunction with a vinyl coating, 2.5% may be adequate. RATES AS ACTIVE: 10,000-20,000 ppm of finished weight of fabric.

Limitation (1)

LEATHER, TEXTILES, VINYLS

resists bacteria, resists mold

COM 95% product #SN

Apply at the rate of 1 to 1.5% of the total weight of the finished treated product. For items, such as tarpaulins, tents, and awnings, subject to extensive weathering, 1.5% accompanied by a compatabile water repellent is recommended. RATES AS ACTIVE: 9500-14,250 ppm of weight of finished treated product.

	Limitation (1)	57
		58
MIXTURES CONTAINING FATTY ACID (C_6-C_{20}) ESTERS OF PENTACHLOROPHENOL		59
1.	with dichlorophen (DPH)	60
TEXTILES		61
resists decay,	COM PCF: 37.6% DPH: 2.4% product #EC	62
resists mildew	Apply by a padding process followed by drying and curing.	63
	On cotton camping material of 200 to 300 grams per	64
	square meter unit area weight use 50 to 80 grams product per	65
	litre water. On cotton awning and packsack fabric 350 to	66
	400 grams per square metre unit area weight, use 50 to 60 grams	67
	of product per litre of water. On cotton sailcloth and	68
	turpaulin fabrics of more than 450 grams per square metre unit	69
	area weight use 45 to 50 grams product per litre water. The	70
	quantities quoted are based on a pick up of approximately 60%	71
	in application. Waterproofing may be applied in the same batch.	72
	RATES AS ACTIVE: PCF: 75.6 g DPH: 4.8 g to PCF: 169.2 g	73
	DPH: 10.8 g per square meter unit area weight.	74
	Limitation (1)	75
		76
2.	with pentachlorophenol plus related chlorophenols (PCP)	77
		78
CELLULOSIC MATERIALS		79
(LOOSE), FIRE HOSE, YARNS		80
resists bacteria,	COM PCF: 25% PCP: 0.5% product #EC	81
resists fungi	DIP, SPRAY, PAD OR BRUSH: Dilute as necessary with warm or cold	82
	water. Apply at levels of 4 to 8 per cent, according to the degree of	83
	protection required, or to meet the requirements of the British	84
	Standard 2087:1971. Apply to fire hose and textile piece goods	85
	by conventional impregnation techniques. Apply to yarns during	86
	winding or doubling by means of a furnishing roller. Loose	87
	fibre stock, wood fibre, and similar materials may be sprayed.	88
	Do not use concurrently with or before a shower-proofing or	89
	water-repellent treatment.	90
	RATES AS ACTIVE: 4 to 8% of fibre weight	91

TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980

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							
12,442	C	CHD		PQ-12 Liq Fung for Lumber & Timber	EC	CUQ 5.0	TCP 20.0
12,801	C	RHC		49-167 Tetrachlorophenol	SO	TCP 94	
KTC - potassium tetrachlorophenate							
16308	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
9,933	C	CHD		Permatox 100 Liquid Fungicide Conc.	SN	STC 22.82 PML 0.4	SMM 13.23
11,039	C	CHD		Chapco SSC Conc Liq Fung for Lumber & Timber	SN	STC 22.82	SMM 13.23
13,585	C	DIM		Diatox	SN	STC 24.2	
10,924	C	VAR		VW&R Guardsman Stain Control - Woodbrite 24	SN	SPC 7.68 BNS 2	STC 16.32
14,874	C	WAB		18-600 Woodsheath Cherry Brown 10.0IG	SU	STC 6.9	
PCP - pentachlorophenol plus related active chlorophenols							
15,407	D	BEG	BPR	Behr Wood Preserv No. 91	SN	PCP 5.0	
12,163	C	CHD		PQ-10 Liq Fung for Lumber & Timber	EC	CUQ 5.0 TCP 2.4	PCP 17.6
11,222	C	NAC		Fenocil Liq Weed Killer	EC	BBU 0.23 lb/gal	PCP 0.31 lb/gal
13,514	C	NAC		National Chemsearch HK-7 Liquid Weed Killer	EC	BBU 0.059 lb/gal	PCP 0.068 lb/gal
13,475	C	SON	BOB	Sta-Brite Liq Controls Sapstain & Mold	EC	TCM 15 TCP 1.2	PCP 8.8
12,319	C	VET	ARH	Mystox LSE Bacteriostatic & Fungistatic Additive	EC	PCF 25	PCP 0.5

