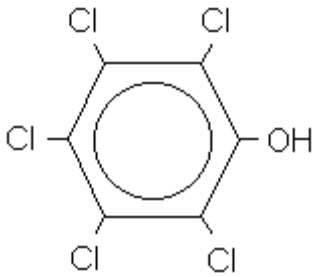
Water Quality

Ambient Water Quality Guidelines for Chlorophenols

First Update



2,3,4,5,6-PCP

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Abstract

This report is one of a series which establishes ambient water quality guidelines for British Columbia. The guidelines represent safe conditions or levels of a variable which have province-wide application and are set to protect various water uses. This report sets guidelines for the chlorophenols, chlorinated monocyclic aromatic hydrocarbons, to protect drinking water, fresh-water and marine aquatic life, recreational waters, food processing industries and wildlife and livestock drinking water. Guidelines were

not set for crop irrigation or other industries due to the lack of suitable data on the effects of chlorophenols for these uses. Each chapter where guidelines are proposed has a rationale section immediately following the proposed guidelines. The guidelines are summarized in three tables, 1.1.1, 1.1.2 and 1.1.3, at the back of this report. CCREM (CCME) has not set guidelines for all the water uses and all the chlorophenols for which we have set guidelines.

No data were found on the effects of chlorophenols on industrial processes. In the manufacture of some industrial products such as photographic chemicals, paints, oils, textiles, glues, starches, cellulosic wood fillers, rubber, protein-based products and shampoos, and in industrial cooling and process waters in mills, chlorophenols are deliberately added to prevent growth of microorganisms. The only place where chlorophenols would be a problem is in water actually incorporated into a food product, or used to wash and process food products. Here, due to taste and odor problems, water of drinking quality is required and the Drinking Water Guidelines are recommended. There are virtually no data on the effects of chlorophenols in irrigation water and no guidelines are set. There is a short section in Chapter 7.2 discussing effects of chlorophenols on seed germination and cytotoxicity in plants.

Only 8 of the 19 chlorophenols are in commercial use; the rest are produced incidentally when organic material is chlorinated. For most organisms there are generally abundant data on the effects of PCP, pentachlorophenol, but few on the effects of other congeners, therefore the ratios of the toxicities of each chlorophenol to PCP, as determined in several experiments, were applied to the best PCP data to determine guidelines for the other congeners. Chlorophenols are notorious for causing taste and odor problems in water at levels below those which are toxic. The half-life of most chlorophenols in nature is short, rarely as long as a month. Once input to the environment stops, levels will drop rapidly; bacterial breakdown of chlorophenols occurs quite readily.

Chlorophenols were used world-wide as broad spectrum biocides; residues and breakdown products were ubiquitous in air, water, sediment, and organisms. Their major use, particularly PCP and TTCPs, has been as an anti-sapstain fungicide in the cut lumber industry, but this use no longer occurs in North America. PCP is now used only to pressure and thermal treat heavy timbers and poles for outdoor construction. Chlorophenols are also used as antiseptics and organic feedstocks for pesticide manufacture; many commercial products contain chlorophenols, notably packaging materials in which they are used as preservatives. The chlorophenols were generally made by chlorinating phenols and by hydrolysis of chlorobenzenes; dioxins were common byproducts of their manufacture, especially in high temperature hydrolysis processes. This dioxin contamination sometimes made it difficult to decide whether an observed effect was due to the dioxin contaminant or to the chlorophenol. Presently PCP used in North America is made by chlorinating phenol under controlled conditions which preclude the production of measurable 2,3,7,8-TCDD in the current product.

The chlorination of wastewater with high organic loads, such as sewage, leads to low levels of chlorophenol production, but this practice is widespread. Major sources of chlorophenol input to the environment included wood treatment facilities and pulp and paper mills. Fly ash from incinerators, power stations, fireplaces, and slash and forest fires also contribute to the widespread distribution of chlorophenols in the environment.

Once released to the environment physical, chemical and biological processes break down chlorophenols, ultimately to carbon dioxide, water and chloride. The half-lives of the chlorophenols range from hours to months, depending upon the isomer and the conditions. Photodegradation is only important in shallow water under high irradiation levels; hydrolysis and oxidation are relatively unimportant in natural chlorophenol degradation; evaporation and volatilization are only important in

shallow water subject to vigorous mixing; adsorption is a major process and most chlorophenols introduced into the environment will eventually be found adsorbed to organic sediments.

Effluents and spills from wood treatment facilities and pulp and paper mills were responsible for a number of fish kills. Fish, immersed in their uptake medium, bioconcentrate chlorophenols up to 1000 times, for whole body loads, in spite of very efficient conjugation and elimination processes. The half-life of chlorophenols in fish is less than 1 day, thus the existence of high levels in fish tissues is indicative of chronic, on-going or current exposure. There is an initial lag period while the metabolic processes of the fish adapt to conjugating and excreting chlorophenols; once adapted, the depuration half-lives are short, generally less than 24 hours.

Chlorophenols affect the common mitochondrial respiration and energy storage processes in all higher plants and animals . The effect is to waste the energy stored in food which is thus not available for growth and maintenance, even though respiration rates rise. This terminal respiration process is essential and universal; results observed in one species are applicable to others, and death will result if it is prevented or severely disrupted. Toxicity levels in different organisms are not identical because of the relative efficiencies of uptake, transport, and elimination of chlorophenols by different organisms. Chlorophenols are immunotoxic, fetotoxic, and embryotoxic, but not neurotoxic or teratogenic. Mutagenicity and carcinogenicity assays on chlorophenols, even at the very high doses used in some tests, have not given definitive answers.

Toxicity to aquatic life, and the generation of an unpleasant taste in fish and shellfish living in chlorophenol contaminated waters, are the effects which are manifested at the lowest chlorophenol concentrations; these effects occur in the sub-µg/L concentration range. In mammals, chlorophenols are not accumulated to high levels in fat due to very rapid excretion as glucuronide conjugates, thus keeping bioconcentration factors low. In almost all mammals the dose of PCP needed to kill one half of the test animals is fairly uniform at about 150 µg/g of body weight.

Microorganisms can adapt their metabolic processes to use virtually any source of carbon, including chlorophenols, for growth. The evidence for fungal and bacterial degradation of chlorophenols in nature is widespread. If the organisms have never been exposed to chlorinated organics, there will be an initial adaptation period of several weeks to a month. Once the adaptive phase is over and a large microbial population has been established, breakdown of the chlorophenols is rapid; subsequent additions of chlorophenols to the environment are quickly degraded, if the concentrations are not excessive. Chlorophenols with chlorines in the ortho or para positions (2, 4 and 6) are less toxic and more readily degraded than those with chlorines in the meta (3 and 5) positions.

Recommended Water Quality Guidelines

HUMAN DRINKING WATER

We recommend adoption of the existing Canadian Drinking Water Quality Guidelines for Chlorophenols, which have been adopted by the BC Ministry of Health, with the addition of a monochlorophenol guideline of **0.1 µg/L**. The existing Canadian Guidelines specify certain specific isomers and omit any mention of others. We have set aesthetic guidelines based on the total concentration of all the isomers for each group of chlorophenols; the toxicity guidelines are specific for certain congeners in each isomer group.

Aesthetic Guidelines (taste and odour)

In raw human drinking water chlorophenols should not exceed the following:

0.1 µg/L monochlorophenols (MCPs)
0.3 µg/L dichlorophenols (DCPs)
2 µg/L trichlorophenols (TCPs)
1 µg/L tetrachlorophenols (TTCPs)
30 µg/L pentachlorophenol (PCP)

Toxicity Guidelines

In raw human drinking water chlorophenols should not exceed the following:

900 µg/L 2,4-dichlorophenol
5 µg/L 2,4,6-trichlorophenol
100 µg/L 2,3,4,6-tetrachlorophenol
60 µg/L pentachlorophenol

LIVESTOCK AND WILDLIFE

Aesthetic Guidelines

The recommended guidelines based on organoleptic effects are the same as the human drinking water aesthetic guidelines.

Toxicity Guidelines

The following guidelines, based on toxicity calculations, are recommended. While not toxic they may prove unpalatable to some species and thus restrict water intake or force animals to search for alternate sources of drinking water. These toxicity based guidelines may be appropriate under some conditions but generally the human drinking water guidelines are recommended. These values assume no other source of chlorophenols in the diet or in the inhaled air.
In livestock and wildlife drinking water, chlorophenols should not exceed the following levels:

For lactating animals under high temperatures and high water intake rates of up to 200mL/kg:

185 mg/L monochlorophenols (MCPs)
46 mg/L dichlorophenols (DCPs)
21 mg/L trichlorophenols (TCPs)
41 mg/L tetrachlorophenols (TTCPs)
17.5 mg/L pentachlorophenol (PCP)

For non-lactating animals under normal temperatures and water intake rates of 20 ml/kg:

1854 mg/L monochlorophenols (MCPs)
460 mg/L dichlorophenols (DCPs)
210 mg/L trichlorophenols (TCPs)
410 mg/L tetrachlorophenols (TTCPs)
175 mg/L pentachlorophenol (PCP)

AQUATIC LIFE

The level of chlorophenols in the water in which fish live, should not exceed the guidelines given below.

0.1 µg/L monochlorophenols
0.2 µg/L dichlorophenols

Toxicity Guidelines (to protect aquatic life)

The recommended toxicity guidelines for the chlorophenols in Table 1.1.2 are in µg/L, calculated for 10C. The temperature conversion factor is 2 for every 10C change in temperature (the value would be multiplied by 2 at 0C and by 0.5 at 20C). The change in the guidelines caused by applying this factor would be small for the normal ambient temperatures found in British Columbia.

Correction for different pH levels is more complex; guidelines for pH values in the range pH 5.7 to pH 9.2 were calculated for the chlorophenols and are found in Table 1.1.2. The guidelines for pentachlorophenol are final since they are based on good primary data. The guidelines for the other chlorophenols are based on a ratio with PCP and these should be considered interim guidelines until properly controlled experiments can provide a unified temperature and pH-dependent regression equation for each chlorophenol. The levels of chlorophenols in water containing aquatic life should not exceed the guidelines given below. These are calculated in µg/L, at 10ºC, from pH 5.7 to pH 9.2.

INTERIM AQUATIC LIFE TOXICITY GUIDELINES* FOR CHLOROPHENOLS

(calculated in µg/L, at 10°C*, from pH 5.7 to pH 9.2)

Chlorophenol Congeners	pH 5.7	pH 6.2	pH 6.7	pH 7.2	pH 7.7	pH 8.2	pH 8.7	pH 9.2
2-MCP	3.9	6.4	11	17	29	48	79	130
3-MCP	3.4	5.6	9.3	15	25	42	70	115
4-MCP	1.7	2.9	4.8	7.8	13	22	36	59
2,3-DCP	1.1	1.8	3.1	5.1	8.3	14	23	38
2,4-DCP	0.6	1.0	1.6	2.6	4.3	7.2	12	20

2,5-DCP	0.5	0.8	1.4	2.3	3.7	6.2	10	17
2,6-DCP	2.0	3.3	5.5	9.1	15	25	41	68
3,4-DCP	0.6	1.0	1.6	2.7	4.4	7.4	12	20
3,5-DCP	0.5	0.7	1.2	2.0	3.4	5.6	9.2	15
2,3,4-TCP	0.5	0.8	1.3	2.2	3.6	6.0	9.9	16
2,3,5-TCP	0.5	0.8	1.3	2.2	3.7	6.1	10	17
2,3,6-TCP	1.6	2.6	4.4	7.2	12	20	33	54
2,4,5-TCP	0.5	0.7	1.2	2.0	3.3	5.6	9.2	15
2,4,6-TCP	1.2	1.9	3.2	5.3	8.8	15	24	40
3,4,5-TCP	0.2	0.3	0.5	0.9	1.4	2.4	3.9	6.4
2,3,4,5-TTCP	0.4	0.6	1.0	1.7	2.8	4.7	7.8	13
2,3,4,6-TTCP	1.1	1.8	2.9	4.9	8.0	13	22	36
2,3,5,6-TTCP	0.5	0.8	1.3	2.2	3.6	6.1	10	17
2,3,4,5,6-PCP	0.2	0.3	0.5	0.7	1.2	2.0	3.4	5.5

multiply the table values by 2 at 0°C and by 0.5 at 20°C

RECREATION

General

There are no data documenting the effects of chlorophenols on recreational uses of water. Human taste and odor thresholds for some chlorophenols in water are available, as are taste thresholds for some chlorophenols in fish meat. No published taste thresholds for crustacean or mollusc meat were found for the chlorophenols.

Primary-Contact Recreation

Swimming involves contact of the face with the water; taste and odor thresholds for the chlorophenols should be met in water used for swimming. These thresholds are the critical factors determining drinking water aesthetic guidelines, and should give adequate protection to waters used for swimming.

Water used for Primary-Contact Recreation should meet the Drinking Water Aesthetic Guidelines.

Aesthetics

For distant scenic and vista uses, no guidelines are necessary; for proximal uses the odor thresholds in water are appropriate. These vary with water temperature and are a function of the molecular weight and volatility of the chlorophenol. The more chlorine substituents present, the lower the volatility and higher the threshold concentration for odor detection.

Water designated for Aesthetic use should not exceed the following.

0.3 µg/L MCPs or DCPs
11 µg/L TCPs
600 µg/L TTCP
860 µg/L PCP

INDUSTRIAL

No data were found documenting the effects of chlorophenols on industrial uses of water. Due to taste and odor concerns, the food and beverage industries should use the drinking water aesthetic guidelines, and the aquaculture industry should meet the aquatic life toxicity guidelines. A few other industries with a need for very high quality water would likely have to use point-of-use treatment to keep chlorophenols below acceptable levels, if the local supply was not adequate.

Irrigation

No data were found documenting the effects of chlorophenols on irrigation uses of water. Terrestrial plants are less sensitive to chlorophenols than are aquatic organisms; any effects found were in the mg/L range. Water suitable for aquatic life or drinking should therefore also be suitable for irrigation. No guidelines were set for this use.

ABBREVIATIONS AND ACRONYMS

A - year, per annum
ACQUIRE - a computerized data base; EPA, Office of Toxic Substances.

ADP - adenosine diphosphate; used in the storage and transfer of energy by organisms.
ATP - adenosine triphosphate; used in the storage and transfer of energy by organisms.
BCF - bioconcentration factor.
CAS - Chemical Abstracting Service.
CB - constant flow bioassay. There is constant water exchange in the test system with metered pollutant input to maintain a constant pollutant concentration. Also called a flow through bioassay, FlTh.
DO - dissolved oxygen level in the water.
DI - de-ionized (distilled) water, very low ion and hardness content.
EC - effective concentration, a concentration which has a noticeable effect on the organism under test.
ECx - concentration of a substance causing a noticeable effect in X% of the test organisms; X is often but not always 50.
ENVIROFATE - a computerized data base, EPA, Office of Toxic Substances.
EtOH - ethanol, ethyl alcohol, C2H5OH.
F - female
FO - fuel oil used as a solvent for a toxicant.
FS - field study, conducted in natural settings, not in a laboratory, subject to uncontrolled variables.
FT - flow through bioassay. There is a constant water exchange in the test system with metered pollutant input to maintain a constant pollutant concentration. Also called a constant flow bioassay, CB.
FW - fresh water, as opposed to marine or estuarine.
h - hour.
HNOAEL - highest no observable adverse effect level. The highest concentration of a pollutant at which no harmful effects are noted.
ICx - the aqueous concentration of a pollutant which causes immobility in X% of the test organisms; X is usually, but not always, 50.
IPR - intraperitoneal injection of the test compound into the organism.
Ko/c - the proportion of a chemical which dissolves in the non-polar organic solvent, octanol, as opposed to the proportion which adsorbs to carbon. This is an estimate of the relative partitioning of the chemical between the water and the sediment.
Ko/w - the proportion of a chemical which dissolves in the non-polar organic solvent, octanol, as opposed to the proportion which dissolves in the polar solvent, water. This is an estimate of the relative fat solubility of the chemical and thus its likelihood of being taken up from the environment by an organism.
LCx - the aqueous concentration of a pollutant which is fatal to X% of the test organisms; X is usually, but not always, 50.
LDx - the dose of a pollutant given to test organisms which causes death in X% of the test organisms; X is usually, but not always, 50.
LOAEL - the lowest observable adverse effect level. The lowest concentration of a pollutant which causes an observable harmful effect to a test organism.
LOG-P - a computerized data base, Pomona College, Medicinal Chemistry Project.
LS - laboratory study under controlled conditions.
LTC - lethal threshold concentration; the concentration of a pollutant at which the first deaths of test organisms begin to occur.
M - male
MAC - the maximum acceptable concentration of a toxicant.
MATC - the maximum acceptable toxicant concentration.
MDC - minimum detectable concentration.
mD - minimum dose required to effect a response in the test organism.
min. dose - minimum dose required to effect a response in the test organism.
min - minute
MISA - the Municipal-Industrial Strategy for Abatement in Ontario.

mon - month
NAT - natural water used in a laboratory experiment as opposed to DI water or tap water.
NOAEL - the no observed adverse effect level for a pollutant; no test organisms showed any observable harmful reactions.
NOLEL - the no lethal effects level; no test organisms died at this concentration of pollutant.
OO - olive oil used as a solvent for the toxicant.
ORAL - the dose of toxicant was given by mouth.
pKa - acid dissociation constant; the ratio of ionized to un-ionized molecules of a compound in solution, at a given pH.
PNO - pine oil used as a solvent for the toxicant.
PO - peanut oil used as a solvent for the toxicant.
PPG - propylene glycol used as a solvent for the toxicant.
PROPERTIES - a computerized data base from Montana State University.
QSAR - Quantitative Structure-Activity Relationships
RTECS - Registry of Toxic Effects of Chemical Substances, U. S. National Institute for Occupational Safety and Health.
RSB - renewal static bioassay; periodic replenishment of water or pollutant over the course of the experiment.
SB - static bioassay; there is no replenishment of water or pollutant over the course of the experiment.
SCU - subcutaneous injection of the toxicant into the test organism.
SD - the Sprague-Dawley strain of rats used in toxicity experiments.
Sh - the Sherman strain of rats used in toxicity experiments.
Sp. - species.
SW - marine or salty water; not fresh.
TLm - lethal threshold median; level at which one half of the test organisms are dead. See also LC and LD.
w - week
96-h LC50 - This is the most commonly used expression of the effects of a toxicant. It means that 50% of the test organisms died after being subjected to the given concentration of toxicant for a continuous period of 96 hours.

DEFINITIONS

Acute toxicity - a lethal or sublethal effect on the test organism which occurs in a short time; usually no more than 96 hours for large long-lived organisms, but in a much shorter time for smaller ephemeral organisms.
Adenoma - a benign (non-cancerous) tumor of glandular origin.
Alkalinity - the quantitative capacity of water, expressed as mg/L of $CaCO_3$, to neutralize a strong acid to a specified pH.
Bioaccumulation - the taking up of a substance from the environment by an organism either in the food or directly from the water via the gills or the skin.
Bioconcentration - the ability of an organism to take up or bioaccumulate a substance to concentrations in the body much higher than the concentrations in the environment.
Biomagnification - the process whereby the concentration of a substance gets progressively higher in

the bodies of organisms further up the food chain at higher trophic levels.
Carcinogenicity - the ability of a substance to cause cancers in organisms.
Carcinoma - malignant (cancerous) tumor originating in epithelial (membranous lining of cavities in the body) tissue.
Chloracne - an acne-like skin eruption caused by chlorinated organic compounds.
Chronic - effects which occur over a long period of time. The test organism is not usually killed but the species may be eliminated in the long-run if the chronic effects are on reproductive success or offspring viability.
Cytology - study of all aspects of the biology of cells or effects at the cellular level of organization.
Dermal - application or absorption through the skin.
Diaphoresis - very profuse perspiration which may be artificially induced.
Ecdysis - the stage in crustacean life-cycles when they have shed their old carapace (shell) and the new one has not yet hardened or become relatively impervious.
Edema - a swelling due to accumulation of blood serum in connective tissue or a cavity in the body.
Embryotoxicity - having toxic effects on the embryo of organisms, generally at the early stages of development after fertilization but before differentiation into a fetus.
Epithelial - living tissues of the gut, respiratory tract or the outer skin; tissues generally in contact with toxicants, often secretory or excretory.
Eukaryotic - a cell with a large well-defined membrane-bound nucleus and other membrane- bound components such as mitochondria (extra nuclear cellular organelles producing energy by respiration) and chloroplasts (extra nuclear cellular organelles which carry out photosynthesis and store energy as starch). Evolutionarily advanced over prokaryotic (primitive) cells and found in all multicellular organisms.
Fetotoxicity - having toxic effects on the fetus of organisms, generally during the later stages of development, after differentiation and up to birth.
Hardness - the total concentration of calcium and magnesium ions, expressed as mg/L of $CaCO_3$.
Homeotherms - warm-blooded animals; those that have internal metabolic control of their own body temperatures.
Hyperplasia - an abnormal or unusual increase in the cells of a tissue.
Immunotoxicity - having toxic effects on the immune system of an organism and affecting its ability to protect itself against diseases.
Intraperitoneal - injected into the body cavity.
Intravenous - given by injection into the blood stream.
Leucocytosis - an increase in the number of white blood cells.
Mitosis - nuclear division of the somatic (body as opposed to reproductive) cells of an organism.
Monocytosis -an increase in the large, phagocytic (engulfing and consuming debris and foreign matter) white blood cells, such as leukocytes.
Mutagenicity -having the ability to increase the frequency or extent of mutations (inheritable changes) in the genes.
Neotenous - animals (often amphibians) which become sexually mature in the larval stages of growth.
Neurotoxicity - having the ability to cause an adverse effect on the nervous system of an organism, affecting nerve impulse transmission or brain function.
Oral - given by mouth.
Papilloma - a benign (non-cancerous) tumor or wart due to an overgrowth of epithelial tissue on papillae (small nipple-like protuberances) of vascular connective tissues; usually in the skin.
Peritoneum -the membrane lining the abdominal cavity of a mammal.
Poikilotherms - cold-blooded animals; those that do not have automatic internal control of their own body temperatures.
Porphyria - a pathological condition characterized by abnormal porphyrin (organic compounds with a central metal atom and 4 pyrrole rings like chlorophyll or hemoglobin) metabolism, excretion of excess

porphyrin in urine and extreme sensitivity to light.
Somatic - the body cells of an organism, as opposed to the reproductive cells.
Subcutaneous - injected under the skin
Teratogenicity - having the ability to cause developmental malformations or deviations from the normal, in the growth and differentiation of embryos and fetuses.

Water Quality

Ambient Water Quality Guidelines for Chlorophenols

1. INTRODUCTION

1.1 Preamble

BC Environment (now called Water, Land and Air Protection) is developing province-wide ambient water quality guidelines for variables that are important in the surface waters of British Columbia. This work has the following goal:

- to provide guidelines for the evaluation of data on water, sediment, and biota;

The definition adopted for guideline is: "A maximum and/or a minimum value for a physical, chemical or biological characteristic of water, sediment or biota, which should not be exceeded to prevent specified detrimental effects from occurring to a water use, including aquatic life, under specified environmental conditions."

The guidelines are applied province-wide, but are use-specific, and are being developed for the following water uses:

- Drinking water
- Aquatic life
- Wildlife
- Agriculture (livestock watering and irrigation)
- Recreation and aesthetics

The guidelines apply to the ambient raw water source before it is diverted or treated for domestic use. The Ministry of Health regulates the quality of water for domestic use after it is treated and delivered by a water purveyor. Guidelines relating to public health at bathing beaches will be the same as those used by the Ministry of Health which regulates their use.

The guidelines are set after considering the scientific literature, guidelines from other jurisdictions, and general conditions in British Columbia. The scientific literature gives information on the effects of toxicants on various life forms. This information is not always conclusive because it is usually based on laboratory work which, at best, only approximates actual field conditions. To compensate for this uncertainty, guidelines have built-in safety factors which are conservative but reflect natural background conditions in the province.

The guidelines will be subject to review and revision as new information becomes available, existing data is proven unreliable or as other circumstances dictate.

1.2 Literature Reviews

A major literature review on the effects of various chemicals, including chlorophenols, on microorganisms, was carried out by Walker in 1988 (241). Other major reviews on chlorophenols were done by Jones in 1981 (8), Jones in 1984 (91), McKee et al. in 1984 (220), IJC in 1980 (258), Krijgsheld et al. in 1986 (335), EPA in 1979 (257), EPS in 1988 (289),Ware in 1988 (405) and NRCC in 1982 (321).

The following computerized data bases were, at least in part, also consulted in carrying out this literature review: PROPERTIES (428), CESARS (422), LOG P (420), ACQUIRE (419), RTECS (418) and ENVIROFATE (459). Many references were consulted through these data bases and the original papers were not read.

The large number of references cited in this report is not by any means exhaustive in this field. The literature on chlorophenols is enormous and has only been sampled. The literature is also selective. Of the chlorophenol congeners, pentachlorophenol (PCP) is by far the most studied. Several of the dichlorophenols (DCPs) and trichlorophenols (TCPs) are virtually ignored.
The pharmacokinetics of chlorophenols in rats and mice have been studied in detail. The effects of chlorophenols on freshwater fish have been well documented, but effects on other groups of organisms, such as amphibians, which appear to be quite sensitive, are poorly understood. Many subjects dealt with in this report, such as recreation, industrial use and irrigation, are virtually ignored by the literature.

1.3 Chlorophenols

There are 19 different chlorophenols (CPs) depending upon the number and arrangement of chlorine atoms on the parent phenol ring; only eight are of major commercial significance. As a group, all the chlorophenols are referred to as different congeners; those with the same number of chlorine atoms are isomers of each other.

Many commercial products contain chlorophenols, often in complex mixtures, and have often been contaminated with the more toxic dioxins and other organic compounds. It may be difficult in some cases to determine whether the observed toxicity results from the chlorophenols or from the contaminants.

Chlorophenols are used as broad-spectrum pesticides, and all act by uncoupling oxidative phosphorylation from respiration. This affects all aerobic, eukaryotic life in essentially the same way, and permits extrapolation of experimental data to organisms not specifically tested. The main reason for different LC50 values in various groups of organisms is the variable efficiency of net uptake, accumulation, and transfer to the mitochondria, which are the sites of chlorophenol action.

Chlorination of phenol greatly increases the toxicity of the resulting molecules relative to phenol. Generally, molecules with more chlorines are more toxic, due mainly to higher fat solubility as indicated by higher octanol/water coefficients (K o/w) values, resulting in greater uptake by organisms.

Table 1.3 gives the relative toxicity of many chlorophenols, in several biological test systems, relative to PCP being assigned a value of 1000. These values have been back-calculated from published LC50, IC50, LD50 and EC50 values, which indicate the dose or concentration of a chemical at which one half the test organisms were affected. Inspection of this table shows how much variability there is in the toxicity of any compound to different organisms, and how different end-point measures affect the magnitude of the numbers generated. A brief description of the test systems, A to P follows:

A is 24-h IC_{50} data for *Daphnia magna*
B is 96-h LC_{50} data for *Poecilia reticulata*
C is EC_{50} data for *Chlorella pyrenoidosa*
D is 48-h LC_{50} data for *Daphnia magna*
E is EC_{50} data for *Lemna minor*
F is 24-h LC_{50} data for *Carassius auratus*
G is EC_{50} data for rat liver enzymes
H is LD_{50} data for rat intraperitoneal injections
I is LC_{50} data for *Pimephales promelas*
J is EC_{50} data for *Tetrahymena pyriformis*
K is EC_{50} data for *Trichoderma viride*
L is EC_{50} data for *Raphanus sativus*
M is EC_{50} data for *Sorghum sudanense*
N is 96-h LC_{50} data for *Palaemonetes pugio* (intermolt)
O is 96-h LC_{50} data for *Palaemonetes pugio* (molt)
P is 24-h LC_{50} data for *Salmo trutta*

1.4 Registration

A British Columbia Ministry of Environment News Release (Aug. 30, 1989:109) reported that an August 24, 1989 Antisapstain Chemical Waste Control Regulation restricted levels in water discharged from lumber storage areas, to 6 µg/L (404). Revisions adopted on August 30, 1990 maintained these restrictions. Holland suspended the use of chlorophenols for indoor wood treatment. Canada, Denmark, Germany and Japan have suspended or restricted agricultural uses. Sweden banned all uses of chlorophenols in 1977 and Germany followed in 1987 (199). On August 1, 1990 Agriculture Canada terminated all sapstain control uses of TTCP and PCP effective December 31, 1990. Heavy duty wood preservation for outdoor use using chlorophenols is the only registered use and is presently being re-evaluated in Canada.

The United States cancelled registration of chlorophenols for herbicidal and anti-microbial uses, and for the preservation of wood in contact with food, feed, animals and livestock. Only certified applicators may use the product. Registration has been cancelled for use as a biocide in the wet end of paper production, and most other uses have also been proposed for cancellation. Even uses for lumber preservation and anti-sapstain fungal treatments are being, or have been, phased out as replacement preservatives are found. Only heavy timber and pole uses are being considered for retention.

2. CHARACTERISTICS

2.1 Synonyms and Commercial Product Names

Table 2.1.1 gives the names of the chlorophenols as used in this report and as generally used by the Chemical Abstracting Service, CAS, the Registry of Toxic Effects of Chemical Substances, RTECS or the Ontario Municipal-Industrial Strategy for Abatement, MISA. Formulae and molecular weights are also given in this table. There are alternate naming protocols and many commercial product names for the various chlorophenols. One needs to be aware of these when searching the literature for data on specific

active ingredients. Table 2.1.2 gives some of the alternate names by which chlorophenols may be found in the literature.

2.2 The Structure of Chlorophenols

There are 19 different chlorophenols, formed by replacing from one to five of the non-hydroxyl hydrogens of the phenol molecule with chlorine atoms. These include three monochlorophenols (MCPs), six dichlorophenols (DCPs), six trichlorophenols (TCPs), three tetrachlorophenols (TTCPs) and one pentachlorophenol (PCP). These compounds, and the parent phenol, are illustrated in Figure 2.2. Table 2.1.1 gives the names, formulae and molecular weights.

There are instances of confusion in the literature, mostly with TCPs and TTCPs, where the incorrect names or the wrong numbering systems are used. Different numbering systems are used which mask the identity of certain mirror image constructions resulting in the recognition of more distinct isomers, or congeners, than actually exist. Many of these incorrect numberings are listed in the synonym lists of section 2.1. For example, two literature sources give data for 2,4,5,6-TTCP which is identical to the properly named 2,3,4,6-TTCP. One reference lists ten DCPs and mentions that two other possible TTCP isomers are rarely mentioned in the literature. One must be aware that some good data may be reported under the wrong name due to carelessness or ignorance of organic chemistry naming protocols.

2.3 Properties and Effects of the Chlorophenols

Table 2.3 gives the CAS, MISA and RTECS registration numbers of the chlorophenols, whether or not they are in commercial use, and a number of physical characteristics which affect their environmental partitioning and biological effects. Only eight of the chlorophenol congeners are in commercial use, but all may be present as contaminants in many products, may be formed by chlorinating water or wastewater, or may result from the breakdown of higher chlorophenols in the environment.

All of the chlorophenols boil at temperatures well above the boiling point of water, most of them over 200C, or else they sublime. The melting points of all but four of the higher substituted congeners are below the boiling point of water. Vapor pressures are low, but not negligible, and tend to decrease as more chlorines are added. All specific gravities are greater than water and rise with increasing chlorine substitution to almost two in PCP. Only 2-MCP is a liquid at ambient temperatures, the others are generally needle-like crystalline solids. Solubility in water decreases with increasing chlorine substitution, as the Ko/w increases and the molecule becomes more hydrophobic. Due to their physical properties the chlorophenols will tend to accumulate in sediments.

Chlorophenols are weak acids; the dissociation constant generally increases with increasing chlorine substitution. The sodium and potassium salts are quite soluble at physiological temperatures and pH levels. PCP has an acid dissociation constant, pKa, of 4.8 and is largely dissociated at ambient levels of pH 7 to pH 8; thus most literature on Na-PCP is relevant for organisms in nature (7, 77, 258). The solubility of PCP is 14 mg/L at 20C, and of Na-PCP, 4 g/L at pH 8. At this pH, 99.9% of the PCP is dissociated and exists as Na-PCP in aqueous solutions (126). In alkaline solutions 2,4-DCP is very soluble (386, 442). Since the more hydrophobic undissociated chlorophenols can penetrate cell membranes and are more fat soluble than the dissociated forms, chlorophenols are more readily taken up by organisms, and are thus more toxic, at lower pH levels. The formulated products and oil-based solutions of chlorophenols are more toxic than aqueous solutions; they are also flammable. The

chlorophenols do not actually burn, rather they decompose on heating to form toxic, volatile, chlorinated gases (199).

Chlorophenols are adsorbed very strongly by activated carbon at the µg/L level. Adsorption is a function of pH, and is affected by competition for adsorption sites by other chlorophenols and other organic compounds. Neutral species predominate below the pKa values and are adsorbed more strongly than anionic species. As the number of chlorine substituents increases, the solubility of the neutral species decreases and adsorption increases; however, as substitution increases, the pKa drops. In a mixture of chlorophenols, competition for adsorption sites will significantly reduce the adsorption of any congener over the value measured in an isolated test. At pH 5.2, fulvic acid from decomposing leaf litter out competes chlorophenols for adsorption sites, while humic and soil fulvic acids are about equal in adsorption competition with the chlorophenols. At pH 9.1, humic and leaf fulvic acids out compete 2,4,6-TCP for adsorption sites (472).

Temperature is not a critical factor in the toxicity of chlorophenols to homeotherms (warm-blooded animals), but is important in poikilotherms (cold-blooded animals), where respiration and metabolic rates are a function of ambient temperatures. Ionized chlorophenols are less lipophilic, and thus the uptake rates and toxicity are greater at low pH. Some chlorophenols, particularly PCP, ionize well below normal ambient pH; so animals generally make contact with ionized chlorophenols in aqueous solutions.

The half-life of most chlorophenols is quite short under most natural conditions: half lives range from days to weeks, or, on occasion months. Accumulation of high levels of chlorophenols in organisms, and the maintenance of such high levels is the result of constant input to the environment. If such input were to cease, the chlorophenol levels would be expected to drop quite quickly in sediments, water, and organisms. Bacteria break down chlorophenols by two different mechanisms: ring cleavage to yield aliphatics instead of aromatics, and dechlorination. Most of the data in the following descriptions of hazardous biological effects by chlorophenols are taken from CESARS (422). No data were found for the other chlorophenol congeners not discussed below.

2-MCP

The pure compound has an unpleasant and penetrating odor. It is a strong tissue irritant and is toxic by skin absorption, ingestion, or inhalation. When heated to decomposition, 2-MCP emits highly toxic fumes (445, 476).

3-MCP

The material is toxic when inhaled, ingested, or absorbed through the skin, and is strongly irritating to tissues. When heated to decomposition, 3-MCP emits highly toxic fumes (445, 526).

4-MCP

This chlorophenol is a strong tissue irritant and is toxic through skin absorption, ingestion or inhalation. The pure compound has an unpleasant, penetrating odor. When heated to decomposition, highly toxic fumes are emitted (426, 445).

2,4-DCP

There is a slight fire hazard with 2,4-DCP; it reacts strongly with oxidizing agents and also gives off hazardous fumes when heated or in contact with strong acids. The compound is a strong eye and tissue irritant, and inhaled dust irritates the respiratory tract. It is toxic if ingested and is readily absorbed through the skin in toxic amounts. Chloracne and porphyria have been reported in manufacturing personnel (315, 426, 442, 445).

2,4,5-TCP

This material is non-flammable and no serious health hazard occurs with normal industrial use. Ingesting large amounts would be harmful, and copious quantities of dust or fumes will irritate eye and nose membranes causing iritis, conjunctivitis, and corneal injury. Skin irritations, redness, and edema may occur, but there is no danger of poisoning by skin absorption. Prolonged skin contact may result in mild to moderate chemical burns (313, 445).

2,4,6-TCP

This material is non-flammable, but heating the salt to 280 C produces a number of dibenzo-p-dioxins in the 0.1 to 0.3 mg/kg range. Dusts cause eye, nose and pharynx irritation and may injure the cornea. The compound is readily absorbed through the skin and causes irritation ranging from redness and edema to chemical burns (445, 476).

2,3,4,5-TTCP and **2,3,5,6-TTCP**

When heated to decomposition, these chlorophenols emit toxic chlorine gas fumes (426).

2,3,4,6-TTCP

The pure material has a pungent odor and is a strong skin irritant. It is non-flammable (445).

PCP

The pure material has a strong, pungent smell. It is non-flammable but generates toxic and irritating vapors when heated. Vapors and dusts irritate skin and mucous membranes. The vapors given off include CO, HCl and chlorinated phenols (483, 527).

2.4 QSAR Analyses of the Relative Toxicities of the Chlorophenols

Quantitative Structure-Activity Relationship Analyses of the results of an experiment testing some of the congeners or isomers in a series of related compounds, can often determine which active groups of the compounds have the most effect on the test organisms, and permit predictions of the toxicities of the remaining congeners or isomers. Table 2.4 and Figure 2.4 give the results of an experiment by Devillers et al. (57). The 24-h IC_{50} values of 17 of the chlorophenols were determined for *Daphnia magna*. The ratios of the 24-h IC_{50} of each congener relative to PCP were calculated and have been added to the table.

Figure 2.2 shows the substitution sites on the phenol molecule and their identification numbers; 2 and 6 are "ortho" sites, 3 and 5 are "meta" sites and 4 is the "para" site. Some observations and predictions

can be made from the data in Table 2.4. These are depicted graphically in Figure 2.4 and discussed below.

All the substitution sites on the phenol molecule are not equal in their effect on toxicity to organisms. As discussed in section 4.2.1, the location of the chlorines affects the efficiency of microbial breakdown of chlorophenols. Meta-substituted or 3,5- compounds, are more resistant to microbial degradation than are ortho- substituted or 2,6- compounds. Of the monochlorophenols (MCPs) 4-MCP is much more toxic than either 2-MCP or 3-MCP. Within any isomeric group of congeners, those with a chlorine in the 4, or para position, are more toxic than the others. In addition, if chlorines are substituted at both the 3 and 5, or meta positions, toxicity is high. If both the meta and the para substitutions are made in the same molecule, toxicity is also high, as shown by 3,4,5-TCP.

Toxicity is reduced by simultaneous 2 and 6, or ortho substitutions. Such substitutions can reduce toxicities that might be expected from previous arguments about 3, 4, and 5 substitutions. The relatively low toxicity of 2,6-DCP compared to 3,5-DCP, and the lower toxicity of 2,4,6-TCP than would be expected from a para or 4 substitution are examples of reduced toxicity due to ortho substitutions. This trend is very evident in the data of Saito et al. (701); even better correlations are obtained if these ortho compounds are omitted from the correlation analyses. Since there is increasing toxicity as more chlorines are added, one would expect PCP to be much more toxic than 3,4,5-TCP; however, since the two extra chlorines are in the 2 and 6 positions, little of the expected extra toxicity is seen. Similar arguments are used by Liu et al. (691) on studies with bacteria.

The relatively low toxicity of the ortho substituted compounds 2-MCP, 2,6-DCP and 2,4,6-TCP has been confirmed by a number of in vivo experiments. Shigeoka et al. (702) used killifish, *Daphnia*, algae and activated sludge microorganisms; Kobayashi et al. (273) used goldfish; Saarikoski et al. (144) used guppies; Nendza et al. (703) used *Escherichia coli*; Ribo et al. (367) used bacteria; and Babich et al. (704) used BF-2 cells.

An experiment by Saito et al. (701) on the uptake of neutral red dye by GF-Scale cells, a fibroblastic cell line derived from goldfish scale cells, is also a good source of comparative QSAR data. In this experiment 15 of the chlorophenols were tested and a good table of physicochemical properties of the chlorophenols is given. This data set is not as complete as that of Devillers and Chambon (57), and it ranks the chlorophenols on uptake of a non toxic dye rather than on toxicity. The order of relative uptake in the Saito (701) experiment correlates well (r = 0.965) with pKa and log Ko/w (measures of ionization and lipophilicity, respectively, which together predict **uptake**) but not necessarily with functional **toxicity**. Uptake and toxicity are not necessarily the same.

Using these arguments one would expect the toxicity of 2,3,4,6-TTCP, which was not tested by Devillers and Chambon (57), to be less than that of the other TTCPs, though perhaps slightly more toxic than 3,4-DCP. An estimated 24-h IC_{50} mean value for their experiment is about 2.70 mg/L with a congener to PCP ratio of 3.55. Similarly, the toxicity of 2,5-DCP is expected to be greater than that of 2,6-DCP, less than that of 3,5- or 2,4-DCP, and more toxic than 2,3-DCP. The estimated value for a 24-h IC50 mean value in their experiment is about 4.5 mg/L or a congener to PCP ratio of 5.92. See Table 2.4.

Since, for most species, there are generally abundant data on the effects of PCP, but few on the effects of other congeners, the ratios of the toxicities as calculated in Table 2.4, and inherent in the equations of reference 144, could be applied to the best PCP data to determine guidelines for the other congeners. This approach was tried and works well, but one can not apply pH corrections to the resulting guidelines. The guidelines resulting from this process are, however, comparable to those derived from a set of pH

dependent equations based on fish toxicity studies, when the pH dependent guidelines are corrected to the same pH and temperature.

2.5 Summary of the Characteristics of the Chlorophenols

Table 2.1 gives the official names of the chlorophenols as used by CAS and in this report; formulae and molecular weights are also given. Table 2.1.2 gives alternate names for the chlorophenols, some based on illegitimate numbering of the substituted chlorines. There are three MCPs, six DCPs, six TCPs, three TTCPs and one PCP; these are all illustrated in Figure 2.2. Table 2.3 gives many physical characteristics of the chlorophenols which affect their biological action or environmental partitioning. Eight chlorophenols are in commercial use and the rest may be produced incidentally when organic material is chlorinated.

Chlorophenols are weak acids and some are fully dissociated at ambient pH levels and are thus available as the salt. Since for most organisms there are generally abundant data on the effects of PCP but few on the effects of other congeners, the ratios of the toxicities, as given in Table 2.4, can be applied to the best PCP data to determine guidelines for the other congeners; such guidelines can not, however, be readily adjusted for other pH levels. Temperature is an important variable for chlorophenol toxicity to poikilotherms where respiration and metabolism rates are functions of the ambient temperature, but is not important for homeotherms. Chlorophenols are notorious for causing taste and odor problems in water at levels below toxicity. The half-life of most chlorophenols in nature is short, rarely as long as months, so, once input to the environment stops, levels will drop rapidly. Bacterial breakdown proceeds by both ring cleavage and dechlorination, the former process occurring first and most readily.

3. DISSEMINATION OF THE CHLOROPHENOLS

3.1 Uses of Chlorophenols

Chlorophenols as a group were used as broad-spectrum biocides world-wide; residues and breakdown products are almost ubiquitous in the environment. Specific uses are as herbicides, pesticides, fungicides, algicides, insecticides, bactericides, molluscicides and slimicides. They were used by the domestic, agricultural, and industrial sectors of society (8, 30, 48, 161, 164, 199, 202, 222, 223, 225, 254, 256, 258, 268, 270, 289). Chlorophenols were used as slimicides in pulp and paper manufacturing (202, 222), and in cooling towers (289); they were also used as general biocides in paints, oils, leather, textiles, cellulose and starch compounds, adhesives, proteins, rubbers, rug shampoos, photographic processing chemicals, and food processing (48, 202, 289).

The major use of penta- and tetrachlorophenols has been as a wood preservative and anti-sapstain fungicide. Fungi cause staining in the sapwood which reduces wood quality and market price; such attacks may also promote attacks by other organisms which cause structural damage to timbers (33, 48, 202, 222, 254, 256, 258, 268, 289). In dark, warm, humid conditions, like the holds of ships, such fungi can grow rapidly (268). Chlorophenols were used primarily for preservation of freshly cut dimension lumber, but they are now used primarily for other kinds of wood preservation such as fence posts, telephone poles, railroad ties, pilings, and timbers used for bridges and in mines. The useful life of such treated timbers is greatly extended, which saves money in replacement costs and also reduces the need

for such wood products. In addition, smaller timbers can be used, since there is no longer a need to make allowance for reduced strength, caused by structural damage, by using oversized timbers. The useful life of treated wood timbers can be extended 5 to 15 fold and timber needs reduced three to six fold over untreated wood. The life-span of untreated wood is quite short: 2 years for mine timbers due to moisture and high temperatures, 5 years for railway ties due to chewing insects, and 1 year for marine pilings due to borers (256).

For wood preservation, PCP is preferred but some TTCPs are also used as active ingredients. The less substituted chlorophenols are less desirable due to their higher odors, greater solubilities in water, greater volatility and potential for skin irritation. Some specific uses of the chlorophenol congeners are given below; data were not available for all of the congeners.

MCP

Monochlorophenols have been used as antiseptics for 100 years (397).

2-MCP

This compound is used as an intermediate feedstock in the manufacture of higher chlorophenols and chlorocresols used as biocides. It is also used to form intermediates in the production of phenolic resins and to extract sulphur and nitrogen compounds from coal (372).

3-MCP

This chlorophenol is also used to extract sulphur and nitrogen compounds from coal (364) and as an intermediate in organic synthesis of other chlorophenols and phenolic resins (442, 445).

4-MCP

The most common monochlorophenol, 4-MCP, is used to extract sulphur and nitrogen compounds from coal (372). It is an intermediate in the synthesis of dyes and drugs, a denaturant in alcohol, a solvent in the refining of mineral oils, and is used in the production of the herbicide 2,4,-D, the germicide 4-chlorophenol-o-cresol and the chlorophenol 2,4-DCP (372, 442, 445).

2,4-DCP

The herbicide, 2,4-D, and the chlorophenol, PCP, are made from 2,4-DCP. Alkali salts of 2,4-DCP are used as germicides and antiseptics, to manufacture the miticide 2,4-dichlorophenol benzene sulfonate, to make the seed disinfectant, antiseptic and moth repellent, 2,2'-dihydroxy-3,5,3',5'-tetrachlorodiphenylmethane and as an intermediate to make soil sterilants, plant growth regulators and wood preservatives (8, 315, 495).

2,4,5-TCP

This isomer is used to manufacture hexachlorophene, a disinfectant and sanitation product for domestic, hospital, and veterinary use. The main uses of 2,4,5-TCP are as fungicides and bactericides on swimming pool related surfaces, household sickroom equipment, food processing plants and equipment,

food contact surfaces, hospital rooms, and bathrooms. It is used in the textile industry and as a raw material to manufacture the industrial and agricultural chemicals 2,4,5-T, Silvex, Ronnel, Erbon, and Sodium 2,4,5-Trichlorophenate (445, 464, 480) .

2,4,6-TCP

The sodium and potassium salts of 2,4,6-TCP are used for wood preservation (222, 225) and as antiseptics, fungicides and bactericides. They are used as preservatives in glue, for wood preservation, as anti-mildew agents in textiles, as germicides, and as defoliant herbicides (232, 445, 464).

2,3,4,5-TTCP

This tetrachlorophenol is used as a fungicide and wood preservative (442, 445) in wood processing plants such as sawmills and plywood plants, wood protection and preservation plants, kraft pulp mills, sewage treatment plants, and pesticide manufacturing and formulating plants (321).

2,3,4,6-TTCP

This tetrachlorophenol is used as an insecticide and wood preservative (442, 222, 225); for the latter use it is usually as the sodium or potassium salt. The major use is as a fungicide in wood preservation (442, 445) in sawmills, plywood plants, wood protection and preservation plants, kraft pulp mills, sewage treatment plants, and pesticide manufacturing and formulating plants (321). Commercial PCP usually contains 3% to 10% 2,3,4,6-TTCP as an active ingredient for wood preservation.

2,3,5,6-TTCP

This tetrachlorophenol is used as a fungicide and wood preservative (442, 445) and in wood processing plants such as sawmills, plywood plants, kraft pulp mills, and wood protection and preservation plants. It is also used in pesticide manufacturing and formulating operations (321).

PCP

Petrochemical drilling fluids contain polysaccharides, starch and XC polymer; Na-PCP was used, in concentrations around 700 to 1400 mg/L, to prevent bacterial fermentation of these fluids (202, 640). About 75% of the PCP used was applied as a wood preservative for poles, pilings and cross-arms. About 15% of the PCP was used as the sodium salt, Na-PCP, in the leather, paper, fibreboard, photography, paint, construction and textile industries and as a molluscicide. It was usually used at a 1 to 10% concentration as an aqueous solution for these purposes. It was also used for slime control in adhesives, proteins, oils, leathers, paints and rubber (538).

In marine situations 1 mg/L of Na-PCP prevents fouling of pipes and conduits (164). PCP was the main chlorophenol used as a slimicide in pulp and paper manufacturing (202, 222) and in cooling towers (289). It is also used as an intermediate in the manufacture of the pesticide 2,4-D and other chemicals (289). PCP is the major chlorophenol used in wood preservation and anti-sapstain treatment where it is often used as the sodium or potassium salt, Na-PCP or K-PCP. It is also used as an insecticide against termites, wood borers, and powder-post beetles (48, 202).

In agriculture, PCP was used as a pre- and post-planting herbicide, especially against grasses in rice paddies (48, 223), and as a general purpose fungicide (223). PCP controls weeds that 2,4-D does not, and is cheaper. It has therefore supplanted 2,4-D in South-East Asia, resulting in extensive fish and shellfish kills (223).

In North America the only currently registered uses for PCP are for pressure and thermal treating of railway ties, utility poles, pilings and outdoor construction materials. All other uses have been stopped.

3.2 Commercial Chlorophenol Production

Table A3-1 in Jones (91) gives an extensive list of over 100 commercial products containing chlorophenols, which were registered under the Canadian Federal Pest Control Products Act as of February 1983. Some of these data are reproduced in Table 3.2. There are two basic ways to produce chlorophenols: one is to successively chlorinate phenol, and the other is to hydrolyze chlorobenzenes. The latter process, especially at higher temperatures, can produce dioxins (222). All technical and formulated products made from chlorobenzenes in this way are contaminated by dioxins to some degree (474). This process is no longer used to make the PCP available in North America.

2-MCP

This product is prepared by the direct chlorination of phenol, passing gaseous chlorine over and into molten phenol at 50 to 150°C, which leads to both 2-MCP and 4-MCP. Fractional distillation is then used to separate the two isomers, which have a difference of 40 to 45C in their boiling points (See Table 2.2). Diazotized O-chloroaniline can also be used to prepare 2-MCP (161, 315).

3-MCP

The commercial preparation of 3-MCP is from meta-chloroaniline through the diazonium salt (495).

4-MCP

Parachlorophenol is prepared in a number of different ways: from chloroaniline through the diazonium salt, from diazotized parachloroaniline, from paranitrosophenol by a modified Sandmeyer reaction, by selective reduction of chlorobromophenols, and by direct chlorination of phenol followed by fractional distillation as mentioned under 2-MCP (161, 445).

2,4-DCP

Phenol, or monochlorophenols, are chlorinated to produce 2,4-DCP; a patented process dissolves phenol in liquid SO2 and treats it with cold gaseous chlorine to yield 98% 2,4-DCP (442).

2,4,5-TCP

The trichlorophenol, 2,4,5-TCP, is made by chlorination of phenol, by hydrolysis of the trichlorobenzene, or by alkaline hydrolysis of 1,2,4,5-tetrachlorobenzene under pressure at 180°C in the presence of

aqueous NaOH and methanol. The hydrolysis methods result in contamination by dioxins, especially TCDDs, tetrachloro-p-benzodioxins, in the last method (222, 474).

2,4,6-TCP

Chlorination of phenol is one method used to produce 2,4,6-TCP. The oxidation of O-dichlorobenzene to form O-chlorophenol, followed by chlorination with chlorine gas, is an alternate method (526).

2,3,4,5-TTCP

The tetrachlorophenol, 2,3,4,5-TTCP, is a by-product of PCP production and is also made by the K2TeO3, potassium tellurate, catalyzed chlorination of phenol. The latter method produces only tetrachlorinated phenols (89, 442).

2,3,4,6-TTCP

This tetrachlorophenol, 2,3,4,6-TTCP, is made by chlorinating phenol and also obtained commercially as a by-product of PCP manufacture at about 4% to 10% by weight. The K2TeO3 catalyzed reaction, as mentioned under 2,3,4,5-TTCP, is also used (442).

2,3,5,6-TTCP

Chlorinating phenols is one method of making 2,3,5,6-TTCP. A low yield is also obtained as a by-product of PCP manufacture (89). The K2TeO3 catalyzed chlorination of phenol, as mentioned under 2,3,4,5-TTCP, is also used (442).

PCP

PCP may be made by chlorinating phenol, or by the hydrolysis of chlorobenzene. The latter process leads to contamination with dibenzo-p-dioxins (222, 474). Anhydrous aluminum chloride or ferric chloride are used as catalysts in the final chlorination stages. Mixtures of 2,6-DCP and 2,4,6-TCP with phenol may also be used as starting materials during chlorination, which proceeds in stages at progressively higher temperatures (161, 464). Chlorinating phenol is the only process in current use for the production of PCP imported into Canada.

3.3 Contaminants in Commercial Chlorophenols

As indicated in section 3.2, one method of producing chlorophenols, the hydrolysis of chlorobenzenes, produces dibenzo-p-dioxin contamination of the product to some degree. Table 3.3.1 gives the levels of some other contaminants in commercial 2,4-DCP. Since this product is used primarily to manufacture the herbicide 2,4-D, either as the acid, amine or ester, similar formulations of the contaminants are being distributed to the environment wherever 2,4-D is being used (91, 684). There are numerous reports in the literature from the 1970s and early 1980s of impurities in technical PCP formulations (222, 37, 217).

The quality and quantity of commercial chlorophenol products vary widely. The actual quality and quantity of the product used to determine acute and chronic levels of chlorophenols for various organisms are not always reported in the literature. This causes several problems with literature values

of toxic levels of chlorophenols. Quantitatively, the toxic level quoted will be too conservative when the product is not pure since a lower actual concentration of chlorophenol is present. Qualitatively, one can rarely be sure just how much of, or which of, the toxic effects are due to the chlorophenol, and how much is due to the contaminants (147). Since common contaminants in chlorophenols were dibenzo-p-dioxins, which are toxic at levels about 1x10-6 of the levels at which chlorophenols are toxic, a very low level of dioxin contamination may have masked the toxicity of the much more abundant chlorophenol. The manufacturing processes, and thus the levels and types of impurities, vary both with supplier, and with time and lot number of the product.

Technical PCP contains a large number of impurities depending upon the manufacturing method. These include other chlorophenols, particularly TTCP isomers; PCDDs, polychlorodibenzo-p-dioxins; PCDFs, polychlorodibenzofurans; polychlorodiphenyl ethers, chlorinated cyclohexanes and cyclohexadienons; polychlorophenoxyphenols; hexachlorobenzene, and polychlorinated biphenyls, PCBs (199). The most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-p-dioxin, was reported not to be found in chlorophenols used as wood preservatives (267); however, a conflicting report indicates that it was present in NaPCP used for this purpose (715).

Table 3.3.2 lists the concentrations of chlorophenols present in some commercial products used in acute and chronic toxicity tests, as reported by the experimenters. Table 3.3.3 lists the major contaminants present in these products and Table 3.3.4 lists the major dioxins present. Table 3.3.5 gives the dioxin and furan analyses of three chlorophenol formulations. These four tables give some idea of the range and concentrations of compounds the test animals were subjected to, and which might have been responsible for some portion of the toxicity ascribed to the PCP. Typical contaminants for formulations used in wood preservation are <1 to 2000 mg/L of chlorinated dioxins, 100 to 1000 mg/L of chlorinated diphenylethers, 50 to 200 mg/L of chlorinated dibenzofurans, and about 10 g/L of chlorinated phenoxyphenols. One PCP formulation available in Sweden was 80% TTCP; PCP was only an impurity (222).

The concentration of 2,3,7,8-TCDD dioxin in the current (1996) formulations of PCP by Vulcan Chemicals and KMG-Bernuth are regulated by the USEPA under the 1986 RPAR agreement at less than the detection limit of 1 µg/Kg (656). The PCP guideline would thus protect people from harmful effects due to dioxin contaminants in the PCP, at the known levels of this contamination. The RPAR agreement limits HCDD to a maximum of 4 mg/L and a mean of 2 mg/L (656). The HCB level may not exceed 75 mg/L. In practice the monthly mean HCDD is between 1.3 and 1.8 mg/L and no 2,3,7,8-TCDD is detectable at detection limits of 50 ng/L. There is no tetra- or penta PCDF at the detection limits of 10 µg/L, hexa PCDF ranges up to 64 mg/L and hepta PCDF up to 105 µg/L.

"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" (320). "The contaminants associated with PCP may be a more serious threat to environmental and occupational safety than PCP itself" (127, 129). The effects of 2,3,7,8-dibenzo-p-dioxin on primates is documented by Allen and Miller (403). It is likely that some of the toxic effects in birds and mammals exposed to chlorophenols are actually caused by the many impurities present in technical and commercial formulations. Chloracne and porphyria tarda effects in the employees who manufacture 2,4,5-TCP, are likely due to the TCDD contaminants and not to the 2,4,5-TCP itself (480).

More and varied toxic effects are reported from animals exposed to these commercial grades of PCP than from animals exposed to purified PCP (35, 38, 312, 316, 317, 318). Yolk sac edema and skull

deformations, common in birds exposed to chronic levels of technical grade PCP, are nearly absent in similar tests done with purified PCP. In a chick edema bioassay, rabbit ear dermatitis assay and rat oral dose hematological effects assay, pure PCP had no effect except increased organ weights in livers and kidneys of rats given 30 mg/kg/day. Technical grade PCP containing 1980 mg/L octa- and 19 mg/L hexachlorodibenzo-p-dioxins, gave positive results in the bioassays and also caused altered blood chemistry and organ anatomy (317).

Cattle fed pure PCP, technical grade PCP, and mixtures of the two at 20 mg/kg PCP for 42 days followed by 15 mg/kg for the next 4 months showed dioxin and furan levels in the liver and fat correlated with the proportion of technical PCP in the diet. Blood hexachlorobenzene levels rose concomitantly (572). The commercial mixture of PCP with its high contaminant levels, and relatively low PCP level, was more toxic than purified PCP or Dowicide EC-7, due to high levels of dibenzo-p-dioxins and phenoxyphenols. PCP, when purified, did not affect growth below 0.085 mg/L, and Dowicide EC-7 did not affect growth up to 0.139 mg/L (127).

In addition to the variation in purity of the pentachlorophenol used in experiments, which ranged from 99%+ to 75%, the PCP was often used as the sodium salt, Na-PCP. Some experimenters (97) carried out parallel tests which showed differences in the LC50s between Na-PCP and PCP, but in many cases the two compounds are not clearly distinguished and are treated as being synonymous. The numbers reported will be different due to the difference in molecular weight of the two compounds, Na-PCP is 8% heavier and thus contains less PCP/mg than does pure PCP, and also due to the differences in the polarity, solubility and Ko/w of the compounds which affect uptake rates by organisms. The differences are well marked in short-term experiments, 12 to 24 hours, but almost disappear by 96 hours. Thus the effects are likely one of uptake rates and not important in long-term, low-level chronic effect exposures. At higher pH levels where dissociation is virtually complete this effect should not be significant. When it is known which compound was used, this is noted in the tables of effects on organisms, and Na-PCP or PCP is specified; some "PCP" entries are likely actually Na-PCP.

3.4 Sources of Input to the Environment

In spite of their relatively short half-lives and relative ease of degradation, chlorophenols are routinely found world-wide in water, soil, sediment and biota (11). This is due, at least in part, to their very widespread use, as well as to misuse, spills, and leaching from treated wood products and waste dumps. Most developed countries have found it necessary to restrict the use of chlorophenols, initially for domestic and agricultural uses, but increasingly for wood preservation uses as well (199). Chlorophenols, mostly PCP, were imported from the US and France into Canada but currently the only imports are from the US The world-wide production was about 30,000 tonnes in 1989; approximately 750 tonnes entered BC (199). About half of the BC total was used in anti-sapstain treatments on cut lumber. Chlorophenols have a relatively short half-life in water, biota and sediment, and once input stops, environmental levels are expected to drop quickly. Chronic input is required to maintain the current environmental levels (243). All of the chlorophenols have been detected in Kraft pulp mill effluent (705).