[1]Registration Number

[2]Market (C = Commercial, D = Domestic)

[3]Registrant (see App. 10, Sect. 10.2.1)

[4]Agent (see App. 10, Sect. 10.2.3)

[5]Formulation (see App. 10, Sect. 10.2.4)

[6]Guarantee (% Active Ingredient, unless otherwise stated.
For code definitions see App. Sect. 10.2)

TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980 (Cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
5,639	D	WIL		Wilson's Soil Sterilizer	EC	DXB 0.25 PRM 2.0	PCP 2.0
12,534	C	DOO		Domtar Pentachlorophenol Oiled-indus	GR	PCP 96	
11,974	C	DOW		Dowicide EC-7 Antimicrobial	GR	PCP 88	TCP 12
11,994	C	DOW		Dowicide 7 Antimicrobial	GR	PCP 95	
12,610	C	DOW		Dowicide 7 Oiled for Control of Bacteria & Fung	GR	PCP 96	
12,864	C	RHC		RCL 49-162 Pentachlorophenol for Manufacturing Purposes Only	GR	PCP 96	
8,168	C	CHD		Pol-Nu Pak Ground-Line Pole Treat Bandage	PA	PCP 8.8	TCP 1.2
8,170	C	CHD		Pol-Nu Penta Preserv Grease for Ground-Line Treat	PA	PCP 8.8	TCP 1.2
8,654	C	CHD		Timpreg Pak Pol-Nu Type Preserv Grease	PA	PCP 8.8 CRT 15	TCP 1.2 SFL 15
8,656	C	CHD		Timpreg Pol-Nu Type Preserv Grease	PA	PCP 8.8 TCP 1.2	CRT 15 SFL 15
10,617	C	CHD		Timpreg B Pol-Nu Type Wood Preserv Grease	PA	PCP 8.8 BNA 15.5	TCP 1.2
12,038	C	CHD		Timpreg B (Special) Wood Preserv Grease	PA	PCP 8.8 CRT 15.5	TCP 1.2 BNA 15.50
13,618	C	STD		Stangard Penta Grease 10 Groundline Wood Preserv	PA	PCP 10	
15,144	C	TIR	BAO	Osmoband Wood Preservative Bandage	PA	CRT 15 SFL 20	PCP 8.8 TCP 1.2
11,782	C	VAR		Pole Preg Wood Preserv Grease	PA	PCP 10 BNA 15	CRT 15
10,633	C	SAJ		Sanitized Van Interior Aerosol	PP	PCP 0.1 MGK 1.67	PYR 0.5 PBU 1
7,580	C	ALS		Penta-Chem Conc Weed Preserv 10-1	SN	PCP 42.5	
7,635	C	BAP		Clear 36-105 Liq Wood Preserv	SN	PCP 5	
10,792	D	BEN		Moorewood Penta Wood Preserv Clear 456-00	SN	PCP 4.8	
8,103	D	CAO		Bulldog Grip Wood Preserv Clear	SN	PCP 4.8	
12,392	C	CAO		Bulldog Grip Wood Preserv Clear	SN	PCP 4.8	
15,110	C	CAT		BWK-98 Liquid Non-Selective Weed Killer & Soil Sterilant	SN	BBU 0.98 PCP 0.89	DXF 1.09
10,889	D	CBE		Mastercraft Clear Wood Preserv & Sealer	SN	PCP 2.85	
13,665	C	CEP		Penta-Mix Wood Preserv SN	SN	PCP 5	
3,267	C	CHD		Penta Preserv Conc 1 to 10 Wood Preserv 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
10,319	D	COP		Fedecor Wood Preserv Clear G-14	SN	PCP 8.8	TCP 1.2
14,594	C	COS		Copeland's Liquid Soil Sterilant	SN	DXF 1.09 PCP 0.89	BBU 0.98