The use of many chlorinated pesticides adds to the chlorophenol load to the environment since some of the breakdown products and metabolites of these pesticides are chlorophenols. The decay of vegetation produces phenols; wastewater from coal and wood distillation, petrochemical refining, steel mills, foundries, chemical plants, livestock dips, and domestic wastes also contain phenols. These phenols are converted to chlorophenols by chlorination in wastewater treatment plants (236). Our dependency on chlorine as a water treatment tool is responsible for much incidental chlorophenol production and input to the environment. Removal of organics from wastewater streams before chlorination, or conversion to UV

and ozone as water sterilization techniques, for both wastewater and drinking water, would eliminate or greatly reduce this source of chlorophenol production and widespread distribution.

The use of chlorophenols in the lumber industry has been the main contributor to the environment. Losses, spills, floods, leaching, at treatment plants and from treated wood, all contribute chlorophenols in large quantities, and chlorophenols are present in pulp and paper mill wastewater (258). The total annual runoff from lumber storage yards, expressed as pure PCP, is estimated at 916 kg to the lower Fraser River below Kanaka Creek, 523 kg to Burrard Inlet and 85 kg to Howe Sound at Squamish (271).

Mackenzie et al., 1975 (673) found that for the period 1960 to 1973, 62 fish kills were reported in BC, 17 caused by pesticides. Four were associated with the use of PCP and TTCP for treatment of wood and poles. In May 1963, 1000 ocean perch were killed by PCP in Sooke Basin as a result of lumber treatment. In August 1972, 1000 trout, salmon and stickleback were killed by PCP in the Little Campbell River due to spraying of hydro poles. In Victoria Harbor in December 1972, 10 tons of herring and anchovy were killed by PCP from a pole and sawmill wood treating plant. In the Manquam Channel in October 1973, 500 shiners and 500 adult and juvenile coho salmon died from PCP and TTCP overflowing from a lumber treatment tank.

MCPs

MCPs are formed when drinking or effluent waters containing phenol are chlorinated; they are present in pulp and paper effluents and wood preservation waste. Microbial breakdown of pesticides like 2,4-D, 2,4,5-T, Silvex, Ronnel, Lindane and benzene hexachloride, produce MCPs in the environment (372, 591).

2-MCP

The herbicide 2,4-D is broken down by Pseudomonas to 2-MCP (441). The chlorophenol 2-MCP is formed in the 1.7 µg/L range during chlorination of municipal waste water (480, 591). This compound is a major cause of odors.

3-MCP

Chlorinated sewage effluents have been found to contain 3-MCP in the 0.5 µg/L range (591). Breakdown of 2,3,4,5-TTCP by soil bacteria produces 3-MCP (17, 606).

4-MCP

Chlorinated sewage effluent contains 4-MCP in the 0.7 µg/L range (591).

DCPs

These compounds are found in leachate from dump sites and landfills (593, 594). There is no consensus yet on whether these compounds are found in significant levels in chlorinated wastewater or cooling water (591, 595, 183, 188).

2,4-DCP

The chlorophenol, 2,4-DCP, is formed in the nano- to micro-molar range during chlorination of municipal wastewater (480), and is a major odor causing compound. It is formed as a breakdown product of the herbicides 2,4-D and Nitrofen (495). Kraft pulp mill effluents contain 2,4-DCP (592).

2,5-DCP

When 2,4,5-T breaks down by photolysis in aerated water, 2,5-DCP is a product (493).

2,6-DCP

The chlorophenol, 2,6-DCP, is formed in the nano-to-micro-molar range by the chlorination of municipal wastewater (480), and is a major cause of odors. It is also produced in the pulp mill bleaching process (267).

3,4-DCP

In soil, 3,4-DCP is found as a breakdown product of PCP (294). Breakdown of 2,3,4,5-TTCP by soil bacteria produces 3,4-DCP (17, 606).

3,5-DCP

In soils 3,5-DCP is found as a breakdown product of PCP (294).

TCPs

TCPs occur in wastes from wood preserving and pulp and paper industries and form, in water and wastewater containing phenol, when chlorination occurs. TCPs are produced as degradation products of Lindane and 2,4,5-T in soil and in livestock, and are thus found in farmland or agricultural land runoff and also in landfill drainage from industrial or municipal wastes (183, 372, 592, 593, 594).

2,3,4-TCP

The degradation of Lindane in soil results in the formation of 2,3,4-TCP (606).

2,3,5-TCP

The degradation of Lindane in soil results in the formation of 2,3,5-TCP. Breakdown of 2,3,4,5-TTCP and 2,3,5,6-TTCP by soil bacteria also produces 2,3,5-TCP (17, 606).

2,3,6-TCP

Breakdown of 2,3,5,6-TTCP by soil bacteria produces 2,3,6-TCP (17, 606).

2,4,5-TCP

The primary degradation product of the herbicide 2,4,5-T, is 2,4,5-TCP (480, 176). The chlorination of wastewater containing phenol results in the production of 2,4,5-TCP (225). There is evidence that 2,4,5-T is also a metabolite or primary degradation product of other pesticides including Silvex, Ronnel, Lindane and benzene hexachloride (480). In leachate from Vancouver industrial and municipal wastes, 2,4,5-TCP has been found at levels up to 2.4 mg/L (594). Breakdown of 2,3,4,5-TTCP and 2,3,4,6-TTCP by soil bacteria, produces 2,4,5-TCP (17, 606). It is also produced in the pulp mill bleaching process (274).

2,4,6-TCP

Corn and peas metabolize pentachlorocyclohexene and 1,3,5-trichlorobenzene to 2,4,6-TCP (230). One of the metabolic breakdown products of the insecticide Lindane is 2,4,6-TCP (225, 226, 275). Kraft pulp mill effluent has been shown to contain 2,4,6-TCP (592), and levels up to 3.12 mg/L were found in municipal and industrial wastes in Vancouver (594). It has been estimated that when water containing phenol is chlorinated, 40 to 50% of the chlorophenols formed consist of 2,4,6-TCP (183). It is also one of the predominant chlorophenols produced in the pulp mill bleaching process (274).
TTCPs

Agricultural lands are sources of tetrachlorophenols to surface waters. They are used in herbicides and as wood preservatives for farm buildings and fences (8). While TTCPs are not likely formed by chlorination of sewage, they are present due to industrial discharges (594). Leachates from landfill sites contain TTCPs (594) and TTCPs form as degradation products from PCP, but the main source is waste from wood preserving industries: levels up to 2.1 mg/L have been found in such discharges (594).

2,3,4,5-TTCP

The World Health Organization, WHO, has determined that 2,3,4,5-TTCP is a metabolite of the pesticides Lindane and PCP (522); it is also a degradation product of PCP and a by-product of PCP production (89). PCP is broken down by sunlight to form 2,3,4,5-TTCP which persists in fish (201).

2,3,4,6-TTCP

This material is found in fly ash from municipal incinerators and thermal power stations which burn peat, wood waste, PCB- contaminated and non-contaminated oils, and municipal wastes (274, 290). It is also formed as a by-product in the manufacture of PCP. Flue gases from fireplaces and other wood burning processes, and smoke from slash, forest and grass fires all release 2,3,4,6-TTCP to the atmosphere (290). The waste from wood preservation facilities is a source of 2,3,4,6-TTCP, and PCP (291). It is also produced in the pulp mill bleaching process (267).

2,3,5,6-TTCP

This congener is found in fly ash from municipal incinerators and thermal power stations which burn peat, wood waste, PCP -contaminated and non-contaminated oils, and municipal wastes (274). The waste from wood treatment facilities contains 2,3,5,6-TTCP and PCP (291). PCP is broken down by sunlight to form 2,3,5,6-TTCP which persists in fish (201).

PCP

Incinerators, fly ash, flue gases, slash fires, fireplaces and other wood burning processes, all release PCP to the atmosphere (290). Hexachlorobenzene and pentachlorobenzene are common waste products which natural microbial metabolism converts to PCP (12, 216).

PCP was measured in sewage treatment plant effluents, over the range of 0.065 to 1.300 µg/L, in 13 samples from 7 plants in Ontario. PCP is used in wood preservatives to treat farm buildings, fence posts, telephone poles, and compost boxes, and is also used in herbicides. Such uses contribute to diffuse runoff to water courses (8). Wastes from other industries and the leachate from landfill sites in Vancouver contain PCP (594). The concentration of NaPCP used in drilling fluids as a bactericide, is about 700 to 1400 mg/L. Used fluid is put into sumps which could range from 2,800 to 11,300 m3 for a partial season of operations. Due to flooding, wash outs, storms and other accidents, this fluid is often released to the environment and contaminates local surface waters (640).

Between 1972 and 1982, EPS documented 26 NaPCP spills in BC; some resulted in fish kills in surface waters and others caused ground water contamination. The usual cause was flooding of dip tanks and drive through tanks by heavy rains, and surface run-off of drip areas after rainstorms (271). In 1977 a railroad box-car was used to ship PCP from the production plant in Alberta to a pole-treatment plant. The box-car was then used to ship feed oats from Northern Alberta to Thunder Bay, Ontario. Finally it was used to ship feed-grain to eastern Ontario. The feed-grain was sufficiently contaminated with PCP that cattle would not to eat it! Car sweepings had 2 µg/kg of PCP (650).

3.5 Levels of Chlorophenols found in the Environment

3.5.1 Air

PCP

The outer layers of treated wood contain up to several hundred mg/kg of PCP. Due to volatilization, air levels of PCP in proximity to large amounts of treated wood, or in confined spaces, will be significantly higher than background. Airborne levels of PCP at production and wood preservation sites range from several mg/m^3 to 500 mg/m^3 (27). Two background air sampling stations in the mountains above La Paz, Bolivia, at 5200 m, measured 0.93 and 0.25 µg PCP/1000 m^3 of air, and four Antwerp, Belgium samples varied from 5.7 to 7.8 µg PCP/1000 m^3 of air (688). The level of PCP in Burlington Ontario rainwater was 10 µg/L in 1982 (685).

3.5.2 Water

3.5.2.1 Drinking Water

Chlorophenol residues in drinking water rarely exceed several µg/L and are usually below one µg/L.

3.5.2.2 Ambient Water

Table 3.5.1 gives some chlorophenol levels found in Fraser River water (269, 720) and Table 3.5.2 gives water and sediment levels of PCP and TTCPs for several British Columbia sites. Table 3.6.1 gives summaries of the chlorophenol levels found in the water of the Fraser Estuary between 1973 and 1987

(693). The range of concentrations of chlorophenols detected along the BC coast in the general proximity to bleached pulp mills is: 2,4,6-TCP, 1.6 to 20.0 µg/L; 2,3,4,6-TTCP, 1.0 to 7.1 µg/L and PCP, 1.7 to 2.8 µg/L (713).

Chlorophenols (92% PCP and 8% TTCPs) were sampled in the waters of railway and utility right-of-way ditches of the lower mainland of BC. The ditches flowed into salmon bearing streams. The water adjacent to poles in utility right-of way ditches contained a mean of 1408 µg/L and water 4 m downstream contained a mean of 13.6 µg/L. Water adjacent to poles in railway right-of way ditches contained a mean of 225 µg/L and 3.8 µg/L 4 m downstream. Water levels adjacent to the poles exceeded the LC_{50} for salmonids and the downstream levels were high enough to cause chronic effects (719).

TCPs

Rhine river water in the Netherlands, sampled in 1978, contained 0.04 to 0.63 µg/L of 2,4,6-TCP and 2,4,5-TCP. Generally, samples from the Fraser River at Hansard, Hope and Marguerite do not show measurable levels of chlorophenols, but in the period from November 1990 to March 1991, there were six detectable values for TCPs of 0.07, 0.08, 0.1, 0.11, 0.13 and 0.17 µg/L at the Marguerite site (698).

TTCPs

In British Columbia surface waters, TTCP levels exceeded 1 µg/L in 1979, but were only 0.1 µg/L in 1986 (87, 269, 720). TTCP levels up to 5.2 µg/L were found in British Columbia waters in 1984 (88). Generally, samples from the Fraser River at Hansard, Hope and Marguerite do not show measurable levels of chlorophenols, but in the period from November 1990 to March 1991 there were three detectable values for TTCPs of 0.05, 0.07 and 0.16 µg/L at the Marguerite site (698).

2,3,4,6-TTCP

Water samples taken from the North Arm of the Fraser River in 1985 contained 0.002 to 15.2 µg/L of 2,3,4,6-TTCP (86).

PCP

"Uncontaminated" areas have background PCP levels at or below the ng/L detection limit in water. Levels several orders of magnitude higher are found near industrial discharges and in areas that have been subject to spills. Levels reach mg/L in chronically contaminated areas (8, 9, 201, 289, 291, 299, 300). PCP is generally high and persistent in water and sewage samples (201, 212), particularly in heavily industrialized areas such as the Great Lakes.

Stream mouths and near-shore areas along the Canadian Great Lakes contained 0.005 to 22.0 µg/L of PCP in 78 of 85 water samples tested. A Lake Michigan watershed contained 0.1 to 40.0 µg/L (225). Rhine river water in the Netherlands contained 0.15 to 1.5 µg/L PCP in a 1978 survey (225). Drinking water in Dade County, Florida, was analyzed for PCP; municipal supplies ranged from <0.030 to 0.340 µg/L, with a mean of 0.098 µg/L and wells ranged from <0.030 to 0.110 with a mean of 0.044 µg/L. These levels were low enough that blood serum levels of residents did not differ when drinking these two sources of water (576).

Up to 7.3 µg/L PCP have been found in British Columbia waters (88). Water samples in the North Arm of the Fraser River contained 0.002 to 2.80 µg/l of PCP (86) and PCP levels in British Columbia surface waters exceeded 1 µg/L in 1979 but were only 0.09 µg/L in 1986 (87, 287). Generally, samples from the Fraser River at Hansard, Hope and Marguerite do not show measurable levels of chlorophenols, but in the period from November 1990 to March 1991 there was one detectable value for PCP of 0.22 µg/L at the Marguerite site (698).

Segments of pressure treated poles were held in continuously circulated water at various pH levels and in dilute HCl for 30 days. Leaching rates ranged from 6.33 x 10-3 to 1.67 x 10-4 mg PCP/kg water / square inch of wood. The amount of PCP in solution increased with exposure time and with higher pH. Solutions reached their peak leaching rate in 1 to 3 days. The maximum concentration reached after 30 days was 65.5 mg/L in the water buffered at pH 9 (724).

3.5.3 Food Products

Chlorophenol residues in fruits and vegetables are usually below 10 µg/kg, as are levels in all meats except liver, which may reach 100 µg/kg. Fish skeletal muscle is usually below 4 µg/kg. Milk from southern Ontario dairies was analyzed for PCP, 2,3,4,6-TTCP, 2,4,5-TCP and 2,4,6-TCP. Detection limits were 0.1 µg/L for PCP and 1 µg/L for the other congeners. No chlorophenols were detected (649). Table 3.5.3 gives the levels of PCP and TTCP found in Canadian food samples from 1975 to 1978. The samples were collected in Alberta.

PCP

Food products often contain PCP (27, 170), usually from contact with treated shipping, storage or packaging materials. Levels of 1 to 100 µg/kg have been found in powdered milk, soft drinks, bread, candy bars, rice, noodles, cereal, sugar and wheat (27). Food fish contain PCP at 1 to 4 µg/kg (31). The PCP metabolite, pentachloroanisole, has been found at levels of 1 to 18 µg/kg in broiler chickens when the birds are raised on wood shavings derived from treated wood (32).

3.5.4 People

People are subjected to chlorophenols, mostly PCP, in their food, water and air. For the U. S. A. and Germany, estimates are 6 µg/d in food, 2 µg/d in water and 2 µg/d in air. Other sources include veterinary supplies, fabrics, disinfectants, photographic solutions, rug shampoos and pharmaceuticals (199).

2,4,5-TCP

A survey of the general population of the US showed only 1.7% of the urine samples were positive for 2,4,5-TCP, with a mean of <5.0 µg/L and a maximum of 32.4 µg/L (22).

PCP

Seminal FLuid
PCP levels in human seminal fluid ranged from 20 to 70 µg/L with a mean of 50 in a 1976 study of US men (27).

Adipose Tissues
Adipose tissue levels of the general public in Florida ranged from 5 to 52 µg/kg with a mean of 25 µg/kg (29). Post-mortem samples from people who took PCP overdoses showed tissue levels of 20 to 140 mg/kg (30). People in industrialized societies are generally contaminated with PCP at tissue levels of 10 to 20 µg/kg (23).

Blood Plasma/Serum
People in industrialized societies are generally contaminated with PCP at 1 µg/L or more in the plasma. This is mostly food chain and wood products exposure, or metabolism of hexachlorobenzene compounds (23). Surveys of plasma levels have been carried out in several countries; plasma levels in six people in New Orleans ranged from 0.07 to 45.4 mg/L. In 23 dialysis patients, levels averaged 16 µg/L while 14 controls averaged 15 µg/L (23).

Occupational exposures in the wood preservation industry result in human serum levels in the µg/L to mg/L range, while the general public levels are in the µg/L range. Urine and fat levels are similar. Workers in a PCP plant in Idaho were checked monthly for serum and urine levels. Serum levels ranged from 0.348 to 3.963 mg/L, with a mean of 1.372 mg/L. Controls had levels of 0.038 to 0.068 mg/L, with a mean of 0.048 mg/L (573). The median plasma level of PCP in 18 workers in a PCP processing factory, with 12 years mean exposure, was 0.25 µg/L. The range of values was 0.02µg/L to 1.5µg/L (721). In an environment where the PCP level in the air ranged from 0.3 to 180 µg/m^3, 10 workers with 4 to 24 years exposure had serum concentrations of 38 to 1270 µg/L (722).

Urine Samples
The kinds of occupational exposures found in the wood preservation industry result in urine levels in the mg/L range, while the general population levels are in the µg/L range. Surveys of urine levels in people have been made in several countries. These are more commonly measured and reported than plasma levels and vary widely depending upon occupational exposures and background levels. Six Hawaiian pest control operators with chronic exposure to PCP had levels of 10 to 36 mg/L, but expressed no obvious symptoms (24). Post-mortem samples of PCP overdose victims showed levels of 28 to 96 mg/L (30). Workers in a PCP plant were checked monthly for serum and urine levels. Urine levels in the workers ranged form 0.041 to 0.761 mg/L, with a mean of 0.164 mg/L, while controls had only 0.0034 mg/L (573). The median urine level of PCP in 18 workers in a PCP processing factory, with 12 years mean exposure, was 125 µg/L. The range of values was 13µg/L to 1244µg/L (721). In an environment where the PCP level in the air ranged from 0.3 to 180 µg/m^3, 10 workers with 4 to 24 years exposure had urine concentrations of 8 to 1224 µg/L (722).

People in the USA. who were not exposed as part of their jobs had PCP levels in their urine which ranged from 1 to 193 µg/L (22). Over 400 urine samples from the general public were analyzed for PCP with a detection limit of 0.005 mg/L. The maximum value found was 0.193 mg/L, the mean value 0.0063 mg/L and 84.8% of the samples had detectable PCP levels (127, 571). One sample of 117 people had a mean of 40 µg/L with a range of <1 to 1800 µg/L and another sample of 173 people had a mean of 44 µg/L with a range of 3 to 570 µg/L (24). In Britain, six people ranged from 2 to 11 µg/L (26). In Japan 20 people ranged from 10 to 50 µg/L (25), and 60 students at a Florida University had a mean of 20 µg/L with a range of 9 to 80 µg/L (27).
3.5.5 SEDIMENTS

Chlorophenols are preferentially adsorbed onto organic particles in the water, and organic sediments become repositories (220); much of the PCP will remain on the sediments but the other chlorophenols will tend to become distributed throughout the other environmental compartments. Table 3.5.2 gives some sediment levels of PCP and TTCPs for several British Columbia sites in 1978. These levels varied from 5 to 270 µg/kg dry weight (87). In the lower Fraser River sediment, PCP and TTCPs (mostly 2,3,4,6-TTCP) varied from less than 3 to 5 µg/kg dry weight in 1987. These were lower levels than those found in 1985 in a similar survey, and much lower than the 1978 data in Table 3.5.2, indicating less use of chlorophenols, better waste management, or deposition of fresh, less contaminated sediments on top of the older sediments. Sediment size also affects the adsorption and chlorophenols do break down, so the net level measured is a function of breakdown and deposition rates (537). It appears that the concentration of chlorophenols in surface sediments is decreasing in BC Table 3.6.1 gives summaries of the chlorophenol levels found in the sediments of the Fraser Estuary between 1973 and 1987 (693).

Chlorophenols (92% PCP and 8% TTCPs) were sampled in the sediments of railway and utility right-of-way ditches of the lower mainland of BC, ditches which flowed to salmon bearing streams. The sediments adjacent to poles in utility right-of way ditches contained a mean of 139 mg/kg and sediments 4 m downstream contained a mean of 0.3 mg/kg. Sediments adjacent to poles in railway right-of way ditches contained a mean of 49.7 mg/kg and 0.4 mg/kg downstream. Sediments at the base of utility poles had a mean value of 2168 mg/kg and soils next to railway ties averaged 38.6 mg/kg. These are all expressed on a wet-weight basis; the mean moisture content was 35 % with a range of 21 to 57 (719).

TTCPs

In British Columbia, sediment levels of TTCPs in excess of 100 µg/kg were found in 1979 and up to 63 µg/kg was found in 1986.

2,3,4,5-TTCP and **2,3,5,6-TTCP**

PCP is broken down by sunlight to form 2,3,4,5-TTCP and 2,3,5,6-TTCP, which persist in sediments (201).

2,3,4,6-TTCP

The concentration of 2,3,4,6-TTCP in sediments of a contaminated lake in Finland was 33.4 to 50.1 µg/kg dry weight (599).

PCP

Sediment levels of PCP are below the µg/kg detection limits in uncontaminated background areas, but rise to mg/kg levels in chronically contaminated areas (8, 91, 201, 289, 291, 299, 300). In heavily industrialized areas, such as around the Great Lakes, PCP levels are generally high and persistent in lake sediments (13, 103, 201, 212), soils (213) and leaf litter (13, 201). In British Columbia sediment levels of PCP in excess of 100 µg/kg were found in 1979 and 107 µg/kg was found in 1986. Table 3.5.2 gives water and sediment levels of PCP and TTCPs for several British Columbia sites. These sites are generally just downstream from, or near, wood treatment plants, and subject to run-off.

The migration of PCP from treated power poles into the surrounding soil showed a gradient with a mean value of 658 mg/kg adjacent to the pole, 3.4 mg/kg at 30 cm from the poles and 0.26 mg/kg at 150 cm.

This latter value is not significantly different from background in industrialized areas. Migration is minimal and biodegradation keeps PCP levels in the environment down (191).

The leaching of PCP from 100 utility poles in the USA was studied and showed tremendous variability both at a site and between sites. Generall surface samples had higher levels than subsurface samples and PCP concentrations dropped rapidly with distance from the pole. The maximum PCP concentration found ranged from 0.14 to 1500 mg/kg but the maximum at most poles was under 100 mg/kg. The mean maximum found was 190 mg/kg (± 340 mg/kg) and the median was 34 mg/kg. Samples collected at all depths and 3, 8, 18 30 and 48 inches from the pole, had 55%, 80%, 92%, 95% and 95% of the PCP concentrations under 1 mg/L. All but 3 samples collected more than 3 inches from the poles had PCP values under 500 mg/L and those collected over 8 inches from the poles had PCP values under 50 mg/kg (723).

3.5.6 Organisms

Table 3.5.4 gives the levels of various chlorophenols in biota from the Fraser River (269, 720). In most biota, PCP levels were below the 1 to 10 µg/kg wet weight level; near wood treatment facilities levels were relatively high (289, 305). Fish upstream in the Fraser River had TTCPs and PCP at the detection limits of 1 to 20 µg/kg; in the lower reaches where contamination from industrial effluent occurred, levels reached 40 to 90 µg/kg except for sculpins, which had a mean of 50 and a peak, near treatment facilities, of 100 µg/kg (87). Chlorophenols were also found in the Fraser River near Prince George and Quesnel, and in the livers of mountain whitefish, *Prosopium williamsonii*, and largescale suckers, *Catostomus macrocheilus*, which live in this reach. Juvenile chinook salmon also overwinter in this reach of the Fraser River and were exposed to high levels of PCP during winter low flows in the river (203). Table 3.6.1 gives summaries of the chlorophenol levels found in the organisms of the Fraser Estuary between 1973 and 1987 (693). The data in this section is given on a wet weight basis.

DCPs

Only large fish in the lower Fraser River, BC, had measurable DCP levels (262). In Canagagique Creek, Ontario, high levels of 2,6-, 2,4- and 3,4-DCP were found in fish; up to 1693 µg/kg (302). In the Weser estuary and German Bight, Europe, the polychaete, *Lanice conchilega*, contained 11.8 µg/kg of 2,4- and 2,5-DCPs (641, 642, 643).

2,4-DCP

Marine organisms near Saint John, NB, living in waters receiving pulp mill effluent, contained the following amounts of 2,4-DCP, as µg/g of lipid, measured as methyl ethers, in the specified tissue; DCP recoveries were only about 45% (8).

Maya arenaria

(clam, body), detectable

Crangon septemspinosa

(Sand shrimp, body), detectable

Pseudopleuronectes americanus

(Winter flounder, muscle), detectable, 2.5, 3.7
(Winter flounder, viscera, skin & fat), detectable,
(Winter flounder, liver), 0.7

Alosa pseudoharengus

(Gaspereau, muscle), detectable, (liver), 0.29

Alosa sapidissima

(Shad, liver), detectable, 0.52

Osmerus mordaz

(Smelt, muscle), 1.1, 9.0
(Smelt, viscera, skin & fat, liver), detectable

Acipenser oxyrhynchus

(Sturgeon, liver), 0.37

Microgadus tomcod

(Tomcod, muscle), detectable, 3.73, 2.0
(Tomcod, viscera, skin & fat), detectable
(Tomcod, liver), 0.74

TCPs

TCPs were measured in the Lower Fraser River fish in 1988, with a detection limit of 1 µg/kg of fish muscle tissue. TCPs were not detected in largescale suckers from the main arm, threespine stickleback from the north arm and peamouth chub and staghorn sculpins from the main and north arms. In the main stem, mean values for peamouth chub were 31 µg/kg, for northern squawfish 13 µg/kg, for largescale suckers 11 µg/kg, and for redside shiners 38 µg/kg. In the north arm values were 12 µg/kg for largescale suckers, 38 µg/kg for northern squawfish, and 28 µg/kg for starry flounder (535).

TCPs were rarely found in fish tissues from the Fraser River (262). In 1987 measurements were made of trichlorophenols in lower Fraser River benthos. The wet weights found were 80 µg/kg in amphipods, 100 µg/kg in other crustaceans, 20 to 60 µg/kg in chironomids, 40 to 400 µg/kg in pelecypods, 200 µg/kg in leeches, <20 to 80 µg/kg in lampreys, 60 to 2000 µg/kg in oligochaete worms and 30 to 2000 µg/kg in polychaete worms (537).

2,4,5-TCP

In the Weser Estuary and German Bight the polychaete worm, Lanice conchilega, contained 19.3 µg/kg of 2,4,5-TCP (641, 642, 643).

2,4,6-TCP

In contaminated lakes in Finland, reported tissue levels of 2,4,6-TCP include: 13.6 to 17.3 µg/kg in pike, 4.67 to 55.9 µg/kg in roach, 1.44 µg/kg in clams, 4.96 to 6.86 µg/kg in sponge and 2.45 µg/kg in plankton (599). In the Weser Estuary and German Bight the polychaete, Lanice conchilega, contained 26 µg/kg of 2,4,6-TCP (641, 642, 643). Marine organisms near St. John N.B., living in water receiving pulp-mill effluents, contained the following amounts of 2,4,6-TCP, as µg/g of lipid, measured as methyl ethers in the tissue specified. TCP recoveries were only about 40% from the columns (8).

Mya arenaria

(clam, body), 0.123 to 0.56;

Crangon septemspinosa

Sand shrimp, body), 0.74;

Pseudopleuronectes americanus

(Winter flounder, muscle), 0.12 to 1.85
(Winter flounder, viscera, skin & fat), 1.41
(Winter flounder, liver), 3.48

Alosa pseudoharengus

(Gaspereau, liver), 0.02

Alosa sapidissima

(Shad, liver), 0.017 to 0.027

Osmerus mordax

(Smelt, muscle), 0.25 to 0.43
(Smelt, viscera), 2.3
(Smelt, skin & fat), 0.67
(Smelt, liver), 0.062

Microgadus tomcod

(Tomcod, muscle), 0.33 to 2.29
(Tomcod, viscera), 3.8

(Tomcod, skin & fat), 2.1
(Tomcod, liver), 0.39

Acipenser oxyrhynchus

(Sturgeon), 0.028

TTCPs

In the southwestern BC area starry flounder tissues contained 0.19 to 2.52 mg/kg of TTCPs; near wood treatment facilities TTCP levels in tissue may reach 20 µg/kg in crabs and 690 µg/kg in mussels (87). A spill at Elk Falls, Campbell River, caused tissue levels of TTCPs of 3530 µg/kg in the invertebrates and 800 µg/kg in the algae (305). Sculpins in the lower Fraser River near treatment facilities had liver levels of TTCPs of 1600 µg/kg (87). TTCPs were measured in fish from the lower Fraser River in 1988. The detection limit was 1 µg/kg dry weight of fish muscle tissue. Tetrachlorophenols were not detected in largescale suckers from the north arm, northern squawfish from the mainstem, starry flounder from the main arm, staghorn sculpins and threespine sticklebacks from the north arm, and peamouth chub living at any site. In the mainstem, largescale suckers had 6 µg/kg and redside shiners 50 µg/kg. Main arm levels were 15 µg/kg in largescale suckers and staghorn sculpins and 76 µg/kg in northern squawfish. In the north arm, northern squawfish had 33 µg/kg and starry flounders 1 µg/kg (535).

Levels of TTCPs in lower Fraser River benthos in 1987 were below 20 µg/kg in amphipods and crustaceans, 10 to 100 µg/kg in chironomids, <20 to 1500 µg/kg in pelecypods, 500 µg/kg in leeches, 20 to 200 µg/kg in lampreys, 200 to 3000 µg/kg in oligochaete worms and 200 to 1000 µg/kg in polychaetes (537).

In the Weser Estuary and German Bight, the polychaete worm, Lanice conchilega contained 67 µg/kg of 2,3,4,6- and/or 2,3,5,6-TTCP (641, 642, 643).

2,3,4,5-TTCP

In the Weser Estuary and German Bight the polychaete worm, *Lanice conchilega* contained 7 µg/kg 2,3,4,5-TTCP (641, 642, 643).

PCP

The levels of PCP in fish are generally high and persistent in the Great Lakes (201, 212) and in industrialized areas generally. Starry flounder tissues in southwestern B .C. contained 0.77 to 2.77 µg/kg PCP on a wet weight basis (87). Near wood preservation facilities the PCP tissue levels may reach 17 µg/kg in crabs, 20 µg/kg in mussels, and 1700 µg/kg in polychaete worms (87, 303). After a PCP spill in Surrey, the tissue levels in Boundary Bay organisms reached 67 to 116 µg/kg in crabs, 171 to 563 µg/kg in oysters and 83 to 108 µg/kg in clams. These levels dropped below detection in 3 months (304). A spill at Elk Falls, Campbell River, caused PCP level of 4560 µg/kg in invertebrates and 3330 µg/kg in algae (305).

Sculpins in the lower Fraser River near treatment facilities had liver levels of PCP of 2100 µg/kg (87). PCP was measured in fish from the lower Fraser River in 1988. The detection limit was 1 µg/kg dry

weight of fish muscle tissue. PCP was not detected in threespine stickleback from the north arm. Mean values in the mainstem were 8 µg/kg for largescale suckers, 10 µg/kg for northern squawfish and peamouth chub, and 19 µg/kg for redside shiners. In the main arm largescale suckers had 6 µg/kg, northern squawfish and staghorn sculpins 4 µg/kg and starry flounders 7 µg/kg. North Arm fish contained 3 µg/kg in largescale suckers, peamouth chub and staghorn sculpins, and 11 µg/kg in northern squawfish (535).

PCP measurements made in 1987 on lower Fraser River benthos found, on a wet weight basis, <20 µg/kg in amphipods, 50 µg/kg in crustaceans, 20 to 300 µg/kg in chironomids, 20 to 2500 µg/kg in pelecypods, 800 µg/kg in leeches, 100 to 3200 µg/kg in lampreys, 30 to 4200 µg/kg in oligochaete worms and 200 to 1000 µg/kg in polychaete worms.

A survey of PCP in a New Brunswick estuary indicated low levels of PCP in almost all samples taken. Levels ranged from 10.8 µg/kg (wet weight) in white shark livers to 0.36 µg/kg in double-crested cormorant eggs (31).

Marine organisms near St. John, N.B., living in pulp mill effluent receiving waters, contained the following PCP levels in the specified tissues, measured as µg/g lipid as methyl ethers (8).

Mya arenaria

(clam, body), 0.43 to 2.3

Crangon septemspinosa

(Sand shrimp, body), 2.4

Pseudopleuronectes americanus

(Winter flounder, muscle), 1.63 to 7.9
(Winter flounder, viscera, skin & fat), 0.49
(Winter flounder, liver), 1.3

Alosa pseudoharengus

(Gaspereau, muscle), 0.82
(Gaspereau, liver), 0.22

Alosa sapidissim

(Shad, liver), 0.58 to 0.81

Osmerus mordax

(Smelt, muscle), 4.04 to 5.6
(Smelt, viscera), 0.75

(Smelt, skin & fat), 0.35
(Smelt, liver), 1.44

Acipenser oxyrhynchus

(Sturgeon, liver), 0.26

Microgadus tomcod (Tomcod, muscle), 0.43 to 5.36

(Tomcod, viscera), 0.75
(Tomcod, skin & fat), 1.0
(Tomcod, liver), 0.17

In the Weser Estuary and German Bight, PCP levels reached 117.5 µg/kg in the polychaete worm, *Lanice conchilega*, and 4.6 µg/kg wet weight in the actinian, *Sagartia troglodytes* (641, 642, 643). In Surinam, NaPCP was used at 3.5 to 4.0 kg of 85% active material per 20 L of water to control water snails, *Pomacea glouca*. Snail tissue levels reached 36.8 ng/kg wet weight. Dead frogs, *Pseudis paradoxa,* and three species of fish, all dead, from the rice fields had 8.1 ng/kg, 31.2 ng/kg, 41.6 ng/kg and 59.4 ng/kg of PCP on a wet weight basis, respectively. Live fish of the same species from nearby unsprayed ditches contained 1.77, 8.76 and 13.4 ng/kg of PCP. These snails were a major food component of several bird species which frequented the rice fields (644). Fish from the Bay of Quinte on the north shore of Lake Ontario contained >200 µg/kg of PCP on a whole fish, wet weight basis (8). Lake Superior fish had whole fish PCP concentrations of 0.1 to 1 mg/kg in *Salvelinus namaycush* (lake trout) and *Salvelinus namaycush siscowet* (fatty lake trout), and 0.02 to 0.60 mg/kg in *Coregonus clupeaformis* (lake whitefish) (8). Cows housed in a barn constructed partly of PCP treated wood, had blood PCP levels from 270 to 570 µg/kg. PCP was found in bone marrow, fat, serum and liver (645, 646, 647, 648).

In 1980, 10 lake trout from the eastern basin of Lake Ontario (Main Duck Island) had PCP levels below the detection limit of 0.5 µg/kg wet weight, as did six lake trout from the western basin (Port Credit). The PCP levels in five of the western trout were 2, 3, 5, 10 and 11 µg/kg wet weight (686). In 1979 young spot-tail shiners from Niagara-on-the-Lake and Centre Creek, Lake Erie had maximum levels of PCP and 2,4,6-TCP of 28 and 33 µg/kg wet weight, respectively, on a whole fish basis. The maximum level of 2,4,5-TCP at Niagara-on-the-Lake was 22 µg/kg (687).

3.6 Summary of the Dissemination of the Chlorophenols

Table 3.6.1 gives summaries of the chlorophenol levels found in the organisms, water, and sediments of the Fraser Estuary between 1973 and 1987 (693). Chlorophenols are used as broad spectrum biocides world-wide; residues and breakdown products are ubiquitous in air, water, sediment, and organisms. The major use has been as an anti-sapstain fungicide in the cut lumber industry, but this use is rapidly being phased out. They are also used as antiseptics and organic feedstocks for pesticide manufacture. Many commercial products contain chlorophenols (Table 3.2) and they are also used as preservatives in packaging materials. They are generally made by chlorinating phenols and by hydrolysis of chlorobenzenes. Dioxins were common byproducts of their manufacture, especially at high temperatures and using hydrolysis processes.

Tables 3.3.1 to 3.3.5 give contaminant levels in commercial chlorophenol products. It can sometimes be difficult to decide whether an effect on biota is due to the contaminant or the chlorophenol. The chlorination of wastewater with high organic loads, such as sewage, leads to chlorophenol production at low levels; this practice is widespread. The major sources are wood treatment facilities and pulp and paper mills; effluents and spills from these plants are responsible for a number of fish kills. Fly ash from incinerators, power stations, fireplaces, and slash and forest fires also distribute chlorophenols widely in the environment.

4. THE FATE OF CHLOROPHENOLS IN THE ENVIRONMENT

Once released into the environment, chlorophenols are subjected to a number of degradative processes. There are physical and chemical processes such as photodegradation, oxidation, hydrolysis, evaporation/volatilization and sorption, and biological processes such as uptake, breakdown and utilization. These all work with varying degrees of efficiency and speed, under different environmental conditions, to reduce chlorophenols to CO_2, H_2O and chloride, in times ranging from days to months. There are a great many different and complex organic compounds which may be intermediates in these breakdown processes. The nomenclature and structures of some classes of these intermediates are shown in Figure 4.1.

In biologically active, buffered, lake water at pH 7.0 and 25C, 2,4-DCP added at 100, 500 and 1000 µg/L was degraded by 56% in 6 days; all of the 100 µg/L concentration was gone in 9 days. If the water was unbuffered and anaerobic, with high organic levels, then 2,4-DCP persisted for 43 days (345). Soil degradation rates of PCP are primarily a function of organic matter content; cation exchange capacity and pH are less relevant. Soil texture, clay content, base saturation and iron oxide were not correlated with degradation rates. Microbial action is the major cause; no degradation occurs without organics being present (606). The primary products of PCP degradation are 2,3,4,5-TTCP, 2,3,6-TCP and 2,4,6-TCP; other products include 2,3,4,6-TTCP, 2,3,5,6-TTCP, 2,3,5-TCP, 2,3,4-TCP and 2,4,5,-TCP (606, 53). Table 4.1 gives a summary of the fate of chlorophenols in water. This table is adapted from McKee et al. (220).

4.1 Physical and Chemical Processes

Photodegradation is only important in shallow water and in high insolation areas. Oxidation under natural conditions is not significant, nor is hydrolysis. Evaporation and volatilization are only important where there is shallow water and vigorous mixing . High temperatures also speed up the process. Adsorption to sediment or suspended particulate matter is an important process for chlorophenols in nature and most chlorophenols introduced into the environment will be found, eventually, in the sediments.

4.1.1 Photodegradation

Photodegradation of chlorophenols depends upon the absorption spectrum of the compound; at sea level, sunlight does not contain wavelengths shorter than 290 nm. Since they are weak acids, chlorophenols exist as non-dissociated molecules or as anionic phenolates, depending upon the pH. The absorption spectrum is different for each species; ionized moieties are more susceptible to

photodegradation than are the neutral molecules (337, 338, 339). The first step in the process is to split the C-Cl bond to form an alcohol, C-OH.

In water of any depth, or with high turbidity or color, the energetic short wavelengths are all absorbed in the upper portion of the water column; photolysis is only a surface phenomenon. The rate may be significant below 280 nm, and/or in alkaline solutions, but is small in waters where the pH is neutral or acidic, and at longer wavelengths.

2-MCP

Molecular 2-MCP is converted to pyrocatechol; anionic 2-MCP to cyclopentadienoic acid dimers (335).

3-MCP

Molecular and anionic 3-MCP is converted to resorcinol (335).

4-MCP

Anionic and molecular 4-MCP is transformed to hydro- and benzo-quinones, trihydroxybenzenes and dihydroxybiphenyls (335).

2,4-DCP

Photolysis is a major transformation process for 2,4-DCP in estuarine waters (242), and occurs readily (221, 210), but is much slower in the winter. The breakdown of 2,4-DCP is more complex than that of the monochlorophenols and leads ultimately to chlorinated dimers (335).

Tests using a 326 mg/L solution of 2,4-DCP, 5 hours/day of noon sunlight for 10 days, or a mercury arc lamp at 254 nm for 5 to 12 hours, and 20 to 25C temperature, gave 50% losses in 5 minutes at pH 7 in the lab. The decomposition pathway was presumed to be 4-chlorocatechol or 4-chlor-1,2-diol-benzene to 1,2,4-benzenetriol and thence to hydroxybenzoquinone and polymeric humic acids (518). At 42.4 mg/L 2,4-DCP in 90 mL, 4 mm deep vials, using a 600 watt mercury discharge lamp, the time to achieve 50% decomposition was pH dependent. It was 34 minutes at pH 4, 4 minutes at pH 7 and 2 minutes at pH 9. At pH 7 with an initial concentration of 36 mg/L, only 12% was left after 80 minutes (345).

2,4,5-TCP

Photolytic degradation of 2,4,5-TCP is fairly rapid and the suggested pathway is to 2,5-DCP or 2,4-dichloro-resorcinol. These then go to 4-chlororesorcinol and ultimately to humic acids (493). In estuarine waters photolysis is a major transformation process for 2,4,5-TCP (242), occurring very readily (221, 210), but much reduced in the winter.

2,4,6-TCP

In the presence of an electron acceptor, 2,4,6-TCP can be broken down to 2,6-dichlorophenoxyl semiquinone radical ions; 66% in 17 hours under UV light at 290 nm (597).

2,3,4,5-TTCP

Photoreactions of 2,3,4,5-TTCP, at 285 nm in water and acetonitrile, occur through reductive dechlorination. The 4-position is photo-labile, but 3- and 5-position chlorines are not photoreactive. The degradation of 2.3 mg/L of 2,3,4,5-TTCP was 30% complete in 6 hours and 73% after 24 hours. There was 10% 2,3,5-TCP in the products after 6 hours and 9% 2,3,5-TCP after 24 hours (457, 524).

2,3,4,6-TTCP

Six hours of irradiation at 285 nm destroyed 51% of the initial 1.2 g/L of 2,3,4,5-TTCP. The breakdown products were pH and time dependent but included 2,3,4- and 2,3,6-TCPs at about the one percent level. The 4 and 6 chlorines were photo-labile, but not the 3 position chlorine (457).

PCP

Photolysis is a major transformation process for PCP in estuarine waters (242) and occurs very readily (221, 210, 473), but is much reduced in the winter months. In the clear, shallow waters of Transvaal, PCP is a poor molluscicide, but in Egyptian snail-infested, turbid, irrigation ditches, PCP works very well (210). This is because the PCP is rapidly degraded by sunlight in the clear waters. Aqueous solutions of PCP are degraded by sunlight or laboratory ultraviolet lamps; 90% of the starting concentration is broken down in 10 hours at pH 7.3, but only 40% in 90 hours at pH 3.3 (20, 221). A residence time of 2 days is estimated for lakes, where PCP can be degraded by photolysis to 0.1% of the original concentration (321). The products include chlorinated phenols, catechols and benzoquinone; tetrachlororesorcinol and tetrachlorohydroquinone; dichloromaleic acid (289), and octachloro-dibenzodioxin (294, 295, 296, 221).

Under laboratory conditions, decomposition of PCP in aqueous solution by sunlight is rapid. A starting concentration of 9.3 mg/L went to 0.4 mg/L in 24 hours and nearly to zero in 48 hours. In effluents and natural waters, other compounds screen out much of the UV. In a rice paddy, 2.4 mg/L PCP dropped to zero in the water in 7 days and from 4 mg/L to 2 mg/L in the soil. In waste from a wood treatment plant, PCP went from 30 mg/L to 0.2 mg/L in 7 days.

The products of solar photochemical breakdown include tetrachloro-p-benzoquinone, 3,4,5-trichloro-6-(2-hydroxy-3',4',5'-tetrachlorophenoxy)-0-benzoquinone, and tetrachlororesorcinol. Dechlorination to 2,3,4,6- and 2,3,5,6-tetrachlorophenols is followed by hydroxylation to 3,4,5-6-tetrachlorocatechol, tetrachlororesorcinol and 2,3,5-6-tetrachlorohydroquinone. Further irradiation yields dichlorocyclopentanedione and 2,3, dichloromaleic acid. Ultimate products are H2O, CO2 and HCl (191). Irradiating NaPCP at high concentrations in aqueous solution leads to formation of octachlorodibenzo-P-dioxins (205). The approximate 1/2 life for PCP undergoing photolysis during the summer in California is 1.5 days (16).

In a 21 day photolysis study in water between 7.5 and 36.3 degrees centigrade, PCP degraded rapidly under natural sunlight with half-lives of 20, 16.3 and 13.6 minutes at pHs of 5, 7 and 9 respectively. The degradation products included tetrachlorohydroquinone, tetrachlorocatechol, tetrachlororesorcinol and dichloromaleic acid or anhydride (725). A 30 day soil photolysis study on sandy loam at v24 degrees centigrade was conducted under alternating 12 hour light and dark cycles. No degradation occurred in the dark phase. The half life in the light was 37.5 days based on first-order degradation kinetics (726). Vapour phase PCP was seen to degrade under simulated sunlight in alternating 12 hour light and dark

periods with a half-life of 36.6 days in the light phase. Degradation products included 2,3,5,6-TTCP (727).

Photolysis is likely responsible for increasing TTCP: PCP ratios downstream from PCP effluents and in surface waters compared to deeper waters as the PCP is degraded to TTCPs (291). Photolytic half-lives of PCP are about 4 hours at pH 7.3 and 100 hours at pH 3.3. Light in the 290 to 330 nm range is effective at photolysis which proceeds most rapidly at high levels of light, pH and dissolved oxygen (221, 20, 210, 159). Since light attenuation occurs with depth, particularly UV attenuation, photolysis is primarily a surface phenomenon and 1 meter of depth has about the same effects as a drop of four pH units, or about 24 times the half-life.

4.1.2 Oxidation

There are few good data concerning chlorophenol oxidation. Chlorophenol decay is found in sterilized natural water under conditions where photodegradation and volatilization/evaporation are not likely. The disappearance rate is concentration dependent and increases with temperature. Phenols are susceptible to auto-oxidation and catalysis occurs on the surface of clay and silica in the presence of several metal ions (18, 19, 341, 342, 343, 344). Highly chlorinated organics are usually resistant to oxidation at temperatures well above normal aquatic-habitat ambient levels. Oxidation is not expected to be important in PCP breakdown in nature (473, 598, 340).

2-MCP and 4-MCP

Oxygen and hydroxyl radicals attack 2-MCP at the C-2 or C-4 sites to form 1,2- or 1,4-benzosemiquinones, and 4-MCP at the C-4 site to form 1,4-benzosemiquinone (335).

4.1.3 Hydrolysis

There are few data on chlorophenol hydrolysis, which is, in any case, an unlikely transformation in the environment. Aryl halides, especially those containing hydroxyls (which includes all chlorinated phenols), have low reactivity to halogen displacement or nucleophilic substitution (340). The covalent bond of a substituent on an aromatic ring is resistant to hydrolysis because of the high negative charge density of the aromatic nucleus. Making the pentachlorophenolate anion from hexachlorobenzene requires treatment with concentrated alkali at 130 to 200C. Further hydrolysis should require more extreme conditions and thus is not important under ambient conditions in aquatic habitats (473).

2,3,4-TCP, 2,3,5-TCP, 2,3,4,5-TTCP, 2,3,4,6-TTCP and **2,3,5,6-TTCP**

The hydrolysis half-lives of these five chlorophenols are about 3 years or more; hydrolysis is not an important natural transformation process (428, 457).

PCP

Hydrolysis is not a factor in PCP breakdown at pH 7 or 9 and is negligible at the lower pHs of 4 and 5. The calculated half lives, from 80 to 160 days, at these low pHs were likely due primarily to volatilization

losses at the 25 and 35 degree centigrade temperatures of the experiments, which included air flow over the solutions (728).

4.1.4 Evaporation / Volatilization

Evaporation is inversely proportional to water depth (336). TCPs, which are soluble and have low vapor pressure, do not generally volatilize from water. They are moderately acidic and will be ionized and solvated in natural waters. Volatilization is likely insignificant in natural environments (598). Volatilization is a function of temperature, water solubility, vapor pressure, solution mixing depth and molar concentration (607). Chlorophenols are quite persistent under ice cover in Canadian rivers during the winter (706).

2-MCP and 4-MCP

In stirred solutions the half-life of 2-MCP at 0.38 cm depth is 1.5 hours; at 1 m depth the half-life is 15 days. For 4-MCP the values are 13 hours at 0.38 cm depth and 4.5 days at 3 cm depth (336).

2,4-DCP

The volatilization half-life of 2,4-DCP at 64 to 100% relative humidity, was about 1 month at 30C (510).

2,3,4-TCP and **2,3,5-TCP**

The Neely 100-day partitioning pattern for 2,3,4-TCP and 2,3,5-TCP is air-68.45%, water-12.15%, ground-10.03% and hydrosoil-9.37%. Chemicals with these properties are expected to vaporize rapidly from, and not persist in, open water (428).

2,4,6-TCP

The volatilization of 2,4,6-TCP from water and soils at 25C was measured as a percent 2,4,6-TCP per mL of water evaporated. For the first few hours, rates were about 1.5% for water and 1% for sandy soils, dropping to 0.1% for humic soils (462). Volatilization is not a significant process in nature (598).

2,3,4,6-TTCP and **2,3,5,6-TTCP**

The Neely 100-day partitioning pattern for 2,3,4,6-TTCP is air-63.95%, water-6.31%, ground-15.38% and hydrosoil-14.36%. For 2,3,5,6-TTCP the respective percentages are air-23.86%, water-28.23%, ground-24.78% and hydrosoil-23.13%. Chemicals with these partitioning coefficients, and a log Ko/w value around 4.5, would be expected to vaporize rapidly from open water and accumulate in the soil (428).

PCP

The volatilization rates of PCP from water, sand, loam and humus were 2.57%, 0.13%, 0.31% and 0.10% in the first hour, and 2.11%, 0.12%, 0.15% and 0.12% in the second hour. These rates are expressed as the percent of applied PCP per mL of water evaporated (581). Volatilization decreases abruptly with increasing pH. At pH 8 virtually no volatilization occurs. The half-life declined from 130 days

at pH 6, to 6 days at pH 4 (607).
4.1.5 SORPTION

All the MCPs adsorb to about the same extent; increasing the number of chlorine atoms results in increased sorption, likely due to extra hydrogen bond formation (246). Sorption in river conditions gave concentration ratios of 117 for suspended matter and 440 to 650 for sediments, when water concentrations were 1.8 to 10.0 µg/L (348, 349). Desorption is slower than sorption and is not 100%; a fraction is irrevocably held by the sediment particles. Sediment levels may thus remain high longer than water levels (347), but this gives microorganisms, which are more densely aggregated on sediments, a greater opportunity to break down the chlorophenols. Adsorption is a function of the partition coefficients of the chlorophenols between the water and the solid phase, sediments or suspended particulates (707, 708, 709).

Chlorophenols are strongly adsorbed by activated carbon at the µg/L level, which is around the odor threshold for most chlorophenols. Adsorption is a function of pH (707, 708, 709); neutral species, which predominate below the pKa, are adsorbed more strongly than anionic species. While increasing the number of substituted chlorine atoms increases adsorption, it also lowers the pKa. The competition for adsorption sites among a group of chlorophenols is thus pH dependent, and those whose pKa is higher than the given pH, will be preferentially adsorbed, displacing those chlorophenols whose pKa values are lower than the ambient pH (506).

Sorption is generally a function of the organic matter in the sediment (707, 708, 709); clays are not as effective. Soil moisture levels do not affect adsorption (507, 508). When natural ponds were dosed with PCP at a total application of 0.3 mg/L, the concentration in the water decreased rapidly and exponentially. Sediment levels rose slowly for 28 weeks then slowly declined. Leaching into surrounding soils did not occur (583). The sorption of some chlorophenols by Bentone 24 and Bentone 18C, Wyoming Bentonite Clays is shown in Table 4.1.5.1

2,4-DCP

Sediment enrichment factors for the Rhine River were 440 times the water concentration, and sediment concentrations were generally in the µg/kg dry weight range (349).

2,6-DCP

Sediment enrichment over the water column in the Rhine River is 20 times (349).
TCPs

All six TCP isomers were measured in Rhine River mouth, 13 water samples and 17 sediment samples. TCPs accumulate in the sediments with various enrichment factors from five to 61 as shown in Table 4.1.5.2 adapted from reference (349).

2,3,4-TCP

In Nebraska sandy loam soil this compound is tightly bound and some irreversible binding may occur based on the high Ko/c values obtained (730).

2,4,6-TCP

In a contaminated lake in Finland, 2,4,6-TCP reached 10.4 to 17.2 µg/kg dry weight in the sediments (599).

TTCPs

All three TTCP isomers were measured in water and sediments from the mouth of the Rhine River. There were 13 water samples and 17 sediment samples. TTCPs accumulated in the sediments with enrichment factors of 35 to 445 as shown in Table 4.1.5.2, adapted from reference (349). As expected from the log Ko/w values of TTCPs compared to TCPs, the mean enrichment of TTCPs is greater than that of TCPs.

2,3,4,5-TTCP

A log Ko/c value of 3.84 indicates a high potential for partitioning to soil. The Neely 100-Day Partitioning Pattern for 2,3,4,5-TTCP is air-39.66%, water-6.05%, ground-28.08% and hydrosoil-26.21%. With a log Ko/w of 4.7, a chemical with these properties would not persist in water; most would end up in soils and sediments (428). In Nebraska sandy loam soil this compound is tightly bound and some irreversible binding may occur based on the high Ko/c values obtained (730).

2,3,4,6-TTCP

The log Ko/c, a calculated value of 3.69, indicates that this compound will bind to soil (428).

2,3,5,6-TTCP

A log Ko/c value of 3.45 indicates a good potential for this compound to partition to soil (428). The Neely 100-day partitioning pattern for 2,3,5,6-TTCP is air-23.86%, water-28.23%, ground-24.78% and hydrosoil-23.13%. Based on Henry's Constant, 0.00562, and a log Ko/w of 4.5, a chemical with these properties should vaporize rapidly from open water with large amounts partitioning to sediments (428).

PCP

PCP is sorbed on acidic soil primarily; little if any sorption occurs on neutral soil. The pH is the most important factor controlling sorption and the total amount sorbed is a direct function of organic content of the soil. Sediments and leaf litter retain high levels of PCP and serve as a sink for continual PCP input to the water. Rainfall both leaches PCP from soil and transports leaf litter to lakes (473, 580). PCP binds strongly to soil and extensive leaching experiments rarely desorbed over 10 to 20% of the amount present (13, 191). A study on the sorption and desorption of PCP in Georgia sandy loam, Ohio clay loam, California sandy loam and Nebraska blue sandy loam indicate that PCP is tightly bound or immobile in all but the California sandy loam where it is moderately bound, based on its Koc value (729). Enrichment factors in sediment over the water column ranged from 960 to 3600 in the Bay of Quinte, downstream from a wood preservation facility (291). In the lower Rhine River, PCP was found in all 17 sediment and 13 water samples. The mean sediment enrichment factor was 20 (340).

Organic material is a strong sink for PCP and the inhibition of degradation under the anaerobic conditions and low pH often found in such sediments leads to high PCP levels in anaerobic, low pH, high organic sediments such as are found in eutrophic conditions. The rate of PCP dissipation in soil is related to temperature, aeration and organic matter, partly dependent on cation exchange capacity and pH, and independent of texture, clay content, base saturation and free iron oxides. Degradation is usually slower in flooded or anaerobic soils than aerobic moist soils. Where the adapted microflora are anaerobes, and there are no adapted aerobic organisms, the reverse may occur. Anaerobic microorganisms use a different degradative pathway than the aerobes. Degradation is primarily by reductive dehalogenation to TTCPs, TCPs and DCPs. Methylation to the anisole also occurs (584).

4.2 Biological Processes

Microbiological breakdown is the major mechanism for removal of chlorophenols from the environment. In the absence of more readily metabolized compounds, some microorganisms use chlorophenols as their sole source of carbon for growth. Higher organisms also take up chlorophenols, accumulate them, detoxify them in various ways, and excrete chlorophenols and various breakdown products. All organisms are able to adapt to higher concentrations of chlorophenols over time, becoming better able to break them down as their metabolic processes adapt in response to the continued presence of chlorophenols. In higher organisms the adaptation is at the metabolic or enzymatic level; in bacteria, with their shorter life-cycles, the whole genetic pool of the population shifts so that chlorophenol metabolizing strains predominate. These adaptation processes take time and response to a first chlorophenol exposure is relatively slow, but once adapted, response to subsequent exposures is rapid.

4.2.1 Microbiological Breakdown

In spite of their extensive use as biocides, the chlorophenols are quite readily broken down by a wide variety of microorganisms, and used as a growth substrate by a number of bacteria. The addition of chlorine to phenol decreases the rate of biodegradation; the more chlorine added, the slower the degradation rate. Biodegradation may be a slow process which only occurs aerobically when other organic carbon sources are not available (184, 185), or may occur rapidly with half-lives up to days (709). Phenols substituted in the 2,4 and 6 positions are more readily degraded than 3-chloro or 5-chloro compounds (18, 19, 49, 159, 292, 293, 350). Monochlorophenols, particularly 3-MCP (18), are more resistant to degradation than many poly-chlorinated congeners (18, 49).

The sediment/water interface is the most important site of degradation since the responsible bacteria are generally attached to surfaces (297, 298). Adsorption to soil is pH dependent, more occurs at lower pH and in soils of high organic or humic content. Degradation rates increase with soil moisture and decrease with clay content (175). Degradation rates are a function of the conditions suitable for the growth of the responsible organisms (709). The aeration, temperature, pH, organic content, moisture level, nutrient level, light intensity and duration, and a cation exchange capacity conducive to microbiological growth, encourage the degradation of chlorophenols (48, 289). The optimum levels of these parameters are quite variable and depend upon the organism which is metabolizing the chlorophenol. In addition the concentration of the chlorophenol is also important. Toxicity occurs at high levels and at low levels other carbon sources may be preferentially metabolized.

The evidence for fungal and bacterial degradation of the chlorophenols is widespread (14 to 19, 21, 49 to 55, 434, 700). Many genera of bacteria have been shown to break down chlorophenols; these include,

Alcoligenes, Aeromonas, Azotobacter, Flavobacterium, Pseudomonas, Cytophaga, Corynebacterium, Arthrobacter, and Brevibacterium (21, 434). Additional organisms which break down PCP include Trichodera viride and Penicillium molds, Coriolus fungi and the peroxidase enzymes produced by some white rot fungi such as Phanerochaete chrysosporium. The chlorophenols are ultimately broken down to chloride and CO2 plus organic fragments, as indicated by work with isotopically labeled carbon. Ring cleavage occurs and the organic fragments are ultimately processed to free CO2.

Microbiological breakdown ensures non-persistence in soil, sediment or sewage effluent (155, 191); only continuous input, at rates higher than degradation rates, makes it possible to find substantial quantities of chlorophenols in the aquatic environment where half-lives are days for MCPs and DCPs, weeks for TCPs and months for TTCPs and PCP (220). Much of the following discussion is tabulated in Table 4.2.1.

Table 4.2.1 shows the rates of degradation of some chlorophenols in sterile and non-sterile soils and indicates that most degradation is biological. It also gives degradation rates for some chlorophenols in sewage sludge under different pH and temperature regimes. One must remember that soil and sediment bacterial populations, which have not previously been exposed to chlorophenols, will show an appreciable lag phase between the application of chlorophenols and the emergence of significant breakdown products. Initially, very few cells will have the necessary metabolic machinery to survive in, and utilize, the chlorophenol. It takes time for these few cells to multiply and become a significant proportion of the bacterial population and also a significantly large number of cells in absolute terms. Once a large population of adapted cells exists, degradation will proceed rapidly. Subsequent additions of chlorophenols within a short period of time will not require this lag time before breakdown products begin to be seen (351, 352, 353, 354, 438, 710). The pelagic bacteria of oligotrophic lakes which have high humic levels, are an exception to the usual need for lag time. Bacteria living in such habitats already have enzymes adapted to degrading the aromatic compounds of humus and can degrade the structurally similar chlorophenols (700).