[1]Registration Number

[2]Market (C = Commercial, D = Domestic)

[3]Registrant (see App. 10, Sect. 10.2.1)

[4]Agent (see App. 10, Sect. 10.2.3)

[5]Formulation (see App. 10, Sect. 10.2.4)

6Guarantee (% Active Ingredient, unless otherwise stated.
For code definitions see App. Sect. 10.2)

TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980 (Cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
15,521	C	COS		Copeland's Liquid Soil Sterilant	SN	BBU 0.98 PCP 0.89	DXB 0.72
9,961	D	CUB	CAX	Cuprinol Wood Preserv Stain Transcolour	SN	PCP 4.75	
15,365	C	DEE	COS	Deer Park Dee-Strict Liquid Soil Sterilant	SN	BBU 0.98 PCP 0.89	DXL 0.72
13,462	D	DES	BAY	Xyladecor	SN	PCP 4.8 DCA 0.6	LIN 0.38
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 Preserv for Field Cuts	SN	PCP 5	
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	
13,748	D	FEF		Fung Wood Preserv Green C4410	SN	PCP 4.75	
13,749	D	FEF		Fung Wood Preservative 10 to 1 Conc C-4464	SN	PCP 39.36	
15,658	D	FLC		Varapel No. 5000 Natural/Neutre	SN	PCP 3.84	
15,659	D	FLC		Varapel No. 5001 Hunter green/Vert chasseur	SN	PCP 3.84	
15,660	D	FLC		Varapel No. 5002 Cordova brown/Brun codoba	SN	PCP 3.84	
15,661	D	FLC		Varapel No. 5003 Charcoal/Charbon	SN	PCP 3.84	
15,662	D	FLC		Varapel No. 5004 Fawn/Fauve	SN	PCP 3.84	
15,663	D	FLC		Varapel No. 5005 Walnut/Noyer	SN	PCP 3.84	
15,664	D	FLC		Varapel No. 5006 Mahogany/Acajou	SN	PCP 3.84	
15,665	D	FLC		Varapel No. 5007 Redwood/Sequoia	SN	PCP 3.84	
15,666	D	FLC		Varapel No. 5008 Maple/Erable	SN	PCP 3.84	
15,036	D	GHC		Protox Clear (Clair)	SN	PCP 5	
15,278	C	GUC		Guardian Chemicals Dead and Gone Vegetation Eradicator	SN	BBU 1.47 PCP 0.89	DXF 0.72
14,881	D	HOH		Paintable Penta Clear Wood Preservative	SN	PCP 4.8	
9,110	D	HOS		Super Solignum 10-10 Clear Wood Preserv	SN	PCP 4.8	
12,510	D	HOS		Super Solignum Wood Preserv Stain 10-14 Walnut	SN	PCP 3.1	
12,512	D	HOS		Super Solignum Wood Preserv Stain 10-16 Teakwood	SN	PCP 3.1	
12,513	D	HOS		Super Solignum Wood Preserv Stain 10-15 Black	SN	PCP 3.1	

[1] Registration Number

[2] Market (C = Commercial, D = Domestic)

[3] Registrant (see App. 10, Sect. 10.2.1)

[4] Agent (see App. 10, Sect. 10.2.3)

[5] Formulation (see App. 10, Sect. 10.2.4)

[6] Guarantee (% Active Ingredient, unless otherwise stated.
For code definitions see App. Sect. 10.2)

TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980 (Cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
12,514	D	HOS		Super Solignum Wood Preserv Stain 10-200 Bungalo White	SN	PCP 3.1	
12,515	D	HOS		Super Solignum Wood Preserv Stain 10-68 Straw	SN	PCP 3.1	
12,516	D	HOS		Super Solignum Wood Preserv Stain 10-66 Drift Wood	SN	PCP 3.1	
12,517	D	HOS		Super Solignum Wood Preserv Stain 10-65 Grass Green	SN	PCP 3.1	
12,518	D	HOS		Super Solignum Wood Preserv Stain 10-63 Dark Brown	SN	PCP 3.1	
12,519	D	HOS		Super Solignum Wood Preserv Stain 10-62 Brunswick Green	SN	PCP 3.1	
12,520	D	HOS		Super Solignum Wood Preserv Stain 10-23 Mahogany	SN	PCP 3.1	
12,521	D	HOS		Super Solignum Wood Preserv Stain 10-22 Cedar	SN	PCP 3.1	
12,522	D	HOS		Super Solignum Wood Preserv Stain 10-21 Redwood	SN	PCP 3.1	
12,991	D	HOS		Solignum Patio Deck & Furniture Finish 10-76 Teakwood	SN	PCP 3.1	
12,992	D	HOS		Solignum Patio Deck & Furniture Finish 10-72 Cedar	SN	PCP 3.1	
12,993	D	HOS		Solignum Patio Deck & Furniture Finish 10-71 Redwood	SN	PCP 3.1	
11,762	C	HYN	MOO	006 Liq Weed Killer	SN	BBU 0.61	DXF 1.09 PCP 0.89
15,271	C	HYP	COS	Grim Reaper Liquid Soil Sterilant	SN	BBU 0.98 PCP 0.89	DXB 0.72
12,262	C	KEK		Norkem 600C Indus Herb	SN	BBU 0.61	PCP 0.89
6,948	D	LAT		Later's Pentachlorophenol SN Ready-to-use Wood Preserv	SN	PCP 5	
6,950	C	LAT		Later's Pentachlorophenol Wood Preserv 1-10 Liq Conc	SN	PCP 40	
10,320	D	LAV		Laurentide Paint Wood Presv Clear G-14	SN	PCP 4.8	
11,713	D	LEG		Rez Penta Wood Preserv Clear	SN	PCP 5	
11,714	D	LEG		Rez Penta Wood Preserv Green	SN	PCP 5	
10,317	D	MCS		Fedecor Preserv Pour Bois Liq Vert G-17	SN	PCP 4.8	
6,984	C	MOB		Pentanol A Penatrating Fung & Sealer Clear M-77	SN	PCP 4.75	
6,410	D	NNP		Tim-Ber-Lox Fung Wood Preserv Green 4410	SN	PCP 4.75	
9,623	C	NNP		Tim-Ber-Lox Fung Wood Preserv 10 to 1 Conc 4464	SN	PCP 39.36	
10,369	D	NNP		Tim-Ber-Lux Fungicided Wood Preservative Clear 4413	SN	PCP 4.75	
11,071	D	NOW		Tarcoate Pentasol-Green Preserv	SN	PCP 4.7	
5,339	D	OSD		Pentox Primer-Sealer Wood Preserv Clear	SN	PCP 4.75	
12,374	D	OSD		Pentox Penta Green Wood Preserv	SN	PCP 5	
13,635	D	OSD		Pentox 1+10 Penta Conc Wood Preserv	SN	PCP 40	
13,636	D	OSD		Pentox Wood Preserv Brown	SN	PCP 3.85	
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

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[4]Agent (see App. 10, Sect. 10.2.3)

[5]Formulation (see App. 10, Sect. 10.2.4)

[6]Guarantee (% Active Ingredient, unless otherwise stated.
For code definitions see App. Sect. 10.2)

TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980 (Cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
16,038	C	PYA	PYD	Pyramid Chemical's PC 551 Soil Sterilant	SN	BBU 0.98 DXB 0.72 PCP 0.89	
15,950	C	RCL		Roslyn Chemical Dura-strick	SN	BBU 0.98 DXB 0.72 PCP 0.89	
14,095	C	REB		Penta Preservative 1-10	SN	PCP 36.3	TCP 5
9,535	D	REC		Penta-Phenol Clear Paintable Wood Preserv & Primer-Sealer	SN	PCP 4.8	
11,836	D	ROK		Timber-Life Wood Preserver	SN	PCP 4.89	
8,227	D	ROR		ROZ-TOX Clear Wood Preserv & Sealer	SN	PCP 2.85	
12,250	D	SCP		Wood Preservitt Clear Paintable Penta Wood Preserv	SN	PCP 4.8	
7,615	D	SHW		Four Star Preserv-Sealer Clear/453	SN	PCP 4.8	
13,381	C	SHW		Four Star Preserv Sealer Clear/453	SN	PCP 4.8	
13,958	D	SMG		Permasan Wood Preservative Clear	SN	PCP 5	
14,415	D	SPP		Smart Paint Wood Preservative Clear 4550	SN	PCP 5.00	
14,416	D	SPP		Smart Paint Wood Preservative Green 4560	SN	PCP 5.00	
8,789	C	STD		Stangard Penta Wood Preserv Conc 1-10	SN	PCP 41	
8,791	C	STD		Stangard Paintable Penta Clear Wood Preserv	SN	PCP 5	
8,799	C	STD		Stangard Penta WR Wood Preserv Conc 1-4	SN	PCP 21	
8,801	C	STD		Stangard Penta WR Water Repellent Wood Preserv	SN	PCP 5	
11,774	C	STD		Stangard Penta Green Wood Preserv	SN	PCP 5	
13,008	D	STD		Stangard Paintable Penta Clear Wood Preserv	SN	PCP 5	
13,010	D	STD		Stangard Penta WR Water Repellent Wood Preserv	SN	PCP 5	
13,091	D	STD		Stangard Penta Green Wood Preserv	SN	PCP 5	
15,987	C	STN		Horntox Clear Wood Preserv	SN	PCP 0.06 ZNN 2.0	
15,988	C	STN		Horntox Green Wood Preserv	SN	CUN 2.0 PCP 0.06	
3,608	C	TEI		Nevarot Water Repellent Wood Preserv	SN	PCP 4.75	
15,143	C	TIR	BAO	Pole Topper Fluid Wood Preserv	SN	PCP 8.8	TCP 1.2
14,212	D	TOL		Color Your World, Pentachlorophenol Wood Preversative	SN	PCP 4.8	
15,244	C	TRO		Trojan Chemicals TRL-08 Soil Sterilant	SN	BBU 0.98 PCP 0.89	DXF 0.72
10,925	C	VAR		Guardsman Pentapreserv Conc 1-10	SN	PCP 42.5	
12,303	C	VAR		Guardsman Penta Preservative	SN	PCP 4.25	
15,976	C	WAB		18-528 Woodsheath Seabrite - 10.01G	SU	PCP 14.2	
14,204	D	WEW		Woodlife Liq Water Repellent Wood Preserv	SN	PCP 5	

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[5]Formulation (see App. 10, Sect. 10.2.4)

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TABLE A10-2 PRODUCTS CONTAINING CHLOROPHENOLS REGISTERED UNDER THE PEST CONTROL PRODUCTS ACT AS OF APRIL 1, 1980 (Cont'd)

REGN[1]	MKT[2]	REGT[3]	AGT[4]	Product Name	Form[5]	Guarantee[6]	Guarantee[6]
14,205	C	WEW		Woodlife Liq Water Repellent Wood Preserv	SN	PCP 5	
14,206	C	WEW		Woodlife 3:1 Concentrate Wood Preservative	SN	PCP 17.9	
SPC - sodium pentachlorophenate plus sodium salts of other chlorophenols							
13,297	C	PBA		Slimicide Formula Y-100 Pellets	PE	SPC 90	
8,146	C	CHD		Chapman Permatox 10-S	SG	BNS 57	SPC 36
11,992	C	DOW		Dowicide G-ST Antimicrobial	SG	SPC 90	SHO 1.5
12,334	C	MOG		Mogul A-421 Microbiocide	SG	SPC 90	
13,483	C	BEZ		Betz Slimicide A-9	SN	SPC 27.6	STD 9.1
						SDD 4.0	QAC 5.0
						IAL 10	
13,601	C	BEZ		Betz Slime-Trol RX-17	SN	IAL 10	STD 9.1
						SDD 4.0	SPC 27.6
						QAC 5.0	
15,574	C	CHD		Napclor-S Antimicrobial	GR	SPC 90	
12,867	C	DEC		Dearcide 712 Liq Cooling Water Microbistat	SN	SPC 32	STD 8.0
12,335	C	MOG		Mogul AG-420 Microbiocide	SN	SPC 10.8	
14,076	C	SAN		Sanfax Pinefax Liquid Disinfectant	SN	IAL 13.5	POI 5
						TYT 2.66	SPC 0.55
						SBC 1.1	SBD 2.81
						SXS 2.8	
12,789	C	SAT	SAJ	Sanitized Brand PWS	SN	SPC 20	
13,073	C	SAT	SAJ	Sanitized Brand Bacteriostat SP		SN	SPC 15
13,995	C	SAT	SAJ	Sanitized Brand SPI	SN	SPC 10	
13,502	C	VIT	VIR	Virginia Algae-Cide No. 2	TA	SPC 90	