After adaptation of the microflora, which takes about 2 weeks, complete biodegradation occurs in only a few days. Using activated sludge from waste treatment plants, the lower chlorophenols are readily degraded, see Table 4.2.1, though at high concentrations of 20 to 100 mg/L toxicity occurs and activity is reduced (350, 351). With pre-adapted microflora degradation is rapid through to complete mineralization, CO2, H2O, and chloride, (185, 355, 356).

Most degradation studies only monitor the loss of the parent compound. Complete mineralization may not occur and then metabolites may accumulate in the environment. Due to losses from various sources, experimental results reported for nominal or starting concentrations are actually caused by lower levels of chlorophenols in solution. Thus, most published LC50 data are a little optimistic; the actual solute levels were a little lower than the nominal amount added.

2-MCP

Water from the Vistula River in Poland degraded 2 to 20 mg/L of 2-MCP in 2 to 13 days, after 2 to 3 weeks of adaptation. Adding adapted bacteria from a previous trial increased the rate of removal (351). In the water column of a small stream, degradation of 2-MCP at 100 mg/L was negligible in 40 days, even at 20C. In aerobic sediments at 0C and 4C, bacterial degradation of 2-MCP occurred more quickly than that of 3-MCP and PCP. Complete removal occurred in 10 to 15 days at 20C, and at the lower temperatures 78% was removed in 30 days (19).

Activated sludge will completely break down 100 mg/L of 2-MCP in 3 days; both ring cleavage and dechlorination occur (184). Anaerobic conditions delay biodegradation (18), but anaerobic sewage sludge degrades 50 mg/L in 3 weeks by a different biochemical pathway than that used in aerobic degradation. One mg/L of 2-MCP added to domestic sewage at 20C was not degraded in 20 to 30 days, presumably due to a lack of adapted bacteria in this medium. In polluted river water, degradation occurred in 15 to 23 days (315).

In soil, 250 mg/L of 2-MCP took 10 days to be reduced to 34%. Subsequent additions degraded at twice the rate. In a 7-day test, the rate of disappearance was twice as fast in unsterilized soil as opposed to sterilized soil (438). Oxidation of 2-MCP to 3 or 4-chlorocatechol by soil and bacteria was found for three strains of Norcardia, three strains of Pseudomonas and one strain of Bacillus and for Mycobacterium coeliacum (439). In shake culture 2-MCP at 50 µg/L was mixed with 4 g of two types of soil at 30C. Complete degradation occurred in 14 and 47 days for the two soil types (350).

The degradation rate of 2-MCP was studied at several concentrations in sludge pre-acclimated to 2-MCP. The concentrations used were 1, 10 and 100 mg/L in experiments run for 6 hours. Only 20% degradation occurred at 100 mg/L, but 97% degradation was observed at 10 mg/L. At the 1 mg/L concentration, 100% degradation occurred in 3 hours (440).

3-MCP

In aerobic sediments, at both 10C and 4C, bacterial degradation of 3-MCP occurred more slowly than that of 2-MCP, 4-MCP and 2,4-DCP. In the water column degradation was negligible, even at 20C. In sediments at 20C, 42% of 100 mg/L was degraded in 30 days (19). In a shake-culture experiment 4 g of soil in 100 mL of medium at pH 7.2 and 30C was inoculated with 3-MCP. When 50 µg/mL (50 mg/L) of 3-MCP was added to two different soils the time to complete degradation was >72 and >47 days (350). Degradation is very slow, if it occurs at all, in otherwise suitable conditions when acclimated bacteria are not present.

Chlorine substitutions in the "meta" position slow down the rate of degradation considerably (18, 350). Well-acclimated bacteria, given no other carbon source for several weeks, were able to degrade 100 mL /L in 2 days at 27C (355), but un-acclimated anaerobic sewage sludge took 7 weeks to completely degrade 50 mg/L (353).

In activated sludge 100% ring degradation and 100% chloride ion production was reported in 3 days after an initial inoculation of 100 mg/L of 3-MCP (184). The soil bacteria *Norcardia* -three strains, *Pseudomonas* -three strains, *Bacillus* -one strain and *Mycobacterium coeliacum* were able to oxidize 3-MCP to either 3- or 4-chlorocatechol (439). In activated sludge from treatment plants previously exposed to phenols, the degradation of 3-MCP in 6 hours was 100% for 1 mg/L, 40% for 10 mg/L and none for 100 mg/L (440).

4-MCP

Stream water did not appreciably degrade 100 mg/L of 4-MCP between 0C and 20C. In 30 days, the aerobic sediments removed all the 4-MCP at 20C and 60% of the 4-MCP at 0C, which was faster than the removal rate for 3-MCP or PCP (19). In river and pond water, bacterial transformation rates are about 0.007 µg/L /hour at a concentration of 1 mg/L (358). In Skidway Estuary water at 21C, the half-life of 25 µg/L 4-MCP was 20 days for complete mineralization; at 9C, no CO2 was evolved (352).

In North Sea coastal plankton communities, initial concentrations of 0.1 and 1 mg/L of 4-MCP disappeared in 5 and 14 days, respectively. Subsequent additions disappeared more rapidly indicating adaptation and biological degradation were responsible (353, 354).

The effect of 4-MCP on urease activity and the loss rate of 4-MCP by bacterial degradation were studied in a clay with 3% organic content, at pH 7.3. Starting with 500 µg of 4-MCP (50 µg/g in 10 grams of soil) urease activity decreased by 30%. After 3, 7 and 14 days the inhibition dropped to 21%, 10% and 3%. On loam soils, the initial depression of urease activity was 37% (447). Soil decomposition of 4-MCP was tested by pre-treating soil for 7 days with 13.3 mg/L 4-MCP, then adding 22.5 mg/L. In 23 days this dropped to 13.0 mg/L (438). In shake culture at 30C using 50 mg/L of 4-MCP and 4 g of soil, total loss times of 9 and 3 days were recorded for two soil types (350).

Activated sludge degraded 100 mg/L in 3 days. Ring degradation and chloride ion development, both to 100%, occurred in this time (184). Soil bacteria, three *Norcardia* strains, one *Pseudomonas*, one *Bacillus* and *Mycobacterium coeliacum*, oxidized 4-MCP to 3 or 4-chlorocatechol (439). In sewage sludge from plants with continuous incoming phenol levels of 0.1 to 0.35 mg/L for a year, there was 100%, 80% and 16% degradation of 1, 10 and 100 mg/L of 4-MCP, respectively, in 6 hours (440). In anaerobic sewage sludge, no significant mineralization of 50 mg/L occurred in 8 weeks (357).

2,4-DCP

In aerobic sediments, at both 0C and 4C, bacterial degradation of 2,4-DCP occurred more quickly than that of 3-MCP or PCP. In water at 20C, 2,4-DCP was broken down slowly, but not at 0C and 4C. The half-life of 100 mg/L in the sediment at 20C was 15 to 30 days, and in water at 0C, the test was terminated after 40 days, at which time no appreciable breakdown had occurred (19). Aerated and buffered lake water removed 0.1 mg/L in 9 days, and 0.5 to 1 mg/L in 30 days. The same rates of loss were found in North Sea water (354).

In shake culture at pH 7.2 and 30C, 50 µg/L of 2,4-DCP was completely degraded in 9 and 15 days by two types of silt loam soil (350). *Arthrobacter sp*. enzymatically dechlorinates 2,4-DCP completely in 4 hours (514). The soil fungus, *Aspergillis clavatis*, degrades 2,4-DCP at concentrations up to 10 mg/L. At higher concentrations growth is inhibited (515). The degradation of 2,4-DCP by microorganisms proceeds through catechol intermediates to succinic acid (441, 516, 517). In acclimated activated sludge, 2,4-DCP at initial concentrations of 50, 100, 200 and 400 mg/L dropped in 2 days at pH 7 and 26C, to 20, 25, 60 and 45% respectively. At 100 mg/L, 100% degradation occurred in 5 days (184).

2,5-DCP

In activated sludge there was 52% ring degradation in 4 days with an initial concentration of 100 mg/L. This was acclimated sludge at pH 7 and 26C (184). In shake culture with an artificial medium which had been previously sterilized, 50 µg/L 2,5-DCP was added along with 4 g of silt-loam soil. At 30C and pH 7.2, the 2,5-DCP persisted through to the end of the experiment at 72 days (350).
3,5-DCP

Adding 5 mg/L of 3,5-DCP to wastewater stimulated oxygen consumption rates; 25 and 50 mg/L depressed oxygen uptake. Sublethal growth effects, as determined by the fermentation tube method, were seen at 3 mg/L (1.3 to 5.0) and the LC50 was 11 mg/L (3 to 20). The onset of the inhibition of cell division in Pseudomonas occurred at 1.9 mg/L (1.25 to 2.50) (444).

2,3,4-TCP

Pseudomonas cepacia (AC1100), adapted to 2,3,4-TCP, dehalogenated the 2,3,4-TCP at a rate of 70% in 3 hours when it was present at up to 20 mg/L. When present at concentrations of 40 mg/L the degradation rate decreased (434).

2,3,5-TCP

Pseudomonas cepacia (AC1100), dehalogenates 2,3,5-TCP, but it is a slow process and concentrations above 0.2 mM, (39.5 mg/L) are toxic (434). At 30C *Pseudomonas* takes 100 hours to complete ring cleavage and breakdown of 2,3,5-TCP (424).

2,4,5-TCP

Microorganisms in soil suspensions convert 2,4,5-TCP to 3,5-dichlorocatechol, 4-chlorocatechol, succinate, chlorosuccinate, Cis,Cis-2,4-dichloromuconate and 2-chloro-4-(carboxymethelene) but-2-enolide (491). In shake culture at pH 7.2 and 30C, 50 µg/mL of 2,4,5-TCP was added to 4 g of each of two different types of silt loam soils. The time for complete disappearance was greater than 72 and 47 days for the two soils (492). With no adapted microorganisms present, chlorophenol breakdown can be very slow.

In an aeration lagoon where the effluents were maintained at 20 to 21C, 18.8 mg/L of 2,4,5-TCP was completely degraded in 7 days (315). In a freshwater nutrient medium, 70% biodegradation of 2,4,5-TCP occurred in 35 days starting with 1 mg/L; in a biological sewage treatment system, no degradation of 2,4,5-TCP was seen in 14 days (600, 601).

2,4,6-TCP

A gram-variable bacillus was isolated which could utilize 2,4,6-TCP as its sole carbon source. Growth occurred at 198 mg/L and in 84 hours all the 2,4,6-TCP was used and cell counts rose over 100 times. The minimum inhibitory concentration was over 400 mg/L. At a 2,4,6-TCP/cell ratio of about 400 µg/mg, and 150 minutes incubation, all the 2,4,6-TCP was used and 64% of the chloride had been released (51).

Slime organisms, similar to activated sludge bacteria, were grown on normal substrate and then switched to 2,4,6-TCP. In 3 days 75% was degraded as measured by chloride ion production (461). In bacterial cultures, starting with 300 mg/L, 95% degradation occurred in 7 to 10 days; starting with 100 mg/L, 70% degradation occurred in 3 hours (602).

Using an initial concentration of 1 mg/L of 2,4,6-TCP, a freshwater nutrient medium degraded 70% in 9 to 18 days (600). In soil culture, 5 to 13 days were needed for complete removal of 2,4,6-TCP, but in acclimated sludge, complete aromatic ring degradation occurred in 5 days (473). In flask cultures with sludge bacteria, it took 7 to 10 days to remove 95% of 300 mg/L of 2,4,6-TCP; at 100 mg/L, 70% was removed in 3 hours. Acclimated sludge can carry out complete aromatic ring degradation of 2,4,6-TCP in 5 days (184).

In soils, complete disappearance takes from 1 to 9 days (350). In soil suspension, 2,4,6-TCP disappeared completely in 5 days. Degradation by the parent strain of Pseudomonas took 120 hours at 30C for 100% ring degradation of 200 mg/L. A mutant strain took only 50 hours (424). In shake culture with two loam soils, 50 µg/L of 2,4,6-TCP was completely degraded in 5 and 13 days (350).

3,4,6-TCP

The growth of a gram-variable bacillus was supported only by 3,4,6-TCP and 2,3,4,6-TTCP; none of the other chlorophenols would support growth (603).

2,3,4,5-TTCP

Pseudomonas cepacia (AC1100), grown on 2,4,5-T as its sole carbon source, was able to dechlorinate 2,3,4,5-TTCP to a limited extent (434).

In a continuous-flow-sludge-blanket-reactor, 300 µg/L of 2,3,4,5-TTCP was dechlorinated, at the rate of 90 to 99% . Even at up to 600 µg/L, yeasts were present in the flocs but died after 5 days at 1 mg/L. Increasing the TTCP from 2.4 mg/L to 8 mg/L caused drastic decreases in the rate of chlorophenol removal. Bacteria identified included *Klebsiella oxytoca*, *K. pneumonia*, *Aeromonas hydrophila* and *Pseudomonas aeruginosa*. One of the yeasts was a *Candida sp*. (523).

In non-sterile soil, after 80 days at pH 7.1 and 23C, only 5% of the added 1 mg/mL of 2,3,4,5-TTCP was decomposed, and 31% decomposition occurred in 160 days. No decomposition occurred in sterile soil (18). A wastewater treatment biomass acclimated to PCP did not readily degrade 2,3,4,5-TTCP at 40 mg/L (458).In soil, 2,3,4,5-TTCP degrades to 2,3,5-TCP, 2,4,5-TCP, 3,4-DCP and 3-MCP (606, 17).

2,3,4,6-TTCP

Fungi and bacteria, extracted from litter in broiler houses, were grown in flasks and treated with 2,3,4,6-TTCP. Of the 116 isolates tested, 99 metabolized 2,3,4,6-TTCP; 68 produced 2,3,4,6-tetra-chloroanisol as a metabolic product. *Penicillium corylophilum* produced the highest amount of methylated product and removed the 2,3,4,6-TTCP in 2 days. *P. brevicompactum* did not form the chloroanisole and took 8 days to remove the chlorophenol (534). Decomposition, as measured by ring cleavage, not dechlorination, of 2,3,4,6-TTCP in soil suspensions, took longer than 72 days for complete disappearance (424, 350). *Pseudomonas cepacia* (AC1100) was grown on 2,4,5-T as the sole carbon source. Tests were then run to determine its ability to dehalogenate halophenols, including 2,3,4,6-TTCP. At 23.2 mg/L for 3 hours 86% dechlorination occurred; above 46.4 mg/L the rate decreased due to toxicity (434).

Wastewater treatment biomass, acclimated to PCP, was tested for its ability to breakdown 2,3,4,6-TTCP. In flasks at pH 7.8, 8.5 mg of 2,3,4,6-TTCP was broken down, per gram of cells per hour, from an original concentration of 40 mg/L (458). The growth of a gram-variable bacillus was supported by 2,3,4,6-TTCP and 3,4,6-TCP, but not by any of the other chlorophenols (603). In soil, 2,3,4,6-TTCP degrades to 2,4,5-TCP (17,606).

2,3,5,6-TTCP

Pseudomonas cepacia, grown on 2,4,5-TCP as the sole carbon source, was able to dechlorinate 2,3,5,6-TTCP by 94% in 3 hours at 23.2 mg/L. The growth medium was between pH 7 and pH 8 and strain AC1100 was used in the tests (434). Mixed culture cells taken from a wastewater fiber wall reactor were suspended in phosphate buffer and 40 mg/L of 2,3,5,6-TTCP was added. The breakdown rate was 3.2 mg/gram of cells/hour (458). In soil, 2,3,5,6-TTCP degrades to 2,3,5-TCP and 2,3,6-TCP (606, 17).

PCP

In spite of its extensive use as a biocide (30), pentachlorophenol is quite readily broken down by a wide variety of organisms, and is used as a carbon source for growth by a number of bacteria (49). Pure or reagent grades of PCP are more readily degraded than commercial products. This is likely due to the contamination of commercial products with dioxins, and other compounds more toxic than PCP, inhibiting the organisms responsible for degradation (49).

The half-life of PCP in water is a function of light intensity and duration as well as biological activity. Times of 2 to 5 days were found in some experimental ponds. Turbidity and algal blooms prolong the half-life. The biological half-life of PCP in aerobic waters is about 14 to 19 days, and is increased in darkness, in soil-free habitats, at a pH below the pKa of 4.8 and under anaerobic conditions (159).

Bacteria can grow with PCP as their sole carbon and energy source and at 25C the conversion rate reached 10 µg PCP per hour per mg of cells (dry weight). In 24 hours, 73% of the added PCP was converted to CO_2 (50).

A one year aerobic soil metabolism study in the dark on sandy loam soils at 25 degrees centigrade gave a half life of 63 days using first order degradation kinetics. The degradation appeared to follow a process of progressive dechlorination and yielded TTCP and TCP metabolites (731). Under similar conditions a 60 day anaerobic study gave a half life of 13.9 days for the aerobic controls but no degradation occurred in the anaerobic state. Additional degradation products included mucochloric acid and 2-chlorohydroquinone (732). A 30 day aerobic study using flooded blue sandy loam at 25 degrees centigrade gave a half life of 4.93 days using first order kinetics. Degradation products included TTCP, TCP and 3,4-DCP (733). A similar one year anaerobic study gave a half life of 33.8 days using first order degradation kinetics, TTCP and TCP were the degradation products identified (734).

The evidence for fungal and bacterial degradation of PCP is widespread. Use of isotopically labeled carbon indicates ring cleavage and CO2 evolution (20). In aerobic sediments, at both 0C and 4C, bacterial degradation of PCP is slower than that of 2-MCP, 4-MCP and 2,4-DCP. In water at 20C, degradation is negligible (19). In rice fields, PCP breakdown via reductive-dechlorination occurs and results in the following metabolites, all containing the persistent meta-chloro substituents which are the hardest to degrade: 2,3,4,5-TTCP, 2,3,5,6-TTCP, 2,3,4,6-TTCP, 2,4,5-TCP, 2,3,5-TCP, 3,4-DCP, 3,5-DCP and 3-MCP (536).

Degradation of PCP in sediments begins with dechlorination to form a series of partially dechlorinated products, followed by oxidation to form substituted catechols or hydroquinones and then by ring cleavage and breakdown into CO2 and chloride (175). Many intermediate products are formed in this process and it may be difficult to tell which really are intermediate products and which were contaminants in the original PCP product. Methylation of the hydroxyl forms pentachloroanisole, PCA, which is rapidly accumulated by aquatic life and has a longer half-life than PCP (141). Intermediates include tri- and

tetra-chlorophenols, anisols, resorcinols, quinones, hydroquinones, benzoquinones, hydroxybenzoquinones and catechols (Fig. 4.1).

Acclimated sludge did not degrade PCP in 4 days, when the PCP was initially present at 100 mg/L. Neither ring degradation nor halide ion production occurred (536). PCP-utilizing bacteria occur, but they are slower growing, and when other nutrients are available these will be preferentially used and PCP degradation rates will decrease (536). Sludges from commercial wood treating operations were readily broken down when composted in permeable soil at PCP levels of 200 mg/L or less. There was 98% breakdown in 200 days, and the soil could then be reused for another batch (191).

Activated sludge bacteria break down PCP to CO2, H2O and HCl. In a pilot plant study, wood preserving plant sludge at 23 mg/L was reduced to 0.4 mg/L (191). In sewage treatment plant aeration lagoons, PCP concentrations of 39.5 mg/L and 81 mg/L dropped to 0.5 mg/L and 0.6 mg/L in 3 days and 30 hours, respectively (191). PCP breakdown by sewage sludge bacteria had half-lives of 0.36 days when aerobic, and 192 days when anaerobic (608). The degradation of PCP by Pseudomonas cultures was temperature dependent. No degradation occurred at 0C and the half-lives were 8 to 10 days at 20C and 80 days at 4C (609). In natural sediments where temperatures, oxygen levels, and naive bacteria levels are low, activity rates will also be low and breakdown rates will be very slow (608, 19). The very variable results noted in the literature for half-lives is likely due to the interplay of these factors, some of which were not recorded in all experiments. The adaptability of natural sediment bacterial populations, when repeated PCP doses are applied, is well documented.

4.2.2 Uptake by Organisms

Since direct partitioning from water is presumed to be the primary uptake mechanism, ratios in fish and in the water should be the same for different congeners. The rate of uptake is a function of the lipophilicity until the K o/w exceeds about 104 (log K o/w = 2.01); this is probably a function of membrane/water interfaces or boundary layer effects, where no mixing occurs (153). Permeability through a membrane is a function of lipophilicity, and two membranes with an aqueous cytoplasm phase between them form a diffusion barrier. Ionized compounds are polar which restricts their uptake through membranes. As the pH rises the ionization of chlorophenols increases and uptake rates decrease (6). If absorption occurs below the pKa, where the compound is not ionized, uptake is maximal and not pH dependent. Clearance rates for chlorophenols are sufficiently rapid that organisms should be in dynamic equilibrium with water levels. The absorption rate of PCP is proportional to the concentration in water to well beyond the LC50 dose for fish. PCP is readily absorbed from the gastrointestinal tract, skin and gills of fish (225). In people, PCP is readily absorbed from the lungs, gut and skin (405, 199). The uptake rate of PCP in leeches is pH and temperature dependent (86); pH dependency in many organisms is well documented (86, 79, 6, 113, 363). Uptake in eastern oysters, Crassostrea virginica, reaches equilibrium after 4 days at constant ambient levels of PCP (119). Uptake rates of 2,4,5-TCP in Pimephales promelas were 0.2 and 0.34 µg/g/hour from solutions of 4.8 and 49.3 µg/L at 22C, pH 7.5 and alkalinity of 41.5 mg/L . Maximum bioconcentration factors (BCF) of 1900 and 1800 were achieved in 1 or 2 days (605). Fasting fish absorb PCP through the gills and store it in the fat from whence it may be passed along the food chain (121).

A 1979 paper by Neely (613) discusses how to model uptake and clearance rates of chemicals by fish and calculate the rate constants. Thus, in the absence of experimental data, a reasonable estimate of the constant, based on fish physiology and chemical properties, can be made.

4.2.3 Accumulation / Magnification in Organisms

Bioconcentrating compounds usually have a Log K o/w >4.5 or water solubilities up to 1 mg/L. Usually K o/w is over 1000 for a BCF of 100 (109). Acute toxicity varies six-fold over the range pH 4 to pH 9 and bioconcentration varies similarly (6). At the circumneutral pH of most waters, PCP is over 99% dissociated and the sodium salt is readily soluble. Below the pKa of 4.7 PCP is soluble in most organic solvents but sparingly so in water (199). Assuming that accumulation is correlated with the equilibrium partitioning between lipid and water phases, a regression equation has been developed with a correlation coefficient of 0.97 (378):

$$\log BCF = \log P - 1.32$$

BCF = biological concentration factor
P = K o/w, octanol/water partition coefficient.

For leeches at 12C and pH 7, a similar regression equation was developed; the BCF doubles for a 10C rise in temperature (109).

$$\log BCF = 1.265 \log P - 1.201$$

One needs to specify pH values for experiments since at higher pH the chlorophenols will be increasingly ionized and thus decreasingly fat soluble. There is reasonable agreement between calculated and measured BCFs in 1-day-whole-body, experiments, but not for longer time periods. Specific organs will have higher BCFs (335). Most experimental BCFs will be derived from waters near neutral, where many chlorophenols are mostly ionized, and thus will be lower than calculations carried out below the pKa would indicate. Comparison of Table 4.2.3.1, which gives the calculated values, and Table 4.2.3.4 which gives measured values for whole fish relative to water, shows this discrepancy clearly. The theoretical BCFs calculated for PCP, based on an undissociated Log Ko/w of 5.2, are too high. At environmental pH levels, PCP is almost completely dissociated and the mean of the measured BCFs is about one quarter of the mean of the calculated BCFs (the one anomalous high calculated value was not included in the average). Table 4.2.3.7 shows the effects of pH on the toxicity of chlorophenols to guppies, *Lebistes reticulata*. The toxicity obviously rises as the pH drops; the mg/L needed to show an LC50 response becomes less at lower pH values.

There is also, as a rule, a sharp drop in the toxicity between pH 7.3 and pH 7.8; the response is not linear over the entire pH range. Even in the ambient range of pH 6 to pH 7.3 the slope is quite steep and a small pH change has a noticeable effect on the toxicity. One other observation arising from this table is that the ratio of the toxicity at pH 8 over that at pH 6 tends to rise as more chlorines are added to the molecule; that is as the molecule becomes more lipophilic. This means that it is relatively more readily taken up by fish in its less dissociated form, which prevails at lower pH levels. From a practical point of view this means that for a unit change in pH there is a greater change in the toxicity with more highly substituted congeners.

PCP accumulates in the fatty tissues of animals; significant levels are found in human adipose tissues (536). Average PCP levels in human female adipose tissue in Florida residents was 0.023 to 0.025 mg/kg; apparently PCP does not bioconcentrate in humans (576).

Accumulation in mammalian tissues is low due to very rapid urinary excretion rates, usually as glucuronide conjugates (289). Leeches however have high bioconcentration capacities for chlorophenols and slow elimination rates. This makes them good indicators of long-term contamination and transient

events (192, 193, 194). PCP will be found in the liver, kidney and gall bladder prior to elimination. Accumulation or magnification in predators is not appreciably more than that in grazers, due, again, to rapid breakdown and elimination (13). Bioaccumulation depends upon the ratios of absorption, metabolism and elimination (153), and, in fish, the fat content is correlated with uptake and concentration (280). Since they are immersed in the uptake medium, and have very efficient uptake sites in the gills, fish are most susceptible to bioaccumulation of PCP with factors up to 1000 for whole fish in sub-lethal PCP concentrations in the water (204, 279). Concentration factors in the gall bladder, where concentration and conjugation occur preparatory to elimination, may reach 5400 (204). Amphibian tadpole stages and neotenous forms are even more susceptible to chlorophenols since they too are fully immersed, have gills, are smaller, and do not have protective scales on most of their body.

Bioconcentration occurs especially in the liver, up to 200-fold for MCPs and DCPs, and up to 10000-fold for 2,4,6-TCP (220). The bioconcentration factor for phenol during a 1-day exposure by *Daphnia magna* was 634 (47). Bioaccumulation factors for C14 -labeled PCP were:

5 for the alga, *Oedegonium cardiacum*
205 for the daphnia, *Daphnia magna*
21 for the snail, *Physa* sp.
20 for the mosquito, *Culex pipiens* and
32 for the fish, *Gambusia affinis*

Of the C14 in the various species, intact, un-degraded, PCP accounted for 11% in the alga, 12% in the snail, 33% in the mosquito, 56% in the daphnia and 51% in the fish (12).

Tables 4.2.3.4 and 4.2.3.5 list literature values of bioaccumulation factors relative to water levels for PCP and for other chlorophenols, respectively. Where the reference gives a range of values, only the highest factor is given and only factors over one are listed. Bioaccumulation factors relative to sediments are listed in Table 4.2.3.6. Factors of less than one are listed in this Table, due to the higher chlorophenol concentrations in the sediment as opposed to the concentrations in water. The BCF is a function of species differences and lipid content, but not temperature (682). Figures 4.2.3.1 and 4.2.3.2 show the ranges of bioaccumulation factors reported for various tissues of fish and for various groups of aquatic organisms, respectively. Note that the scales on these figures are logarithmic; there is a great deal of variation in bioaccumulation factors from different species and under different conditions.

The ratio of PCP to 2,3,4,6-TTCP in fish is larger than the ratio in the water, suggesting that preferential bioaccumulation of PCP over 2,3,4,6-TTCP is occurring. This is a reflection of the higher Ko/w of PCP. In sculpins the ratio was 1640/440 and in starry flounder 380/100, for a ratio of 3.7 for PCP over 2,3,4,6-TTCP (269, 720). This is in good agreement with the ratio of 4.6 for the Ko/w values of PCP and 2,3,4,6-TTCP as given in Table 2.2, verifying the assumption that uptake rates are a function of lipid content.

In sunfish, bass and catfish the bioconcentration factors for PCP were 500x in muscle, 1500x in gills and 8000x in livers, when tests were done using 0.1 mg/L PCP in the water (201). The bioconcentration factor for PCP in *Gambusia affinis* was 296 at pH 8 (12).

Fingerling chinook salmon, *Oncorhynchus tshawytscha*, were exposed to 2,3,4,6-TTCP and PCP, in "Woodbrite 24" at 0.7C for 62 days. Woodbrite 24 has 250 g/L of chlorophenols, consisting of 156 g/L of TTCPs and 93 g/L of PCP. The solution used for the fish was 0.002 mg/L of total chlorophenols (1.3 µg/L of TTCPs and 0.7 µg/L of PCP), and fish tissues reached 0.224 mg/kg TTCPs and 0.43 mg/kg PCP

(203). The ratio of TTCPs to PCP in Woodbrite 24 is 1.68 to 1.00, but the ratio in the tissues of the experimental fish ranged from about 0.65 to 0.52, using NaPCP (203). This is a further illustration of the earlier point that PCP is preferentially bioaccumulated over TTCPs.

It is not the relative proportions of chlorophenol compounds in the water which determine their ultimate proportions in tissues, but rather their relative uptake rates from the water. These uptake rates are proportional to the Log Ko/w values rather than to the concentrations in the water.

Juvenile rainbow trout, 250 to 450 g, were exposed to a chlorophenol mixture for 6 days at pH 7.3, 12C and water hardness of 80 mg/L. The levels of the various chlorophenols in the bile and in the plasma were measured at the end of the exposure period and are given in Table 4.2.3.8. The ratios of the chlorophenols in the bile, and especially in the plasma, were quite different to the ratios in the water which is a reflection of the relative Log Ko/w s of the compounds. Plasma level ratios approximated the product of the log Ko/w of the chlorophenol and the ratios of the chlorophenol in the water.

Small rainbow trout, *Oncorhynchus mykiss*, 8 to 10 g in size, were exposed to 26 µg/L of PCP for 24 hours. In a subsequent experiment, the previously treated fish were placed in clear running water for a further 24 hours. PCP levels in the liver, blood, fat and muscle were measured at intervals during exposure and subsequent cleansing. The results are given in Tables 4.2.3.9 and 4.2.3.10. These experiments show that muscle levels never got very high, fat levels were slow to clear, blood levels cleared rapidly with a half-life of less than 24 hours, and most PCP was found in the liver. Even the high levels in the liver had a half-life of less than 24 hours.

A similar experiment was carried out for days rather than hours using the bluegill sunfish, *Lepomis macrochirus.* A sub-lethal concentration of 0.1 mg/L of PCP was used with 6-month-old fish of 16 to 42 g, at pH 7.2 to 7.7, 17 to 21C, 135 to 185 µs/cm conductivity and DO of 7.4 to 8.6 mg/L. PCP was measured and adjusted to maintain losses from uptake, evaporation, adsorption, decomposition, photodissociation and bacterial breakdown. Values given are means of 3 to 6 replicates; the fish were not fed. PCP in the tissues is expressed as µg PCP/g wet weight of fish. During the treatment phase, levels rose for 8 days, then, due to elimination, detoxification and metabolic breakdown, fell to an equilibrium value. As in bacterial degradation processes, there is a lag phase while metabolic machinery becomes optimized to deal with the new toxin (121). These results are given in Tables 4.2.3.11 and 4.2.3.12. These experiments demonstrate again the short half-life of PCP in fish tissues; by the fourth day of recovery in clean water, when sampling began, levels were already below one half of the starting concentrations reached after 16 days of exposure.

Tables 4.2.3.2 and 4.2.3.3 give some bioaccumulation factors for PCP and TTCPs, relative to the sediments, in various tissues of benthic species of marine and freshwater habitats in the lower Fraser Valley and around Victoria. These are drawn from the same data set as the data in Table 3.5.2, which gives the actual sediment levels. These values demonstrate the wide range of values which may be found when one looks at different organisms and different habitats.

The bioconcentration and elimination of C14 -labeled 2-MCP was studied in 100 bluegill sunfish, *Lepomis macrochirus*. Work was done in continuous-flow-aquarium-culture with a turnover rate of six to seven aquarium volumes per day. Water parameters were DO 5.9 to 8.6 mg/L, pH 6.3 to 7.9 and hardness of 35 mg/L. The fish were exposed for 28 days at 16C to 9.18 µg/L of 2-MCP and then transferred to clean water to measure elimination over 7 days. The bioconcentration factor at equilibrium was 214 and the half-life in the tissues was less than 1 day (379, 423).

Rainbow trout, *Oncorhynchus mykiss*, were exposed to pulp mill bleaching effluents in Baltic Sea water, salinity 7 ppt, diluted to 2.5 ppt, at 9C and pH 7, in a flow-through system. The concentrations of chlorophenols and other contaminants in the effluent were not measured. A 2-week exposure to effluent from the chlorination step led to 16 µg of chlorophenol/g fat in the livers; 5 weeks led to 17 µg/g fat. Using effluents from the extraction step, a 2 week exposure gave 25 µg/g fat, and 5 weeks, 45 µg/g fat. These were all liver samples with 2.3 to 2.9% fat content.

When effluent from a chlorination step using 5% chlorine dioxide and 95% chlorine was used, instead of the preceding 100% chlorine, the liver fat levels were 2.9 µg/g after 6 weeks in chlorination step effluent and 1.5 µg/g in extraction step effluent. After an 11 week exposure these levels were 2.8 µg/g and 2.1 µg/g respectively, and 1 and 0.8 µg/g fat in muscle tissue. In all cases levels in the liver dropped to zero 3 weeks after exposure was discontinued, demonstrating the short half-life of chlorophenols in fish livers. (187).

In goldfish subjected to 200 µg/L PCP, the gall bladder levels reached 44 µg/g in 5 hours and 539 µg/g after 24 hours, at which time the whole body load was 2475 µg/g. After a further 24 hours in clean running water, the whole body load dropped to 1720 µg/g as excretion took place, but the gall bladder levels rose to 1077 µg/g since excretion took place via the gall bladder (136).

In Bluegill sunfish continuously exposed for 28 days to 2.5 µg/L of PCP the mean steady state concentration of PCP in the edible tissues was 500 µg/kg for a BCF or bioconcentration factor of 190X, in the non-edible tissue was 2100 µg/kg for a BCF of 790X and in the whole body was 1300 µg/kg for a BCF of 490X (735).

In the Weser Estuary and German Bight, the actinian, *Sagartia troglodytes* lives tightly attached to the polychaete worm *Lanice conchilega* thus both were exposed to the same PCP levels which averaged 0.04 mg/L. The BCF for S. troglodytes was about 70 to 180 while for *L. conchilega* it was 2600 to 8500 (641, 642, 643). This is a good demonstration of the inter-species variability in bioaccumulation of chlorophenols.

Oysters, *Crassostrea virginica*, concentrated NaPCP 41 and 78 fold when exposed to 25 and 2.5 µg/L in the water. Purging took only 4 days in clean water (119). The eel, *Anguilla anguilla*, exposed to 0.1 mg/L of PCP in sea water for 8 days, accumulated 33.4 mg/kg in liver, 9.4 mg/kg in muscle and 4.4 mg/L in blood. After 8 days, levels dropped to 11.9 mg/kg, 3.6 mg/kg and 2.1 mg/L respectively. In fresh water, exposure to 0.1 mg/L of PCP resulted in accumulations of 8.8 mg/kg in liver, 0.81 mg/kg in muscle and 1.7 mg/L in blood after 4 days, and in 55 days levels dropped to 1.3 mg/kg in liver and 0.08 mg/kg in muscle. After 38 days blood levels dropped to 0.31 mg/L (667). Table 4.2.3.13 gives the relative PCP accumulations in a fish, a shrimp and an oyster, relative to water. These are all marine organisms and the results are from 96-h exposures in flowing sea-water.

4.2.4 Elimination

In the gall bladder, chlorophenols are conjugated, usually with glucuronides, for excretion in the urine. Thus, high transient levels may be found in the gall bladder (204, 289). The rapid excretion rate of chlorophenols keeps the bioaccumulation factor relatively low, in spite of the high calculated values in Table 4.2.3.1 based on Ko/w figures, and permits periodic low doses to be tolerated with little permanent toxic effect (263). It takes naive fish about 8 days to develop a fully functioning elimination mechanism for chlorophenols (121).

The liver is the main metabolic organ for breaking down or detoxifying chlorophenols, before transport to the gall bladder for conjugation and ultimate elimination in the urine via the kidney (208, 289). Uptake and elimination of chlorophenols by fish is rapid with a half-life of 2 days for MCPs and DCPs and 10 days for TCPs, TTCPs and PCP (220). Oysters, *Crassostrea virginica*, purge themselves of PCP in 4 days (119).

Some depuration half-lives of chlorophenols, mostly PCP, are given in Table 4.2.4.1. The values are recorded in hours and the longest half-life reported is a whole body value for trout of 168 hours or 7 days.

2-MCP

The half-life of 2-MCP in the whole body of the bluegill sunfish, *Lepomis macrochirus*, is less than 1 day (379). Dogs excreted 87% of the 2-MCP as glucuronic and sulphate conjugates (315, 372) and rabbits also conjugate 2-MCP (315).

2,4,5-TCP

Depuration in *Pimephales promelas*, which had reached BCFs of 1800 to 1900 in 2 days, was rapid with a biological half-life of 12 hours following transfer to clean water. These fathead minnows lost 84% to 92% of their 2,4,5-TCP load during the first day after exposure stopped (605).

2,4,6-TCP

Rapid clearance from the body, in the urine, occurs for 2,4,6-TCP (225). In male rats, radioactive 2,4,6-TCP was given by stomach tube for 3 days at 1 mg/kg diet. Of the amount applied, 80% was eliminated in the urine and 20 % in the feces within 5 days. A subsequent autopsy showed no detectable levels in liver, lungs or fat (460). Rainbow trout livers were cleared of 2,4,6-TCP 21 days after dosing was discontinued (187).

2,3,4,6-TTCP

This chlorophenol was excreted unchanged in the urine, for the most part, when given by intraperitoneal injection to rats at 10 mg/kg. Within 72 hours of the dose being given, 66% could be recovered unchanged from the urine. Over 95% is eliminated within 48 hours. Trichloro-p-hydro-quinone is a minor metabolite (450).

2,3,5,6-TTCP

About 35% of an intraperitoneal dose of 2,3,5,6-TTCP, given to a rat, was excreted as tetrachloro-p-hydroquinone, within 24 hours (45).

PCP

PCP is reportedly excreted in the urine unchanged for the most part (278, 406). In rats and mice, some PCP is dechlorinated to TTCP- and TCP-hydro-quinones (225). In goldfish, PCP is excreted as a conjugate (135, 136, 204) and the half-life for clearance is 10 hours (204). The half-life of PCP in

mammals, including people, is measured in hours (276, 277); in trout it is about 7 days for the whole body (131, 577), 6.7 hours for blood, 4.8 hours for liver, 23.0 hours for fat, 6.9 hours for muscle, 10.3 hours for gills, and 6.9 hours for the heart (134, 141).

Work with midges, *Chironomus riparius*, showed that 89% of the PCP was still unchanged after the experiment, and that pH had no effect on the amounts or nature of the breakdown products (6). The clam, Tapes philippinarum, forms sulphate conjugates of PCP as a means of detoxification prior to excretion, instead of the more common formation of glucuronides (143).

In mammals, there is little fecal excretion, long-term tissue accumulation, or storage, of PCP. Detoxification is by oxidative conversion to quinone or glucuronic acid conjugation and excretion in the urine (199). In people, mice and rats, the primary mode of PCP elimination is the urine. The amount and rate of excretion increases with increasing levels of PCP in the body. Initial elimination is rapid, but complete removal of all the residue may take a month. In the mouse, 72 to 83% is excreted in 4 days and in the rat 68.3% in 10 days. Trace amounts are respired (536). Urinary excretion in mice and rats is mostly free PCP or tetrachlorohydroquinone but glucuronide conjugates also occur. The plasma half-lives of 10 mg/kg were 15 hours in rats and 78 hours in Macaca mulatta, monkeys (464). In Rhesus monkeys essentially all excretion is urinary. The half-life in plasma was 84 hours for females and 72 hours for males; excretion half lives were 92 hours for female and 41 hours for males (570). In male and female rats given 10 or 100 mg/kg PCP, the rapid phase of elimination had a half-life of 17 hours in females and 13 hours in males; this phase eliminated over 90% of the dose of PCP. Excretion was rapid in the urine as PCP, its glucuronide conjugate, or as tetrachlorohydroquinone (277). Female mice were given 15 to 37 mg/kg PCP by either subcutaneous or intraperitoneal injection. The half-life was about 24 hours, and 72 to 83% was excreted in the urine in 4 days, mostly as PCP (407).

PCP is rapidly excreted in fish, after formation of the glucuronide and sulphate conjugates, with tissue half-lives of less than 24 hours (205). Rainbow trout, Oncorhynchus mykiss, fed PCP in their diet reached equilibrium between constant uptake and elimination after a period of time which was a function of the dose. Fish receiving 10 µg/kg in their diets reach equilibrium of 2 µg/kg in 28 days. Higher dose levels reached a higher equilibrium level and took longer to achieve it. The half-life of PCP in the whole body was about 7 days (131).

In Bluegill sunfish continuously exposed for 28 days to 2.5 µg/L of PCP the half life for depuration or elimination from the whole body, of 1300µg/kg, was 1 day and 98% was eliminated in 14 days. Metabolism of PCP by fish tissues was minimal (735).

4.3 Summary of the Fate of Chlorophenols

Once released to the environment, physical, chemical and biological processes break down chlorophenols, ultimately to CO2, H_2O and chloride, with half-lives ranging from hours to months. Table 4.1 gives a summary of the fate of chlorophenols in water. Photodegradation is only important in shallow water under high irradiation levels; hydrolysis and oxidation are not important in nature; evaporation and volatilization are only important in nature in shallow water subject to vigorous mixing; adsorption is a major process and most chlorophenols introduced into the environment will eventually be found in the sediment, usually on organic sediments.

Microorganisms can adapt their metabolic processes to use virtually any source of carbon for growth. Once the adaptive phase is over and a large population has built up, breakdown is rapid and subsequent

additions to the environment are broken down quickly, if the concentrations are not excessive. If the organisms have never been exposed to chlorinated organics there will be an initial lag period while adaptation occurs. The evidence for fungal and bacterial degradation in nature is widespread and Table 4.2.1 gives degradation rates under different pH and temperature conditions. Chlorophenols with chlorines in the 2,4 or 6 positions are more readily degraded than those with chlorines in the 3 or 5 positions.

Bioaccumulation of chlorophenols is species specific; under identical conditions different species may accumulate very different amounts of chlorophenols. Bioaccumulation in the same organism will vary depending upon the environmental conditions. Bioaccumulation factors are different for accumulation from water than for accumulation from sediments and differ from marine to fresh waters. The pH affects the accumulation factor by affecting the dissociation of the chlorophenol and thus the fat solubility and uptake rate. The bioaccumulation factors for different tissues in the same organism vary widely, and whole body values are low compared to values for detoxification and excretion organs. Temperature affects bioaccumulation, much more in poikilotherms than in homeotherms, since all enzymatic reactions are temperature dependent with rates generally doubling with a 10C rise in temperature. Compounds which bioconcentrate usually have Log Ko/w values over 4.5 or water solubilities under 1 mg/L. Usually for a BCF of 100 the Ko/w will be over 1000. In mammalian tissues, bioaccumulation is generally low due to high urinary excretion rates, but in poikilotherms accumulation may be much higher. Biomagnification does not generally occur, again due to rapid excretion and detoxification rates. Preferential bioaccumulation of compounds with higher Ko/w values occurs even when other compounds are present at much higher concentrations. The half-lives of bioaccumulated chlorophenols in vertebrates are quite short, hours or days, due to very efficient excretory mechanisms.

In mammals, chlorophenols are not accumulated in fat to a very high level due to very rapid excretion as glucuronide conjugates, thus keeping bioconcentration factors low. Since fish are immersed in their uptake medium and have gills which are very efficient uptake organs, they bioaccumulate chlorophenols up to 1000 times for whole body loads, in spite of very efficient conjugation and elimination.

Tables 4.2.3.4 and 4.2.3.5 give bioaccumulation factors relative to the water and Table 4.2.3.6 factors relative to the sediments for fish. The half-life of chlorophenols in fish is less than 1 day as shown in Tables 4.2.3.10 and 4.2.3.12; thus the existence of high levels in fish tissues is indicative of chronic exposure. There is an initial lag period while the metabolic processes of the fish adapt to conjugating and excreting chlorophenols, but once adapted, the depuration half-lives are short as indicated in Table 4.2.4.1., generally less than 24 hours. In nature many aquatic organisms do not leave their contaminated sites and thus the effectiveness of the rapid detoxification is limited (711, 712).

5. BIOLOGICAL EFFECTS OF CHLOROPHENOLS

The effects of chlorophenols on organisms are reported under the following headings: cytotoxicity, immunotoxicity, embryotoxicity, fetotoxicity, teratogenicity, mutagenicity, carcinogenicity, and enzymatic/metabolic effects. Most cellular, organ and whole-body effects have been determined on laboratory animals, usually rats and mice, and most genetic effects have been determined on bacteria. Since the effects of chlorophenols are the same in all eukaryotic aerobic organisms, data from one species are generally applicable to others. The main reason for different guidelines in different habitats or for different organisms is the relative efficiency of uptake and transport of chlorophenols to the active

site in the mitochondria. Table 5.0 summarizes some cytotoxicity, teratogenicity, fetotoxicity, and embryotoxicity data of the chlorophenols.

5.1 Cytotoxicity

The only cytotoxicity effects reported consist of chromosomal aberrations and effects on mitosis. These are summarized in Table 5.0.

4-MCP

Root cells of the bean, *Vicia faba*, were exposed to 250 mg/L. This caused genetic malfunctions including a decrease in the mitotic index, anaphase bridges, lagging chromosomes, cytomixis and disturbances in telophase (448, 502, 503).

2,4-DCP

Buds of the bean, *Vicia faba*, were sprayed with 16.3 g/L 2,4-DCP and root cells were exposed to 62.5 mg/L. The buds and root cells had genetic malfunctions including meiotic alterations of chromosome stickiness, lagging chromosomes and anaphase bridges; in addition, root cells had cytomixis, disturbed prophase and metaphase, and chromosome disintegration (448, 502, 503).

PCP

The roots of the bean, *Vicia faba*, were exposed to PCP at 174, 87 and 43.5 ng/L. This caused genetic malfunctions including a decrease in the mitotic index, anaphase bridges, lagging chromosomes, cytomixis and disturbed meta- and ana-telophase (448, 502, 503). Chromosomal aberrations, breaks and gaps, were examined in workers in a wood treatment plant. Air, serum and urine PCP levels were monitored. No differences were found between workers exposed year-round and controls, but the sizes of the study groups were only six people and four people, respectively (573).

5.2 Immunotoxicity

PCP

Immunotoxic effects of PCP are reported in mice, rats, cattle and chickens (199); the ability of organisms to resist bacterial infection is compromised (306). PCP causes thymic hypoplasia in cows; suppresses total leucocyte counts, gamma globulins and IgG in young pigs; reduces humoral immunity and impairs T-cell cytolytic activity in vitro in mice; and increases cell-mediated immunity in rats (736).

5.3 Neurotoxicity

PCP

Neurotoxic effects are reported for PCP, but many are likely due to dioxin contaminants instead of the PCP (199, 307). PCP does apparently increase cell detachments in mouse neuroblastoma cells and cause a transient alteration in brain tissue enzyme activity in rats (736).

5.4 Fetotoxicity / Embryotoxicity

Some chlorophenols are reported as fetotoxic but not teratogenic (288, 294, 314, 320, 321, 322, 737, 738).

2,4,5-TCP

Pregnant mice received 9.0 or 0.9 mg/kg orally during days six to 15 of gestation. Embryo mortality increased at 9.0 mg/kg as did the rate of resorptions. No fetotoxic effects were reported at the 0.9 mg/kg dose rate(480).

TTCPs

The tetrachlorophenols reduce the number of offspring, neonatal survival and the growth rate of the weanlings; but higher dose rates are required than for PCP (38, 288, 294, 322).

2,3,4,6-TTCP

Female 250 g Sprague-Dawley rats were given oral doses of 2,3,4,6-TTCP in oil and sacrificed on day 21 of gestation. The dose rates were 10 and 30 mg/kg/d. There was delayed ossification of the skull in 17% of the 30 mg/kg/d group; the 10 mg/kg/d dose was a no effect level. There was no difference between purified and commercial grade 2,3,4,6-TTCP (322).

In a similar experiment doses of 99% pure 2,3,4,6-TTCP were given at 25, 100 and 200 mg/kg/d . Maternal toxicity occurred at 200 mg/kg/d and reduced weight gain at 100 mg/kg/d, but no effects were seen at 25 mg/kg/d. Pre-implantation losses of 3 to 4% occurred at 100 mg/kg/d and 200 mg/kg/d. Malformed female fetuses were found, but only at non-statistically significant rates (531).

PCP

High dose rates of pure PCP, 26 to 30 mg/kg, reduced the number of offspring, neonatal survival and weanling growth rates of rats (38, 288, 244, 322). PCP is fetotoxic, delays development, reduces litter size, reduces neonatal body weight, survival and growth (199). PCP is embryotoxic but not teratogenic (225, 227). No teratogenicity was observed but some fetotoxicity and maternal toxicity occurred in rats, hamsters and mice whose mothers were given doses of PCP in the 0.34 to 60 mg/kg range during gestation (558, 564, 565, 566, 567). PCP was usually found to be fetotoxic to rabbits and rats but there were some experiments which did not show such effects at the dosages used. Dose related effects were

sometimes absent and NOEL values were sometimes found to be in the tens of mg/kg/day range (737, 738, 739, 740, 741).

5.5 Teratogenicity

Some chlorophenols are fetotoxic but not teratogenic (288, 294, 314, 320, 321, 322, 737, 738).

2,4,5,-TCP

Mice were given 0.9 or 9.0 mg/kg orally at days six to 15 of gestation. No teratogenic effects were noted at either dose level (480).

2,3,4,6-TTCP

In rats, 2,3,4,6-TTCP is not teratogenic (322).

PCP

Though it is embryotoxic, PCP is not teratogenic (227, 288) at doses up to 30 mg/kg/day for 2 months prior to mating and continuing through lactation. PCP is not teratogenic (737, 738).

5.6 Mutagenicity

Rasanen et al., 1977, considered that, due to negative Ames tests, there was little chance of carcinogenic or mutagenic activity in any of 2,3-, 2,4-, 2,5-, 2,6-, 3,4- or 3,5-DCP; 2,3,5- 2,3,6-, 2,4,5- or 2,4,6-TCP, or 2,3,4,6-TTCP (231). There is, however, some question in the literature about the ability of the Ames test to identify organochlorine mutagens consistently. Table 5.6 summarizes the results of the Ames assays with *Salmonella typhimurium*, which are discussed below.

4-MCP

Ames assays, at 200 µg/plate, were carried out on *Salmonella typhimurium* -TA1535, TA1537, TA1538, TA100 and TA98. Only TA1537 showed a slight increase over background rates and 4-MCP may be considered a borderline mutagen (446).

2,4-DCP, 2,5-DCP, 2,6-DCP, 3,4-DCP, 3,5-DCP, 2,4,5-TCP, 2,4,6-TCP, and **2,3,4,6-TTCP**

The Ames assays with *Salmonella typhimurium* -TA98, TA100, TA1535, and TA1537 were carried out at 0.5, 5, 50 and 500 µg/plate, using male Wistar rat livers. The 500 µg plates were toxic but negative for mutagenicity; no tests showed any increase in revertant colonies (231).

2,3,4-TCP, 2,3,5-TCP and **2,4,6-TCP**

The Ames assays at 0.1, 1, 100 and 1000 µg/plate, using *Salmonella typhimurium* -TA100, were negative for mutagenic effects (430).

2,3,5-TCP and **2,3,4,6-TTCP**

The Ames assays using *Salmonella typhimurium* -TA1538 were negative for mutagenicity (429). This reference also used the *Salmonella* strains and concentrations listed under reference (231).

2,4,6-TCP

The mutation rate in *Saccharomyces cerevisiae* was increased by 2,4,6-TCP (200), but this congener was not active in the Ames test (231). The MP-1 strain of this yeast was treated with 400 mg/L 2,4,6-TCP and no difference was noted in inter- or intragenic recombinations. There was a weak forward mutation response, but no increase in mitotic crossovers or gene conversions (465).
2,3,4,6-TTCP

Treating V79 chinese hamster cells with 12.5, 25, 50 or 100 µg/mL (mg/L) of 2,3,4,6-TTCP did not produce any mutants (528). In a similar experiment, a weak mutagenic response was noted (529).

PCP

PCP is not mutagenic in the Ames test (437), host-mediated assay (285) or sex-mediated lethal test on *Drosophila* (286). PCP was negative for mutagenicity in *Salmonella typhimurium*, *Escherichia coli,Serratia marcescens* and *Drosophila melanogaster* (406). It was negative in the intergenic recombination test in *Saccharomyces cerevisiae*, but positive for forward mutation and intragenic recombinations in the yeast (200, 410). PCP is reported as positive in the *Bacillus subtilis* assay (408), the mouse spot test (200) and in cultured human white blood cells or lymphocytes (410). These latter positive results are all reported as slight or weakly positive. PCP is not considered mutagenic (199).

In *Saccharomyces cerevisiae* MP-1, a yeast, forward mutations were induced by 400 mg/L PCP. Cell survival was only 59%, but of the survivors there were about 3x as many mutations under the very heavy PCP dose (563). Mitotic crossovers were not affected by this dose, but mitotic gene conversions doubled. Using the mammalian spot test, and 50 or 100 mg/kg PCP injected into the mothers peritoneal cavity, the authors concluded that PCP is a weak mutagen (563).

PCP is negative in the in vivo micronucleated polychromated erythrocyte assay on mice up to 120 mg/kg (742). *Salmonella typhimurium* strains TA 100, TA 98, TA 1535, TA 1537, and TA 1538, and Escherichia coli strain WP2hcr, gave negative results for mutagenicity, both with and without metabolic activation, (743, 744, 745, 746). PCP did not cause DNA damage in cultured Chinese hamster ovary cells at up to 10µg/mL (747) and a genotoxicity review indicated that PCP is, at most, a very weak inducer of DNA damage producing neither DNA strand breaks nor differential toxicity in bacterial recombinant assays in

the absence of metabolic activation, it did not cause an increase in SCE induction nor induce gene mutations (748).

5.7 Carcinogenicity

2-MCP

In the mouse, *Mus* sp., a skin test of 2-MCP in benzene gave either papillomas, or papillomas and carcinomas (413).

3-MCP

Skin tests of 20% 3-MCP in benzene on the mouse, *Mus* sp., did not result in any carcinomas but did produce papillomas. The test was repeated twice a week for 15 weeks using 25 µL of the test solution (413).

2,4-DCP

Female Sutter mice were given dermal applications of 2,4-DCP at 41 mg/kg body weight, twice a week for 15 or 24 weeks. In both tests the mice had more papillomas than the controls, 1.07/0.07 and 1.62/0.15 respectively. No controls had epithelial carcinomas while the rate was 11/27 and 6/16 respectively for the test animals. EPA reviewed these data and concluded: firstly the high concentration of 2,4-DCP was enough to cause damage by irritation which may have been responsible for the papillomas, secondly no malignant neoplasia was observed unless an initiator was used, thirdly pathology was done only on a gross level, and fourthly the mice were in creosote-treated wood cages which could cause a carcinogenic response by itself (413).

2,4,5-TCP

Available data (in 1979) do not permit one to make an evaluation of the carcinogenicity of 2,4,5-TCP (464). Mice given 600 mg/kg in the diet for 6 months did not form hepatomas (482). Dermal application of 1 drop of 21% 2,4,5-TCP to the backs of mice twice a week for 16 weeks resulted in half the mice with observed skin papillomas, as opposed to none in the controls (483). Mice given 600 mg/kg in the diet had increased liver weights and tumor formation (484), and mice given one subcutaneous injection at a one g/kg rate did not have increased tumor numbers after 78 weeks. These were both male and female mice that were 4 weeks of age when treated (464). Mice given skin treatments of 2,4,5-TCP in benzene produced many papillomas (413).

2,4,6-TCP

High dose rates of 2,4,6-TCP are carcinogenic to mammalian laboratory animals (228, 38, 319, 232). The National Cancer Institute index finds 2,4,6-TCP carcinogenic, inducing lymphomas or leukemias in male but not female rats and in both sexes of mice where it induces hepatocellular carcinomas or

adenomas (232). The IARC indicated that available data did not permit an evaluation of carcinogenicity (464).

Commercial grade 2,4,6-TCP was given to young mice by stomach tube, at the rate of 100 mg/kg, daily for days seven to 28. For the next 74 weeks their diets contained 260 mg/kg of 2,4,6-TCP. Hepatoma and sarcoma rates were not significantly higher than in controls.

The same strains of mice were given a single subcutaneous injection of 464 mg/kg of 2,4,6-TCP and observed after 78 weeks. Tumor incidences did not increase over the control group (464). Mice given skin treatments of 2,4,6-TCP in benzene did not produce any papillomas or carcinomas (413).

F344 rats were put on a diet of 0, 5 or 10 g/kg of 2,4,6-TCP beginning at 6 weeks of age and continuing until they were 107 weeks old. Mean weights of treated rats were lower than controls, but there was no dose-related trend in deaths. Lymphomas or leukemias occurred in male rats, and were dose related, but did not occur in females at rates higher than the control group. Leukocytosis and monocytosis of peripheral blood, and hyperplasia of the bone marrow, occurred at a higher rate than in controls (232).

Mice received 2,4,6-TCP in their diets. Male rats received 5 g of 2,4,6-TCP/kg of rat, or 10 g/kg, for 105 weeks. One group of female rats received 10 g/kg for the first 38 weeks followed by 2.5 g/kg for the next 67 weeks. A second group received 20 g/kg for the first 38 weeks followed by 50 g/kg for the next 67 weeks. Body weights of treated rats were lower than in the controls but there was no dose-related trend in mortality. Hepatocellular carcinomas or adenomas occurred and were dose related (232). To put these high dose-rate experiments into perspective, the drinking water guideline for TCPs is 2 µg/L, or a daily dose of 3 µg for a 75 kg man; which is a rate of 40 ng/kg/day. In contrast the female mice receiving 50 g/kg in their food were receiving about 250 mg/25 g animal or a rate of 10 g/kg/day. This is about 2.5 x 108 times the guideline level for man.

PCP

The dioxin contaminants in some PCP products are carcinogenic (227) but PCP itself is not (227, 38, 319, 232), even when rats receive chronic doses in their diet (38). PCP may be responsible for some Hodgkin's disease and leukemia in woodworkers, but there is no adequate epidemiology. Toxic impurities such as dioxins may be responsible rather than the PCP (233, 234, 235). PCP was not found to be carcinogenic in mice and rats after oral doses that caused obvious toxicity (411), nor in sub-cutaneous doses to rats (412). It does not promote DMBA-induced skin carcinogenesis in Sutter mice (413). PCP is not carcinogenic in rats (199).

Mice were given 46 mg/kg PCP orally, daily, for 3 weeks, and then fed PCP at 130 mg/kg in their food for another 1.5 years. No increase in tumors was noted. Both sexes of two strains of mice were used (319,411).

Female and male Sprague-Dawley rats were given PCP orally for 24 months (females) or 22 months (males) at 0,1,3,10 and 30 mg/kg. There was no increase in tumors at any dose (539). Mice given skin treatments of PCP in benzene did not produce papillomas or carcinomas (413).

5.8 Enzymatic / Metabolic Effects

5.8.1 Mode of Action

The chlorophenols, pentachlorophenol in particular, have a number of effects on organisms which are the basis for their uses as pesticides. They are weak acids and their solubilities increase in alkaline solutions. Although absolute solubilities are low (mg/L), the solubilities are high relative to solute concentrations which cause toxic effects in aquatic life (µg/L).

In the mitochondria, stored energy in foods is converted to a flow of electrons which converts ADP to ATP on its way down an enzyme pathway to the oxygen of the air. Although PCP uncouples oxidative phosphorylation in all eukaryotic cell mitochondria, it does not inhibit electron transport. The net effect is to stop the formation or release of organic phosphate from ATP, thus greatly reducing the energy supply to the cell, while increasing oxygen demand and respiration rates. Oxidative phosphorylation is uncoupled at low concentrations (39, 60, 61, 289) and inhibited at higher levels (39). This affects all aerobic organisms and is the primary mode of action of the chlorophenols. Food reserves quickly become depleted but no useful work can be accomplished since the energy is not trapped as ATP but is wasted in respiration.

Enzymatic activities are affected (59, 62, 63, 64, 139) and limb regeneration in crustaceans is inhibited (74). Although food intake increases, growth still decreases due to decreased food conversion efficiency resulting from the uncoupling of oxidative phosphorylation (78, 94, 140, 151, 156, 308, 309). Since oxidative respiration increases with rising temperature, doubling for approximately every 10C rise, chlorophenols have greater effects at higher temperatures, especially in poikilotherms (5, 86).