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[5]Formulation (see App. 10, Sect. 10.2.4)

[6]Guarantee (% Active Ingredient, unless otherwise stated.
For code definitions see App. Sect. 10.2)

10.1 Chlorophenol Products Registrants

Code	Registrant Name and Address
ALC	Alchem Ltd., P.O. Box 5002, 1055 Truman St., Burlington, Ont. L7R 3Y9
ALS	Allied Chemical Services Ltd., 5507 First St. S.E., Calgary, Alta. T2H 1H9
BAP	Bapco Paint Ltd., P.O. Box 9011, Surrey, B.C. V3T 4Y4
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
CAT	Cantol Ltd., 199 Steelcase Rd., P.O. Box 2400, Don Mills, Ontario M3C 2T9
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
COP	Co-operative Federee de Quebec, 1055 Marche Central, Montreal, Que. H4N 1K3
COS	Copeland Laboratories Ltd., 41 Racine Rd., Rexdale, Ont. M9W 2Z6
CUB	Cuprinol Ltd., Adderwell, Frome, Somerset, England
DEC	Dearborn Chemicals, 3451 Erindale Station Rd., Mississauga, Ont.
DEE	Deer Park Chemical, 110 Green Meadow Dr., Deer Park, N.Y. 11729, U.S.A.
DES	Desowag-Bayer Holzschutz GMBH, 4 Düsseldorf 30, Roßstraße 76, Germany.
DIM	Diachem ot 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
FEF	Federated Co-operatives Ltd., 401-22nd St. E., P.O. Box 1050, Saskatoon, Sask. S7K 3M9
FLC	Fletco Coatings Ltd., 4260 Vanguard Rd., Richmond, B.C. V6X 2P5
GHC	Gibson - Homans of Canada Ltd., 101 de la Berre, Boucherville, Que. J4B 2X6
GUC	Guardian Chemicals (Division Skyline Products Ltd.) P.O. Box 3029, Fort Saskatchewan, Alberta T8L 2T1
HOH	Home Hardware Stores Ltd., 34 Henry St. W., St. Jacobs, Ont. N0B 2N0
HOS	House of Sturgeon (National) Ltd., 200 Norelco Dr., Weston, Ont. M9L 1S4
HYN	Hysan Corp., 910 W. 38 St., Chicago, Ill. 60609, U.S.A.
HYP	Hyde Park Chemical, 170 Dupont St., Plainview, N.Y. 11803 U.S.A.
KEK	Kem Manufacturing Canada Ltd., 6660 Campobello Road, Mississauga, Ontario. L5M 2C2
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
MCS	Co-op Atlantic, P.O. Box 750, 123 rue Halifax St., Moncton, N.B. E1C 8N5
MOB	Mobil Chemical Canada Ltd., 645 Coronation Dr., West Hill, Ont. M1E 4R6
MOG	The Mogul Corp. of Canada Ltd., 8400 Cote de Liesse, Ville St-Laurent, Montreal, Quebec. H4T 1G7
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
NOW	Northern Wood Preservers Ltd., Box 990, Thunder Bay, Ont. P7C 4X0