The chlorophenols, and PCP in particular, are lipophilic and the increasing ionization which occurs with rising pH reduces uptake. Thus acute toxicity and bioconcentration are reduced about six-fold over the range of pH 4 to pH 9 (6). The differences in LC_{50} values may vary 14-fold from season to season depending upon life-stage and metabolic activity. In any one season there can be a 40-fold difference in LC_{50}s from species to species (113). There is a trend towards greater toxicity as the degree of chlorine saturation increases. This is likely due to greater uptake since the Ko/w increases concomitantly (78).

In homeotherms, the basal metabolic rate is about 13 µg 02/min/g - 0.8; in poikilotherms it is about 2 µg O2. Basal ATP production is thus about 6.5 times less in fish than mammals. If the sizes of the enzyme pools were different in fish and mammals, but reaction rates the same, the lethal PCP dose would be 6.5 times higher in mammals than fish.

However, if the enzyme pools were the same size but the rate differed, the LD50 of PCP would be about the same in fish and mammals (587, 588, 589). In mammals, from mice to cows, the LD50 is quite uniform at about 150 µg/g (315, 287) and the EC50 for mitochondrial activity is about 280 µg/g (63). In fish the majority of species tested fall at about the 200 µg/g range, 60 to 2000 is the spread between salmonids and guppies. Considering the amount of variability in the data due to pH and temperature effects, this is good agreement and seems to indicate that enzyme pools are much the same size and the rate of reaction varies with organism size. Thus a similar amount of PCP ties up the same proportion of oxidative enzymes in all eukaryotic life, and most variation in dose effects for whole organisms are a function of how readily the PCP can reach the active enzyme sites in the mitochondria. Thus, the LC_{50} concentration in the same animal may vary about 10-fold depending upon how the dose is given: whole body exposure, dermal patches, inhalation, oral, intraperitoneal, sub-cutaneous or intravenous.

The effect of PCP is additive; it is the integrated total dose over a period of days which counts, and several small doses equal one large one. This results in the product of the LC_{100} and the time course over which it was determined being a constant for relatively short periods of time (69), as shown in Table 5.8.1.

One sublethal effect of PCP is to cause increased oxygen consumption. In the eel, Anguilla rostratus, 0.1 mg/L doubles the basal respiration rate. Thus it can be estimated that one molecule of PCP blocks the functioning of two ATP sites (590). During PCP toxicity in fish there is a large loss in lipids, but net growth still occurs, and thus protein or carbohydrate increases must occur. Since aerobic energy production is blocked, anaerobic catabolism (breakdown) of fats provides energy more effectively than burning carbohydrates. The rate of use of the fats must be higher than the equivalent use of carbohydrates under aerobic metabolism since the ATP production rate is much lower. The electron transport process thus increases and oxygen demand rises. Effects include slowed glycolysis (carbohydrate metabolism), increased lipid catabolism, increased O2 consumption, and increased heat loss due to failure to capture the energy in the electron transport process.

There is an apparent anomaly of purified PCP being more toxic than the dioxin-contaminated commercial grade, i.e. it causes an LC_{50} response at a lower dose rate. The commercial product has high levels of many different toxic compounds which induce the mixed-function oxidase enzyme system which serves to detoxify many toxins, including PCP. This same effect can be achieved by pre-treating the test organisms with some similar toxic material and generating a high enzyme level before treatment with purified PCP.

If this pretreatment is not carried out the slower induction of these enzymes to useful levels by pure PCP alone, leads to the organism being subjected to higher levels of PCP for longer periods of time. Thus, even though the applied dose is the same, the length of time it is active at that dose level in the animal differs significantly. To achieve the same effect on animals who have had their enzymes pre-induced, requires almost three times the dose of pure PCP (288, 586).

5.8.2 Examples of Effects

A great many similar experiments have been performed on rats and mice and enzymatic extracts in in vitro systems. Only a few examples are given here since there is no doubt about the mode of action of chlorophenols and their effects on enzymatic systems by shutting down energy flow. Mitochondrial oxidative phosphorylation is uncoupled by chlorophenols. This affects the microsomal enzyme system by disturbing electron transport between flavine and cytochrome P-450, resulting in inhibition of hydroxylation (433, 477).

2-MCP

Oxidative phosphorylation in rat liver mitochondria was 50% inhibited by 2-MCP at 67 mg/L, as measured by polarographic oxygen consumption techniques (425). At 5 mg/L 2-MCP caused reversible inhibition of etiolated-pea-tissue-culture-cell-catalase activity and of crystalline beef-liver catalase (315).

3-MCP

Oxidative phosphorylation was inhibited 50% at 20 mg/L in rat liver mitochondria, as measured by oxygen consumption techniques (425).

4-MCP

Oxidative phosphorylation was inhibited by 50% at 20 mg/L of 4-MCP in rat liver mitochondria, as measured by polarographic oxygen consumption techniques (425). In an in vitro system 4-MCP inhibited the activity of lactate dehydrogenase and hexokinase by 50% at concentrations of 3.6 and 7 g/L respectively (449).

2,4-DCP

Mitochondrial oxidative phosphorylation is uncoupled by 2,4-DCP (504, 505); inhibition is 50% at 6.8 mg/L as measured by polarographic oxygen consumption (425). Lactate dehydrogenase activity was inhibited by 50% at 1.3 g/L and hexokinase activity inhibited 50% by 711 mg/L 2,4-DCP (449). Liver microsomal detoxification processes are disturbed by inhibiting P-450, the terminal oxygenation enzyme (505).
3,5-DCP

This chlorophenol inhibits phosphorylation and the electron transport system (433).

2,3,5-TCP

A tissue culture cell line, BF-2, derived from bluegill sunfish, *Lepomis macrochirus*, was exposed to 2,3,5-TCP and assayed by the uptake of neutral red dye. The EC50 was 65 mg/L (423).

2,4,5-TCP

In 200 g female rats fed 0.05% 2,4,5-TCP in their diet, effects were seen in liver microsomal and nuclear membrane cytochrome P-450 in 14 days (477). In rats, 400 mg/kg of 2,4,5-TCP given orally decreased microsomal NADPH-cytochrome C reductase activity and cytochrome P-450 content (479). At 200 mg/kg daily for 14 days, taken orally by male rats, 2,4,5-TCP was not toxic to the liver as indicated by hepatic glucose-6-phosphatase and serum sorbitol dehydrogenase (479). The demethylation of P-nitroanisole was inhibited, in vitro, by 50 mg/L of 2,4,5-TCP, and 20 mg/L inhibited liver microsomes from conjugating naphthol (479).

The chlorophenol 2,4,5-TCP is an effective -SH group inhibitor in enzymes of wood rotting fungi (487). Mitochondria are completely uncoupled or inhibited at 10 mg/L, but 5 mg/L albumin in the mitochondria counters this inhibition (488). In rat liver mitochondria, oxidative phosphorylation was inhibited by 50% in 0.6 mg/L of 2,4,5-TCP, as measured by polarographic oxygen consumption techniques (425).

2,4,6-TCP

In *E. coli*, 2,4,6-TCP inhibits the lactose permease system (B-D-galactoside transport) in proportion to its concentration, beginning at 10 mg/L for 10 minutes (470). Chloride transport in mammalian red blood cells is inhibited 50% by 260 µg/L of 2,4,6-TCP (471). Oxidative phosphorylation in rat liver mitochondria is inhibited 50% by 3.6 mg/L 2,4,6-TCP, as measured by polarographic oxygen consumption techniques (425). In the marine mollusc, *Navonox inermis*, 2,4,6-TCP uncouples oxidative phosphorylation. Applied to neurons in an isolated ganglion, it causes dose-dependent reversible increases in membrane potential and conductance. The effect is on the permeability of potassium relative to other alkali cations and conduction of potassium relative to chloride (469).

2,3,4,6-TTCP

The rat liver microsome detoxification system is affected, likely by inhibition of electron transport between flavine and cytochrome P-450, by as little as 7 mg/L of 2,3,4,6-TTCP in vitro (505).

2,3,5,6-TTCP

A tissue culture cell line, BF-2, derived from Bluegill sunfish, *Lepomis macrochirus*, was exposed to 2,3,5,6-TTCP and assayed by the uptake of neutral red dye. The EC_{50} was 62.6 mg/L (433).

PCP

The livers of rats have enzymes which dechlorinate PCP to tetrachloro- or trichloro-p-hydroquinones (40). The microsomes of liver are active in detoxification processes. This important activity is disrupted by PCP and makes the organism susceptible to other toxins which it might otherwise be able to neutralize. PCP uncouples oxidative phosphorylation and selectively inhibits the terminal oxygenation enzyme P-450. PCP is toxic below 1 mM or 266 mg/L (505, 574). In vitro, lipase is 50% inhibited by 26.6 mg/L PCP and shows threshold inhibition at 2.128 mg/L (575).

5.9 Summary of Biological Effects

Chlorophenols affect the mitochondria of all eukaryotic aerobic organisms in the same way by uncoupling oxidative phosphorylation. This affects the microsomal enzyme system by disturbing electron transport between flavine and cytochrome P-450, resulting in inhibition of hydroxylation. In the mitochondria, stored energy in foods is converted to a flow of electrons, which convert ADP to ATP. ATP is essential for all biological activities which require energy. Uncoupling allows respiration to continue, but stops the conversion of ADP to ATP. Thus respiration rates rise, but no energy is stored and food reserves become depleted. The major reason for different LC_{50} values in different organisms is the relative efficiency of uptake, transport, and elimination of the chlorophenols by different organisms. Chlorophenols are immunotoxic, fetotoxic, and embryotoxic but not neurotoxic or teratogenic. Chlorophenols are probably not mutagenic or carcinogenic; even at the very high doses used in some tests, the results are ambiguous. In almost all mammals the LD_{50} for PCP is quite uniform at about 150 µg/g.

There is no biological justification for extrapolation of toxicity testing to concentration ranges many orders of magnitude over the normally encountered levels (466). One would expect that under abnormally high

toxicity loads the normal metabolic operations of the organism would be disturbed, overloaded or bypassed and that the normal bodily processes that protect against carcinogens could be impaired or unable to cope. Organisms evolved amidst mutagens, carcinogens and other challenges to their survival, and developed the defenses to meet these challenges, but only at the concentrations which would normally be expected in the natural environment.

Testing mutagens, and other toxins, at levels far above the levels at which they are, or will be, encountered, does not necessarily prove anything about whether or not they are mutagenic or toxic at normally encountered concentrations.

6. DRINKING WATER

6.1 General

The major effect of chlorophenols, and PCP in particular, is to uncouple oxidative phosphorylation; this results in inhibition or greatly decreased efficiency in converting ingested food to energy and useful products. This is an essential process in all aerobic organisms, and death will result if the process is stopped. Since the effects on all animals, including people, are essentially the same, and are discussed in Chapter 7 on terrestrial animals they are not repeated here; nor are effects on people singled out and treated separately. Such effects have virtually no bearing on the setting of human drinking water guidelines since organoleptic effects occur at lower concentrations than acute or chronic toxic effects, and such taste and odour effects determine the drinking water guidelines. If one can not taste or smell chlorophenols in the water, there is no health risk and there is also a safety margin over the lowest concentration found to cause a health risk.

6.2 Taste and Odour

Chlorophenols are responsible for taste and odour problems in water; such effects are limiting in determining guidelines, since they occur below the LC_{50} values for organisms (335). The actual guidelines suggested by various agencies for chlorophenols are in the µg/L range, lower than any observed physiological effect level, even after safety factors are applied to the results of taste and odour test panels to allow for more sensitive people in the general population. Tables 6.2.1 and 6.2.2 give some taste and odour thresholds for chlorophenols in water. These tend to be temperature dependent since temperature affects the volatility of chlorophenols. The lowest taste or odour threshold is highlighted in bold type, and given first, for each chlorophenol congener. Taste thresholds tend to be one or two orders-of-magnitude lower than odour thresholds and, since these data are derived using human test panels and water with different characteristics, there is a large amount of variability in the results.

2-MCP, 3-MCP, 4-MCP, 2,4-DCP and **2,4,6-TCP**

Taste and odour problems in drinking water are reportedly caused by these chlorophenols at very low concentrations in drinking water, in the µg/L range (183, 362, 366, 335, 372), but reference 183 reported little effect by 4-MCP and 2,4,6-TCP at these low levels.

6.3 Literature Guidelines

Table 6.3 lists published guidelines for various chlorophenols in drinking water. The most current Canadian guidelines for taste and odour of chlorophenols, by Health and Welfare Canada in 1989 (690), are the same as those in 1987 (110): 30 µg/L for PCP, 1 µg/L for 2,3,4,6-TTCP, 2 µg/L for 2,4,6-TCP and 0.3 µg/L for 2,4-DCP. No guidelines are set for the MCPs. CCME (CCREM) adopted these 1987 Health and Welfare guidelines.

The WHO guidelines for 1984 (225) are all 1 µg/L, including 2-MCP and 4-MCP. Taste thresholds appear to be one or two orders-of-magnitude lower than odour thresholds (225). The MCPs are less toxic than their more chlorinated congeners, but there are data to suggest that their taste thresholds could be as low as 0.1 µg/L (335).

6.4 Recommended Guidelines

We recommend adoption of the existing Canadian Drinking Water Quality Guidelines for Chlorophenols which have been adopted by the BC Ministry of Health (110, 690), with the addition of a monochlorophenol guideline of 0.1 µg/L (335). The existing Canadian Guidelines only specify specific isomers and omit any mention of others; we have set the guidelines based on the total concentration of all the isomers for each group of chlorophenols.

Aesthetic Guidelines (taste and odour)

The combined concentrations of all the monochlorophenols, MCPs, should not exceed 0.1 µg/L in raw drinking water (335).

The combined concentrations of all the dichlorophenols, DCPs, should not exceed 0.3 µg/L in raw drinking water .

The combined concentrations of all the trichlorophenols, TCPs, should not exceed 2 µg/L in raw drinking water .

The combined concentrations of all the tetrachlorophenols, TTCPs, should not exceed 1 µg/L in raw drinking water .

The concentration of pentachlorophenol, PCP, should not exceed 30 µg/L in raw drinking water (690).

Toxicity Guidelines

The concentration of 2,4-dichlorophenol should not exceed 900 µg/L in raw drinking water (690).

The concentration of 2,4,6-trichlorophenol should not exceed 5 µg/L in raw drinking water (690).

The concentration of 2,3,4,6-tetrachlorophenol should not exceed 100 µg/L in raw drinking water (690).

The concentration of pentachlorophenol should not exceed 60 µg/L in raw drinking water (690).

6.5 Rationale

The reported concentration of 2,3,7,8-TCDD, dioxin, in the Dowicide EC-7 formulation of PCP and in the product currently imported from the US, is <0.05 mg/kg. Thus the concentration of 2,3,7,8-TCDD, the most toxic dioxin, in water containing 30 µg/L of PCP, would not exceed 1.5 picograms/L (1.5 ppq or 1.5 x 10-9 µg/L). This is in the range that EPA is considering setting dioxin discharge limits for pulp and paper mills. In various US states, these limits range from 0.1 to 10.0 ppq, mostly in the 1 to 3 ppq range (403). This is also at or below the range being considered for 2,3,7,8-TCDD in drinking water in BC (692). The PCP guideline would thus protect people from harmful effects due to dioxin contaminants in the PCP, at the known levels of contamination.

The lowest PCP dose having a reported observable effect is 5 mg/kg which is embryotoxic in rats. This is equivalent to about 350 mg/day for the average person. Assuming that this person got all of the daily PCP dose from a daily water intake of 1.5 L, drinking and cooking water would have to reach 240 mg/L of PCP to have such an effect. The lowest LC_{50} is seven times as great, or 1900 mg/L of PCP in rats and other species (225, 40, 199). If a standard safety factor of 100 was applied to this LC_{50}, then a concentration of about 20 mg/L should not cause any toxic effects, either acute or chronic, in humans. This level is 1000 times higher than the guideline, which is based on taste and odour considerations.

The Health and Welfare Canada levels for chlorophenols are 30 µg/L for PCP, 1 µg/L for 2,3,4,6-TTCP, 2 µg/L for 2,4,6-TCP and 0.3 µg/L for 2,4-DCP (690). These guidelines are met by the group guidelines. If only one isomer was present in a group, and it was present at the guideline level, there could be a slight taste problem for a small number of people. Normally one would expect several isomers to be present at once, and thus no one isomer would reach objectionable levels; however even if such a condition occurred there would be no health hazard due to the safety factor applied at these levels.

The congeners which could cause taste or odour problems at these guidelines levels include 2,3-DCP, 2,3,6-TCP and 2,4,5-TCP; the latter is the only one in commercial use. The major rationale for these guidelines is the fact that they are, or incorporate, the Canadian Drinking Water Quality Guidelines. One must also consider that these are raw water guidelines and that chlorination of water prior to delivery to the consumer may increase the level of chlorophenols to a small extent. In order to minimize such an effect, all raw water should be filtered before it is chlorinated. The reason for the combined chlorophenol guidelines is to accommodate isomer-level testing procedures, which are more economical to carry out, but the levels are set such that even if all the chlorophenol present in an isomer group were the most sensitive isomer, the specific guidelines would still be met.

7. TERRESTRIAL LIFE

Data on non-mammalian, non-aquatic animals is virtually non-existent. Chlorophenols prevent the transfer of non-persistent viruses by repelling the aphid vector (474). PCP in the blood is bound to protein which reduces tissue plasma ratios, but slows the clearance rate by the kidneys (277). Once absorbed, PCP is distributed throughout the body and accumulates in the liver, kidneys, brain, fat and spleen (407, 408, 409, 199). Most reported exposures to PCP are to technical grade material which is

contaminated with dioxins and many other compounds (Section 3.3) and the reported effects of PCP may be wholly or partly attributable to these contaminants.

As PCP concentrations increase, effects progress from sweating to thirst, fever, rapid pulse and finally to respiratory and cardiac arrest (225). Other effects include systemic effects in the kidney and liver (227), chloracne in rabbits, kidney and liver damage in animals (225, 161), lung damage (161), increased blood pressure, nausea, increased respiration rates, bleeding, hyperglycemia, weak eye-reflex and motor activity, glycosuria, lung congestion, convulsions, vascular system damage, eye irritations, histological changes in liver, kidneys, spleen and skin, skin irritation, respiratory disorders, neurological changes, headaches, weakness, drowsiness, irritability, hyperpyrexia and coma, mucous membrane irritation, reduced organ weights and altered organ functions, and death in severe cases. Growth rates are reduced (289, 294, 312, 313, 314, 315, 199).

7.1 Fungi and Bacteria

Table 7.1 gives the levels of some commercial chlorophenol preparations necessary to control fungi and bacteria which cause problems in the pulp and paper industry (658). These are quite resistant organisms and the levels of active ingredients required for complete control are well into the mg/L range; this is in contrast to the levels at which fish are affected which are in the µg/L range. This illustrates the potential toxicity of pulp and paper wastes in aquatic habitats.

7.1.1 FUNGI

Table 7.1 gives the levels of some commercial preparations of chlorophenols needed to inhibit two fungi common in the pulp and paper industry, *Aspergillis niger* and *Penicillium expansum* (658). Table 7.1.1 gives some EC_{50} and EC_{100} values for growth effects on various fungi by a number of chlorophenols. Fungi are quite resistant and mg/L levels are required for control. Sapstain fungi in unseasoned wood include *Trichoderma virgatum* and *Penicillium* sp. which are controlled by 0.46% v/v of TTCP and Aureobasidium controlled by 0.92% v/v of TTCP. PCP controls *Trichoderma harzianum* at 0.25% v/v and *Phialophora* sp. at 0.125% v/v. Neither PCP nor TTCP are effective against the brown mold, *Cephalosus fragrans*, when used at commercial rates. It requires 1.84% v/v of TTCP and 1.0% v/v PCP for control (657).

4-MCP; 2,4-DCP; 2,4,6-TCP; 2,3,4,6-TTCP

Tests were carried out with the fungus *Trichoderma viride* at 25C for 40 hours, to determine the $EC_{50}$for control of growth (381). The results were 47.6 mg/L, 8.6 mg/L, 6.9 mg/L, and 0.8 mg/L for 4-MCP, 2,4-DCP, 2,4,6-TCP and 2,3,4,6-TTCP, respectively. As is evident from these results, increasing the number of chlorines increases the toxicity and the degree of growth suppression.

2,3,4-TCP; 2,3,5-TCP; 2,3,4,5-TTCP; 2,3,4,6-TTCP; 2,3,5,6-TTCP

Tests with 16 fungal species were carried out to determine what concentration would completely inhibit growth on agar. For 2,3,4-TCP the range was 5.9 to 24.7 mg/L, for 2,3,5-TCP it was 2.96 to 11.8 mg/L, for 2,3,4,5-TTCP the range was 1.74 to 13.9 mg/L, for 2,3,4,6-TTCP it was 6.96 to 23.2 mg/L, and for 2,3,5,6-TTCP the range was 29 to 464 mg/L (432).

2,3,5-TCP

When *Aspergillis niger mycelia* were incubated at 22C on agar plates containing 25 mg/L of 2,3,5-TCP, growth was suppressed (431).

2,3,4,5-TTCP; 2,3,4,6-TTCP; 2,3,5,6-TTCP

Five fungi were tested to determine the concentrations of TTCPs which completely inhibited growth. For the five species, grown at 20C for 7 days, *Candida albicans*, *Aspergillis fumigatus*, *Trichophyton rubrum*, *Trichophyton mentagrophytes* and *Microsporon canis*, the lowest EC100, in mg/L of medium was, respectively:

23.2, 6.96, 23.2, 2.32 and 0.696 for 2,3,4,5-TTCP;
23.2, 23.2, 23.2, 6.96 and 2.3 for 2,3,4,6-TTCP; and
23.2, 69.6, 23.2, 23.2 and 6.96 for 2,3,5,6-TTCP (452).

PCP

The metabolism of glucose by the citric acid (Kreb) cycle in *Aspergillis niger*, is blocked by 1.3 mg/L PCP. Direct oxidation occurs and no ATP formation results (569).

7.1.2 BACTERIA

Table 7.1 gives the levels of some commercial preparations of chlorophenols needed to inhibit two bacteria common in the pulp and paper industry, *Bacillus mycoides* and *Aerobacter aerogenes* (658). Table 7.1.2 gives the effects of chlorophenols on bacteria, including the microtox assay using Photobacterium phosphoreum. The EC_{50} values determined for some natural water sources correlated positively with the humic concentration of the water (435) when a Microtox assay was conducted.

The microtox assay is relatively sensitive for a bacterial test compared to the more robust sewage culture bacteria. The major difference is likely that the sewage bacteria have been acclimated to chlorophenols for some time and populations relatively efficient at breaking down chlorophenols have been established. In any case, sensitivity levels are still lower than those of more responsive organisms like fish and amphibians. Bacteria are part of the solution to organic pollution problems since they actively break down most compounds.

Due to the short life cycle, rapid growth, and very effective gene transfer mechanisms in bacteria, adaptations of populations occur quite quickly in long-term experiments. Also, if previously exposed cultures are re-used in subsequent tests, the EC_{50} values will get progressively higher with time. Consequently, tests done on bacterial mixtures like sewage sludges will give quite variable results, which are not likely to be reproducible. For most of the different standard bacterial assay tests which have been developed, there is at least one order-of-magnitude greater toxicity to chlorinated phenols than to the parent phenol. As indicated in Section 5.8.1 increasing chlorination tends to produce higher toxicity.

2,4-DCP

The growth of *pseudomonads* in the presence of 2,4-DCP at 25 and pH 7.1 to pH 7.8 was strongly inhibited above 25 mg/L. The log phase of growth is dependent upon both concentration and prior adaptation of the culture (511). The TTC dehydrogenase method for determining bacterial toxicity, using activated sludge as the bacterial source, indicated a sensitivity level of 50 mg/L and an EC_{50} of 500 mg/L for 2,4-DCP (512).

2,4,6-TCP

In strain 018 of *Bacillus subtilis*, 2,4,6-TCP inhibited proline and glycine transport mechanisms (468).
2,4,5-TCP and PCP

The bacteria in dairy cultures responsible for yogurt, kefir, butter and cheese are inhibited at 100 mg/L of either 2,4,5-TCP or PCP (486).

2,3,4,5-TTCP

Pseudomonas fluorescens suspensions were treated with 2,3,4,5-TTCP and PCP in two sets of tests. In the first test series, the cells were first exposed to 0, 10, 25 or 35 mg/L of 2,3,4,5-TTCP, then subsequently exposed to 0 or 5 mg/L of 2,3,4,5-TTCP, or 5 mg/L of PCP, after a recovery period during which the initial dose was removed. The second test series began with 10 mg/L of PCP as the initial dose, followed by 1, 5 and 10 mg/L of 2,3,4,5-TTCP. When the first and second doses were both 2,3,4,5-TTCP, no mortality occurred either with an initial dose of 10 mg/L, or a subsequent 5 mg/L dose of 2,3,4,5-TTCP.

An initial dose of 25 mg/L caused 86.6% death and the subsequent 5 mg/L dose, 64.2% death. An initial dose of 35 mg/L caused 99.9% mortality, and the subsequent 5 mg/L dose, 55.7% death. When the first dose was 2,3,4,5-TTCP and second dose PCP, the initial doses of 10, 25 and 35 mg/L caused 0%, 87.2% and 99.4% mortality; subsequent PCP doses of 5 mg/L caused 96.4% to 99.9% deaths. Cells first treated with PCP at 1 mg/L and then 2,3,4,5-TTCP at 5 or 10 mg/L, had mortalities of 17.7% and 32.1% respectively (519). It is presumed that first exposing the cells to 2,3,4,5-TTCP sensitizes them to further chlorophenol exposures. A concentration of 10 mg/L of 2,3,4,5-TTCP is a no-effect level and the LC50 is between 10 and 25 mg/L

PCP

In in-vitro assays, *Pseudomonas fluorescens* was less sensitive to PCP at 20C and more sensitive at 4C or 30C. Cells were most sensitive at maximum stationary phase (8.6 mg/L), less sensitive at early log phase of growth (18 mg/L) and least sensitive at mid log phase (29 mg/L). After 1-hour exposures, mortality reached 100% at 70 mg/L; there was no mortality at 10 mg/L. No further mortalities occurred after a 16-hour recovery period (557).

The light output of luminescent bacteria exposed to PCP for 2 to 5 minutes decreased in proportion to the PCP dose. Sensitivity was measured at 5 µg/L (585). The uptake of proline and glycine by *Bacillus subtilis* is inhibited by as little as 1.3 mg/L of PCP (568).

7.2 Plants

There are no data documenting the effects of chlorophenols in irrigation water on the growth of crops or other terrestrial plant life. Since plants share the same mitochondrial terminal oxidation system with other eukaryotic life, one would expect similar reactions at similar concentrations of chlorophenols delivered to the mitochondria. Under normal aerobic, non-flooded soil conditions, chlorophenols would be in the ionized salt form, poorly dissolved in water, and strongly bound to soil particles, especially organics. Irrigation water, which is otherwise suitable for human consumption or which supports aquatic life, should be quite acceptable for crop irrigation. The effects of chlorophenols in water on seed germination in radishes, *Raphanus sativus*, and sudan grass, *Sorghum sudanense*, are shown in Table 7.2 (651). Radish is somewhat more sensitive to most chlorophenols than sorghum, but both plants are far less sensitive than fish and amphibians. Due to a lack of data, no guidelines are set for chlorophenols in irrigation water, but levels below the 100 µg/L range are not expected to have any noticeable effect on most crops.

2-MCP

Tomato plants convert 2-MCP to the glycoside, Beta-0-chlorophenyl-gentiobiocide, which is isolated from the roots but not the shoots (364).

4-MCP

When the roots of the broad bean, *Vicia faba*, are exposed to 250 mg/L of 4-MCP, mitosis is affected with lagging chromosomes, stickiness, fragmentation, cytomixis, and general disturbance of mitotic stages (448).

2,4-DCP

Soaking seeds of *Gossypium barbadense* and *Triticum vulgare* in a 1 g/L solution of 2,4-DCP for 24 hours completely inhibits germination and also inhibits the emergence of *Vicia faba* seedlings (499).

2,4,5-TCP

When "Woedar", 45% 2,4,5-TCP, was applied to rice at 2,3 or 5 kg/ha there was no difference noted in starch, protein, or ash levels, at any treatment level, compared to controls (481).

7.3 Wildlife

There are few data on wildlife. However, due to the ubiquitous nature of the effects of chlorophenols on mammals, what data there are on livestock and laboratory animals should be applicable. The acute oral LD_{50} for northern bobwhite quail given a single dose of PCP was 627 mg/kg and the NOEL was 175 mg/kg (749). The dietary LC_{50} for bobwhite quail was 5.581 mg/g and the NOEL was 562 µg/g (750); for mallard ducks the respective values were 4.184 mg/g and 562 µg/g (751). The oral LD50 was determined to be 380 mg/kg in mallards and 504 mg/kg in pheasants (753).

7.4 Livestock

PCP is repellent to livestock, which avoid grazing on heavily treated pastures (162); similar avoidance is to be expected by wild ungulates. Animals with well-developed urinary systems are most likely to recover from PCP poisoning, but baby pigs are deficient when still young, and acute, lethal poisoning has occurred when farrowing occurs in newly constructed and excessively treated pens. Used crankcase oil was often used as a carrier for the PCP and the combination is lethal if it is not thoroughly dried (660). Two Hereford cows died after drinking 5% PCP in kerosene. Extreme necrosis of the liver and kidneys occurred (661). Wood preservative containing PCP may be very toxic while still wet on freshly treated wood, but, properly applied and well dried, the wood has little toxicity (659).

2,4-DCP

Sheep and cattle were fed 2,4-DCP at 9, 30 and 60 mg/kg body weight, daily, for 28 days. Muscle and fat tissues had no 2,4-DCP, but concentrations in liver and kidney tissues were high. If the diet was stopped 1 week before analyses, the kidney levels were much reduced, but liver levels were still high (315).