OSD	Osmose Wood Preserving Co. of Canada Ltd., 1080 Pratt Ave., Montreal, Que. H2V 2V2
PBA	Perolin-Bird Archer Ltd., 110-2nd St., Cobourg, Ont. K9A 4M2
POS	Pole Sprayers of Canada Ltd., 980 Ellicott Street, Buffalo, N.Y. 14209 U.S.A.
PYA	Pyramid Chemical Corp., 20 Jerusalem Ave., Hickville, N.Y. U.S.A.
RCL	Roslyn Chemical Ltd., P.O. Box 13066, Toronto, Ont.
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
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.
SCP	St-Clair Paint & Wallpaper Co. Ltd., 38 Dufflaw Road, Toronto, Ontario. M6A 2W1
SHW	The Sherwin-Williams Co. of Canada Ltd., 2875 Centre St., Montreal, Que. H3K 1K4
SMG	Smith Barregar Ltd., 115 West Third Ave., Vancouver, B.C. V5Y 1E7
SON	Sonford Products Corporation, Southern Division, P.O. Box 5570, Jackson, Mississippi 39208, U.S.A.
SPP	S. P. Paint Factory, 135 Parmount Road, Winnipeg, Manitoba R2X 2W6
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.

TOL	Tonecraft Ltd., 10 Carson St. Toronto, Ont. M8W 3R5
TRO	Trojan Chemicals Div. of Valley Camp Ltd., 41 Racine Road, Rexdale, Ont. H9W 2Z6
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.
VIT	Virginia Chemicals Inc., 3340 West Norfolk Rd., Portsmouth, Virginia 23703, 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
WIL	Wilson Laboratories Ltd., Brock and Hatt Sts., Dundas, Ont. L9H 2H9

10.2 Chlorophenol Products Active Ingredients

Code	Active Ingredient
BBU	Bromacil present in free form, as dimethylamine salt, or as lithium salt
BCP	o-benzyl-p-chlorophenol
BNA	borax, anhydrous
BNS	borax
BTU	bis (tri-n-butyltin) oxide
CRT	creosote
CUN	Copper as elemental, present as copper naphthenate
CUQ	copper - 8 - quinolinolate
DAP	dehydroabietylamine pentachlorophenate
DCA	dichlofluanid
DNP	dinitrophenol
DXB	2,4-D present as dimethylamine salt
DXF	2, 4-D present as isooctyl esters
IAL	isopropyl alcohol
KTC	potassium tetrachlorophenate

KDC	potassium dichromate
LIN	gamma - BHC from lindane
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
PRM	2-methoxy-4, 6-bis (isopropylamino)-s-triazine
PYR	pyrethrins
PAC	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
TCM	2-(thiocyanomethyl thio) benzothiazole

TCP	tetrachlorophenol
TNM	trisodium nitrilotriacetate monohydrate
TPP	p-tert amylphenol
TYT	tetra sodium ethylenediaminetetra acetate
ZNN	Zinc as elemental, present as zinc naphthenate

10.3 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
BAY	Bayer Canada Inc., 7600 Trans Canada Hgwy. Pte. Claire, Que. H9R 1C4
BOB	Borden Brokers Ltd., P.O. Box 2168, Vancouver, B.C. V6B 3V3
BPR	Behr Process of Canada, 4624 - 11th St. N.E., Calgary, Alta. T2E 2W7
CAX	Hoechst Canada Inc., 100 Tempo Ave., Willowdale, Ont. M2H 2N8
COS	Copeland Laboratories Ltd., 41 Racine Road, Rexdale, Ont. M9W 2Z6
FIT	Art W. Fish, P.O. Box 88, Bonnie Dr. Route 1, Winfield, B.C. V0H 2C0
MOO	Moon River Holdings, 69 Bloor St. E., Toronto, Ont. M4V 1E4
PYD	Pyramid Chemical Corp., Markham, Ont. L3R 2Y2
SAJ	Sanitized Process Canada Ltd., Suite 1700, 2200 Younge St., Toronto, Ont. M4S 2C6
TAY	Tom Taylor, Co. Ltd., 136 Adelaide St., Toronto, Ont. M5C 1L6
VIR	Peter Eustis, Vir-Chem of Canada Ltd., 1440 Tenth St., E. Cornwall, Ont.

10.4 Formulations - Codes

Code	Chlorinated Phenol Products Formulations
EC	emulsifiable concentrate
GR	granular
PA	paste

PE	pellet
PP	pressurized product
SG	soluble granule
SN	solution
SO	solid
SU	suspension
TA	tablet
WP	wettable powder

APPENDIX 11

APPENDIX 11

11 REFERENCES

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