2,4,5-TCP

Cows were fed rations containing 2,4,5-TCP at levels from 10 to 1000 mg/kg for 2 to 3 weeks. No residue of 2,4,5-TCP greater than 0.05 mg/kg was found in milk or cream at diet levels up to 30 mg/kg. At the 1000 mg/kg dose, mean residues in milk were 0.24 mg/kg and in cream 0.19 mg/kg (464).
PCP

The wood from which farrowing pens were constructed was treated with 4.4% PCP, 0.63% other chlorophenols, and 81.6% petroleum distillates, only days before pregnant sows were introduced. Contact with the still wet lumber resulted in enough skin absorption to cause fetal mortality and the birth of weak pigs. PCP on the teats and mammary glands discouraged nursing. Piglets had pathological lesions in kidneys, bladder, liver, spleen, stomach, and intestinal and respiratory tracts (315).

A calf given a single dose of 3200 mg of PCP in 40 gallons of water (17 mg/L) did not show any effects in 4 days. When their drinking water was spiked with 51 mg/L of CaPCP or 60 mg/L of KPCP, calves showed some effects in 5 weeks and 7 weeks, respectively (167). Some LD_{50}s for calves are 140 mg/kg given orally (323), and 35 to 50 mg/kg in an 11-day trial (323). The oral LD_{50} for sheep was 120 mg/kg (323).

7.5 Laboratory Animals

Tables 7.5.1 and 7.5.2 give the effects of PCP and other chlorophenols on mammals and birds, mostly laboratory animals, on a mg/kg, mg/L and mg/animal basis. In a 2,4,5-TCP feeding experiment with rats (313), the concentration of TCP was expressed as mg/kg of diet. In this instance the concentration was converted to an estimate of the mg/kg dose using 250 g as the estimated mean weight of the rats and 25 g as their estimated mean food consumption per day. Both these estimates are at the high end of the possible range.

Mixtures

Rats were fed a mixture of 90:10, PCP:TTCPs at zero, 1, 3, 10 and 30 mg/kg/day in a 2-year feeding study. The acute LD_{50} was estimated to be 150 mg/kg. At the highest dose of 30 mg/kg/day there was

slightly reduced weight gain in females, decreased litter survival and progeny growth rates, increased serum glutamic pyruvate transaminase activity in males and females, increased urine specific gravity in females, and increased pigment in the liver and kidney of males. Liver and kidney pigmentation occurred in females at 10 mg/kg. The NOELs were 10 mg/kg/day in males and 3 mg/kg/day in females (38).

2-MCP

The lowest intravenous dose causing a lethal effect was 120 mg/kg in rabbits (364). The lowest $LD_{50}$for rats was 230 mg/kg given intraperitoneally (229), 670 mg/kg given orally, and 950 mg/kg given subcutaneously (364). There were no oral NOEL doses recorded for 2-MCP, but the estimated level would be about 0.01 of the LD_{50} or 7 mg/kg. Some other LD_{50} data for 2-MCP include 440 mg/kg for the blue fox, 950 mg/kg given subcutaneously to rabbits (364), and 2230 mg/kg given orally in olive oil and 3160 mg/kg given subcutaneously to rats (652). The lethal dose given intraperitoneally to rats is reported as 230 mg/kg and given subcutaneously to guinea pigs is 800 mg/kg (364).

3-MCP

The lowest intraperitoneal dose causing an LD_{50}effect was 335 mg/kg in male rats (229, 372); the equivalent oral dose was 697 mg/kg and subcutaneously it was 1.73 g/kg, both also in rats (372). There were no references to oral NOEL doses, but an estimated value would be about 7 mg/kg in rats. Other LD_{50} data include treating male rats with 1865 mg/kg orally or 4360 mg/kg subcutaneously, both in olive oil (652).

4-MCP

In rats the lowest LD_{50} intraperitoneal dose was 250 mg/kg (372); the equivalent oral dose was 500 mg/kg (372, 653) and it was 1.03 g/kg given subcutaneously in olive oil (372, 652). There were no references to oral NOEL doses, but an estimated value would be 5 mg/kg in rats. Other LD_{50} data for rats are 281 mg/kg intraperitoneally (229), 660 mg/kg orally in olive oil (372, 652), and 1.5 g/kg dermally (372, 653). The oral LD50 for mice is given as 800 mg/kg orally (372).

MCPs

In rats 250 mg/kg is given as the LD_{50} for MCPs as a group (315, 232).

2,4-DCP

An oral dose of 150 mg/kg to pigeons caused no deaths and 87 to 95% was eliminated in 5 days (497). In mice NOEL doses found were 25 and 100 mg/kg for 6 month periods; the lowest effect level for these mice was 230 mg/kg, and the LD_{50} for mice was 1600 to 1630 mg/kg (312). For rats the oral LD_{50}s were 3670 mg/kg for males and 4500 mg/kg for females (312). The LD_{50}s for rats were found to be 430 mg/kg given intraperitoneally (229), 1720 mg/kg given in fuel oil (652) and 1730 mg/kg given subcutaneously (496).

2,6-DCP

In rats there are sub-lethal effects on mitochondrial enzymes in the liver at 5.5 mg/kg (425), while oral LD_{50} effects do not occur until 2940 mg/kg (426). This is a ratio of sub-lethal effects: LD_{50} of 5.5 : 2940 or 0.00187 which is considerably less than the standard factor of 0.01 for many compounds. Perhaps one needs to use a greater safety factor in estimating NOEL levels from LD_{50} levels where chlorophenols are concerned. The LD_{50}s for rats, where the 2,6-DCP was given intraperitoneally, were 390 mg/kg (229, 372) and where given subcutaneously the value was 1730 mg/kg (426).

DCPs

In rats the LD_{50} for DCPs as a group is reported as 250 mg/kg (315, 232).

2,3,6-TCP

There are few data for this compound in mammals, with only one intraperitoneal LD_{50} value of 308 mg/kg in rats (229) being found and no oral dose data. Data from references 364 and 372, which show the ratio of oral to intraperitoneal doses to be about 2.29, would indicate that an appropriate oral dose is 705 mg/kg for an LD_{50} and thus a NOEL dose should be about 7 mg/kg.

2,4,5-TCP

There was no percutaneous absorption of 2,4,5-TCP from the intact skin of rabbits or guinea pigs (476). When rats are fed alpha-hexachlorocyclohexane, 2,4,5-TCP is found as a metabolite in the urine (490). Feeding rats 30 to 1000 mg/kg/day for up to 17 weeks caused little, if any, liver damage (478). Rabbits received 20 oral doses of 2,4,5-TCP over 28 days. No changes were seen in kidney or liver at 1 to 10 mg/kg and only slight effects were noted at 100 and 500 mg/kg (313). Rats received 18 oral doses of 2,4,5-TCP over 24 days. At 1000 mg/kg they lost 10 g in the first 10 days, but later regained the weight. Livers were enlarged at autopsy but no other obvious effects were noted. No effects were seen at 30, 100, or 300 mg/kg (313). In a 98-day experiment, male and female rats were fed 10, 30, 100, 300, or 1000 mg/kg of diet as 2,4,5-TCP. Only mild reversible effects were seen at the 300 or 1000 mg/kg dose, no effects were observed at lower rates of exposure. At 1000 mg/kg there were diuretic and growth retarding effects (313). Oral LD_{50} values for rats are reported as 2800 mg/kg (476), 2960 mg/kg (313), 3000 mg/kg (464), and 4000 mg/kg (475); oral LD_{50} values are 2560 mg/kg and 3160 mg/kg (313), and an oral LD_{100} value is 3980 mg/kg (313). Other responses to doses of 2,4,5-TCP are an LD_{50} of 2260 mg/kg for a subcutaneous dose in fuel oil to rats, an LD_{50} of 820 mg/kg given orally in fuel oil to rats, and an LD_{50} of 355 mg/kg given intraperitoneally to male rats.

2,4,6-TCP

In male rats the intraperitoneal LD_{50} dose of 2,4,6-TCP is 276 mg/kg (229).

3,4,5-TCP

The LD_{50} for male rats of 3,4,5-TCP given intraperitoneally is reported as 372 mg/kg (229, 418).

TCPs

In male rats 250 mg/kg is reported as the LD_{50} dose for TCPs as a group (315, 232).

2,3,4,5-TTCP

Male, 250 to 300 g, and female, 220 to 270 g, rats were treated with 2,3,4,5-TTCP at 2 g/kg. It was dissolved in ethanol and applied to an 8 cm2 shaved area of the skin. After 14 days dermatosis consisting of large, hard, scar tissue occurred, but only one male rat died. The dermal LD_{50} is greater than 2 g/kg (106).

Five days after 200 g rats were given intraperitoneal injections of 498.5 or 1507 mg/kg of 2,3,4,5-TTCP, the livers were removed and microsome fractions prepared. The higher dose rate induces twice as many revertants in the Salmonella mutagenicity test and doubles aryl hydrocarbon hydroxylase activity. The lower dose increased AHH activity only 1.6 times and mutagenicity 1.2 to 1.4 times. Cytochrome P-450 levels were slightly elevated at either dose rate (520).

Injecting 5 mg/egg of 2,3,4,5-TTCP into the fluids around a 17-day chick embryo causes an increase in porphyrin in chick liver cells after 24 hours incubation at 38C (453). Similar effects are found with identical doses of 2,3,4,6-TTCP and 2,3,5,6-TTCP; in the latter case, porphyrin production is increased four-fold (453).

Some LD_{50} values for female mice are 97 mg/kg given intraperitoneally in 40% ethanol, 133 mg/kg given intraperitoneally in propylene glycol, 400 mg/kg given orally in 40% ethanol, and 677 mg/kg given orally in propylene glycol; for male mice a value of 572 mg/kg is given for ingestion in 40% ethanol; and for female gerbils the value is 533 mg/kg when given orally in propylene glycol (450).

2,3,4,6-TTCP

Male rats were given 2,3,4,6-TTCP orally in olive oil. In one test series on 300-g rats they received 10, 50 or 100 mg/kg daily for 55 days. Intestinal necroses occurred in three animals from the 100 mg dose rate, liver necroses occurred in one rat on 50 mg/kg and two rats on 100 mg/kg. There was no effect at 10 mg/kg and no effect on other tissues at any dose. A short-term, 24 -hour, high dose trial was carried out at 0, 300, 360, 410, 432, 518, and 632 mg/kg. There were no effects on brain, kidneys, and muscles. Stomach, spleen, and small intestine effects were seen in some animals; generally livers were affected, one rat at 432 mg/kg, one rat at 518 mg/kg and seven rats at 622 mg/kg (324). Some LD_{50} values for 2,3,4,6-TTCP are 698 mg/kg when given orally in propylene glycol to female gerbils; for female mice 735 mg/kg given orally in propylene glycol, 82 mg/kg given intraperitoneally in propylene glycol, and 131 mg/kg given orally in 40% ethanol; for male mice 163 mg/kg given orally in 40% ethanol (450), and for male rats 130 mg/kg given intraperitoneally in olive oil (229).

2,3,5,6-TTCP

The LD_{50}s for 2,3,5,6-TTCP were determined to be 979 mg/kg for female gerbils when given orally in propylene glycol, 89 mg/kg given orally in 40% ethanol to male mice and, when given to female mice, 48 mg/kg when given intraperitoneally in 40% ethanol, 109 mg/kg when given orally in 40% ethanol, 109 mg/kg when given intraperitoneally in propylene glycol, and 543 mg/kg when given orally in propylene glycol (450).

PCP

Rats injected with 20 mg PCP increased their body temperature 1.5 to 2.0C (544). Male and female rats were fed technical grade PCP for 62 days before, during, and following mating until the 24th month. Dose rates were 0, 3, or 30 mg/kg. No maternal effects were noted during gestation but by 21 days post-partum, maternal body weight differed for those on the 30 mg/kg rate. The number of liveborn pups and their survival at 7, 14, and 21 days post-partum, decreased on the 30 mg/kg dose. Skeletal deformations were evident in pups subjected to 30 mg/kg. No effects were seen at 3 mg/kg (539).

Rats were given technical grade PCP at 0, 0.4, 4, and 40 mg/kg. No fetal deaths occurred but fetal weight decreased up to 20% at 40 mg/L. In dams treated with 0.4 mg/kg, there was 20% mortality of new-born pups. Implantation of the ovum appears to have been prevented by 0.4 mg/kg in the dams; food consumption was not affected (558). Pure PCP was given rats at 0, 0.34, 3.4, or 34 mg/kg. Food intake was not affected; no fetal deaths were noted. Fetal weight decreased 20% in 34 mg/kg dosed rats. Deaths of newborn pups was 12 to 13% in the first 2 weeks when dams received 3.4 or 34 mg/kg (558).

Pregnant Sprague-Dawley rats received pure or commercial PCP in corn oil at 0, 5, 15, 30, or 50 mg/kg. Maternal weight gain decreased at 30 and 50 mg/kg dose rates when treated from days 6 to 15. No fetuses survived 50 mg/kg pure PCP.

The no effect level was 5 mg/kg/day for pure PCP (288). Among cats that slept on sawdust bedding containing 1 to 600 mg/kg PCP, two 5-year old pregnant cats and nine 1 to 6 month old cats died. They had enlarged kidneys, liver degeneration, poor blood clotting, hemorrhaging, lung lesions, and enlarged lymph nodes (559). Beagle dogs were given technical PCP orally for a year at dose rates of 0, 1.5, 3.5 and 6.5 mg/kg/d. The LOAEL was 6.5 and the NOAEL was 3.5 mg/kg/day (752).

In a series of experiments reported by Jones et al. 1968 (541) on rabbits, the LD_{100} varied from 22 to 300 mg/kg for periods ranging from 1.5 to 10 hours, when the method of application included intravenous, dermal, oral, and subcutaneous in pine, fuel, olive, and Dione oils. The most sensitive treatment was a 1.5-h LD_{100} administered intravenously. There are too many experiments, reported in Table 7.5.1, to repeat here; they are mostly on rats, documenting the effects of PCP. The lowest effect level was an LD_{20} for ovum implantation in rats of 0.4 mg/kg (558).

7.6 Humans

4-MCP

Human skin, obtained at autopsy, was shown to be permeable to, and damaged by, 4-MCP at 0.75% w/v (372).

2,4,5-TCP

When 1 mg/kg 2,4,5-TCP was fed to eight people, none was detected in urine or feces (489).

2,4,6-TCP

Human spermatozoa lose the cytoplasmic sheath around the central nuclear material and tail fibres, at a dose of 0.2 ng/cell (467).

PCP and TTCPs

For 1 year a 21-year old man handled wet wood treated with 3% PCP and 1.5 % TTCP. He bled from gums, skin, bowel, and urinary tract, suffered from anemia, and died 5 months later from systemic hemorrhaging, including cerebral bleeding (560).

PCP and other Chlorophenols

Five men worked in a plant where wood was dipped in a preservative containing 4.1% PCP and 0.9% other chlorophenols in 83% petroleum distillate. All were hospitalized with sweating, fever, anorexia, and weight loss. The oldest, 58, died of cardiac dilation, pulmonary congestion, and liver and kidney cell degeneration. All showed PCP in their urine (561).

PCP

A 10 minute immersion of the hands in a 0.4% PCP solution, 106.5 mg/L, by a man produced red, painful hands for 2 hours. After 2 days a 24-hour urine specimen contained 236 µg/L PCP; elimination of the remaining PCP was gradual. Levels did not drop to normal for 4 weeks and 27% was still present after 3 weeks (545).

Four families drank and bathed in well water containing 12.5 mg/L PCP for some time. Symptoms included irritated throats, red faces, and hand and leg weakness. Health improved in 2 to 3 days after stopping use of the water (545).

Studies of woodworkers with long-term exposure to PCP fail to show consistent, significant effects on organs, nerves, blood, reproduction or immunology (199). In a wood-working plant in Winnipeg, Manitoba, five industrial PCP poisonings, one fatal, occurred in 1963. Inadequate precautions in handling and using the toxic material were the reasons and once proper precautions were initiated and adhered to, no further incidents occurred (561). Other literature reports of fatalities were also traced to ignorance or negligence by the workforce (561, 662). In Germany, 10 cases of PCP intoxication occurred at one PCP manufacturing plant. Symptoms were irritation of the mucosa and upper respiratory tract, neuralgic pain and generalized acne (663).

In St. Louis, MO, in 1967, 6 to 14-day old infants were severely affected by PCP poisoning which led to two deaths. Over the next 5 months, 11 more were affected but blood transfusions resulted in immediate recovery from the profuse diaphoresis. The PCP was absorbed from diapers and bed linen laundered in PCP. Serum PCP dropped from 11.8 to 3.1 mg/100 mL in 24 hours following the exchange blood transfusions. An autopsy on one infant that died in 3 hours showed PCP levels of 2.1 to 3.4 mg/100 g of tissue in kidney, adrenal, heart, blood vessels, fat, and connective tissue. PCP levels in diapers were from 2.64 to 17.20 mg/100 g and in crib pads from 4.89 to 178.7 mg/100 g. At the same time, PCP levels in the serum and urine of adults attending prenatal clinics averaged 4 µg/100 mL and in infants at unaffected hospitals serum levels were 11 µg/100 mL and urine levels 2 µg/100 mL (664, 665).

In a case control study on workers in Hawaii there was no significant difference in the general medical health between currently PCP exposed workers and former wood treatment operators. There was no evidence of increased deaths or cases of cancer in the Hawaiian timber treatment operators (754).

Most of the preceding anecdotal material on people serves simply to indicate the various sources of poisonings and the effects; there is no concentration or dose data useful in setting guidelines. This is not unusual for effects on people since controlled tests are rarely carried out. A dermal application of 10 g/L is reported to have an effect and an 18 g dose to a 70-kg person, 257 mg/kg, is reported as an LD_{100} level (163).

7.7 Literature Guidelines

Table 7.7 gives some recommended chlorophenol intake limits for humans; there are no guidelines for animals. The given limits have been converted to an equivalent µg/kg based on an adult 70-kg person. The lowest PCP limit listed is 3 µg/kg/day for people (224). In Table 7.5.1 the lowest PCP dose rate which has an effect, an LD20 for ovum implantation in rats, is 0.4 mg/kg; at 0.34 mg/kg no effects were recorded (558).

7.7 Recommended Guidelines: Livestock and Wildlife

ORGANOLEPTIC GUIDELINES

The recommended guidelines based on organoleptic effects are the human drinking water guidelines as presented in Chapter 6. These values assume no other source of chlorophenols in the diet or in the inhaled air.

TOXICITY GUIDELINES

Based on toxicity calculations the following guidelines are recommended with the proviso that such levels, while not toxic, may prove unpalatable to some species and thus restrict water intake or force animals to search for alternate sources of drinking water. Under drought or similar conditions these toxicity based guidelines may be appropriate, but generally the human drinking water guidelines are recommended. These values assume no other source of chlorophenols in the diet or in the inhaled air.

For lactating animals under high temperatures and high water intake rates (up to 200 mL/kg):

In drinking water the combined concentrations of all the:

monochlorophenols, MCPs, should not exceed 185 mg/L,
dichlorophenols, DCPs, should not exceed 46 mg/L,
trichlorophenols, TCPs, should not exceed 21 mg/L,
tetrachlorophenols, TTCPs, should not exceed 41 mg/L.

The concentration of pentachlorophenol, PCP, should not exceed 17.5 mg/L.

For non-lactating animals under normal temperatures and low water intake rates (about 20 mL/kg):

In drinking water the combined concentrations of all the:

monochlorophenols, MCPs, should not exceed 1854 mg/L,
dichlorophenols, DCPs, should not exceed 460 mg/L,
trichlorophenols, TCPs, should not exceed 210 mg/L,
tetrachlorophenols, TTCPs, should not exceed 410 mg/L.

The concentration of pentachlorophenol, PCP, should not exceed 175 mg/L.

7.9 Rationale

There is another study which gives an NOEL value of 3.0 mg PCP/kg (38, 539) which would result in a value of 15.0 mg/L for lactating animals in hot weather and 150 mg/L for normal conditions. This is a marginally different result from the newer dog studies which are accepted as the best available data.

Assuming that 3500 µg PCP/kg or 3.5 mg PCP/kg, the lowest acceptable and most recent NOEL level from table 7.5.1 (752), is a safe dose level for animals, one can calculate water concentrations knowing the mean weight of the animal and its daily water requirements. The calculated numbers would be based on toxicity, not on organoleptic effects, and may well be too high to be palatable to wildlife and livestock, thus restricting their water intake. This would also assume that the water was the only source of PCP for the animal and that no PCP came from the diet or from inhalation. An extreme water intake rate for lactating animals in hot weather would be 200 mL/kg; this translates to 3.5 X (1000/200)= 17.5 mg/L of PCP in the water, as an upper limit based on toxicity.

A more normal water consumption rate is 20 mL/kg under average temperature conditions and for non-lactating animals gives a PCP drinking water guideline of 175 mg/L.

In people, where water consumption rates are lower, a value of 20 mg/L was calculated in Chapter 6 for a 70-kg person drinking 1.5 L /day.

Using the toxicity ratios shown in Table 2.4, since the effects of chlorophenols are ubiquitous to eukaryotes, and choosing the most toxic isomer in each group of isomers, the appropriate level, in mg/L, when PCP is 17.5, would be 41 for TTCPs, 21 for TCPs, 46 for DCPs and 185 for MCPs. The appropriate level, in mg/L, when PCP is 175, would be 410 for TTCPs, 210 for TCPs, 460 for DCPs and 1854 for MCPs. These are toxicity guidelines and may prove to be unpalatable to some animals.

Using the human drinking water guidelines, based on organoleptic effects, is probably more appropriate, since most animals can smell and taste much lower concentrations than humans.

8. AQUATIC LIFE

Chlorophenol toxicity, especially that of PCP, increases with higher temperatures (5, 156, 311) and lower dissolved oxygen levels (310) due to higher metabolic and respiration rates under these conditions, which in turn require increased food and oxygen uptake. Chlorophenol toxicity, especially that of PCP, is pH dependent, especially near the pKa value since pH controls the ionization status of the chlorophenol

and thus its lipophilicity and uptake rate (6). Based on published EC_{50} and LC_{50} test data for marine and freshwater molluscs, worms, crustaceans, fish and algae, the majority of the EC_{50} or LC_{50} values are in the 0.1 to 1.0 mg/L PCP range. More sensitive species and life stages have lower values (91). Generally speaking, fish, molluscs, worms and crustaceans are acutely affected by PCP below 1 mg/L and chronically affected in the low µg/L range. Algae are affected below 1 µg/L (199).

8.1 Marine Life

8.1.1 COMMUNITIES

PCP

The species compositions of estuarine plankton communities were altered at 76 µg/L of PCP for most species and at a level as low as 7 µg/L for molluscan larvae (202). Marine benthos community structure was altered at 141 µg/L, but not at 1.8 µg/L, in an estuary (117). Based on laboratory tests the NOEL for PCP in marine benthic communities is 13 to 14 µg/L (91).

Experiments by the Institute of Ocean Sciences in 1981, in enclosures in Saanich Inlet, started with 10 µg/L and 100 µg/L PCP. Levels were reduced by 67% in 25 days, mostly by photolysis. Effects included a shift in the centric diatom balance, reduced phytoplankton numbers, reduced sedimentation, and decimation of a major diatom population. Bacterial populations were reduced initially but recovered, as did phytoplankton, but with a shift in species ratios (689).

8.1.2 MARINE ALGAE

3-MCP and 4-MCP

In Table 8.1.2 there are a number of reports of growth inhibition by 3-MCP and 4-MCP on the diatom, *Skeletonema costatum* at 2.5 to 5.0 mg/L (369, 372), *Dunsbella tertiolecta*, a green alga, and *Porphridium* sp., a red alga, at 10 to 15 mg/L (369), and marine plankton assemblages at 0.3 to 1.0 mg/L (354).

PCP

Table 8.1.2 gives some effects of PCP on marine algae. The lowest reported effect is 0.2 mg /L for C14 uptake rates in *Isochrysis galbana*. Reduced photosynthesis, as measured by C14 uptake rates, were caused by 0.5 mg/L in *Thalassiosira pseudonana*, 1.0 mg/L in *Glenodinium hallii* and *Skeletonema costatum*, a diatom, and 0.5 mg/L in an estuarine species assemblage (562).

In laboratory cultures of the red alga, *Champsia parvula*, nominal concentrations of PCP at 465 and 280 µg/L caused decreases, 95% and 50% respectively, in fertilization and sporocarp development. These were 2-day exposure tests followed by a week in clean water to allow development of any sporocarps to visible size (2).

Kelp, *Macrocystis pyrifera*, suffered 50% reduction in photosynthesis in 4 days by 0.3 mg/L NaPCP (331). To eliminate all photosynthesis in 4 days took 2.66 mg/L of PCP; in 2 days the required dose was 1 mg/L (174, 218).

8.1.3 MARINE INVERTEBRATES

Table 8.1.3.1 gives some effects of chlorophenols on marine molluscs, Tables 8.1.3.2 and 8.1.3.4 give effects on marine worms, Tables 8.1.3.3 and 8.1.3.6 give effects on marine crustaceans and Table 8.1.3.5 gives effects on miscellaneous marine invertebrates.

Molluscs

Table 8.1.3.1 gives some effects on marine molluscs. Studies on the clam, *Mya arenaria*, were done with a series of chlorophenols. A static bioassay with 4-MCP at pH 8.0 showed 37 mg/L to be the 96-h LC_{100}value; this was 2.4 mg/L for 2,4,5-TCP, 3.9 mg/L for 2,4,6-TCP, and 11.8 mg/L for 2,3,4,6-TTCP, in laboratory static bioassays at 10C. For 3,5-DCP, 9.8 mg/L was the 35-h LC_{50} value in a laboratory study (386).

PCP

The eastern oyster, *Crassostrea virginica*, takes up PCP to an accumulation level which is inversely dependent upon the ambient level. At 25 µg/L the accumulation was 41 fold and at 2.5 µg/L the accumulation was 78 fold. However, 4 days after the exposures ceased the oysters were fully purged of PCP (119). The 96-h LC50 for larvae was found to be 40 µg/L (78). Abnormal growth of embryos, recorded as 48-h EC_{50} values, also occurred at 40 µg/L of NaPCP (118, 555).

The numbers of molluscs in estuarine benthic communities were reduced at 15.8 µg/L of PCP, but not at 1.8 µg/L (117). Reported 96-h LC_{50} values for this oyster in PCP are 40 µg/L in a static bioassay and 77 µg/L in a flow-through bioassay (205). The related oyster, C. gigas, shows chronic problems with larval development and embryology at NaPCP and PCP levels from 7 to 110 µg/L (120, 257).

The Bajanus Organ of clams accumulates high levels of PCP (30). The larval development of *Bursatella leachi* is completely suppressed at 26C and 17 ppt salinity over a 9- week period by only 7 µg/L of PCP; and 76 µg/L PCP has chronic effects on the development and maturation of miscellaneous planktonic larvae under the same conditions (202).

Studies with NaPCP, either as the pure material or as the commercial product "Santobrite", on the bay mussel, *Mytilus edulis*, showed a NOEL level of 100 µg/L, increasing percentages of abnormal embryology as the concentration increased to 400 µg/L, and 1 to 4 -day EC_{100} and LC_{100} responses at 1 mg/L (164, 680).

Barnacles and Anemones

In laboratory flow-through bioassays using NaPCP, there were no effects at 0.1 mg/L, but at 1 mg/L attachment, growth, and survival were affected; for attachment and growth 1 mg/L is the 1-d EC_{100}level and for survival 1 mg/L is the 3-d LC_{100} level (164).

Tunicates and Bryozoans

The tunicate, *Molgula*, and the bryozoan, *Bugula*, showed a 1-d LC_{100} response to 1 mg/L of NaPCP (164).

Urchins

Table 8.1.3.5 gives effects on sea urchin embryos. The sea urchin, Arbacia punctata, was affected by PCP at 0.3 mg/L in early embryo stages and 0.9 mg/L at 20C affected the sperm. These effects are 4-h EC_{50} and 1-h EC_{50}, respectively (182).

Worms

Tables 8.1.3.2 and 8.1.3.4 give some effects of chlorophenols on marine worms. Nematode biomass and density were not affected by PCP concentrations of 1.8, 7.0, or 15.8 µg/L, but 161 and 622 µg/L decreased biomass and caused species composition shifts (95).

The polychaete worm, *Ophryotrochea diadema*, was tested at pH 8.1, 21C, 70% dissolved oxygen saturation and 33% salinity. The larval forms were more sensitive to PCP than the adults. The ratio of the 96-h LC_{50} to the NOEL ranged from 76 to 1 up to 333 to 1 for PCP. Table 8.1.3.4 gives some experimental results for PCP levels down to 3 µg/L. The larvae are more sensitive to PCP than the adults; there are significantly more deaths in PCP concentrations at and below 100 µg/L. The polychaete, Neanthes succinea, had reduced planktonic larval development and survival at 26C and 17 ppt salinity when exposed to 0.076 mg/L of PCP. In a static bioassay the related N. arenaceodentata responded with a 3-d LC100 to 0.435 mg/L of NaPCP.

Crustaceans

There are two very obviously different life-stages in marine crustaceans as far as sensitivity to chlorophenols is concerned. When the animals are in inter-molt stages and have a hard, relatively impervious carapace, they are only about one-half as sensitive to chlorophenols as when they are in ecdysis, and uptake rates are maximum. Guidelines for chlorophenols has to take this more sensitive life-stage into account (65, 66). Table 8.1.3.6 gives comparisons of molt and intermolt toxicity in the grass shrimp, *Palaemonetes pugio*. There is a 30 fold increase in NaPCP uptake at ecdysis over intermolt stages. At 1 mg/L NaPCP, 76% of the deaths occurred within 48 hours of first ecdysis. At 500 µg/L 66% died 24 to 48 hours after second ecdysis but only a few died after the first ecdysis. There was no effect at 100 µg/L. The 96-h EC50 was 0.436 mg/L of NaPCP (68).

Table 8.1.3.3 has a great deal of data on the effects of chlorophenols on shrimp: *Crangon crangon*, *C. septemspinosa*, *Leander japonicus*, *Penaeus aztecus*, *P. duorum*, *Palaemonetes varians*., *P. elegans*, and *P. pugio*. The lowest 96-h LC_{50} is 84 µg/L for P. elegans exposed to PCP (73). For PCP, 100 µg/L is reported as an NOEL level in P. pugio (68),while the lowest reported EC_{50} is 473 µg/L for limb regeneration in the same species (74). There are also data on other chlorophenol congeners, particularly for *Crangon septemspinosa* (386, 419), and *P. pugio* (521). In *P. pugio* O2 consumption was not affected by 1.5 or 5 mg/L NaPCP during intermolt and pro-ecdysial portions of the molt cycle. Late pro-ecdysial stages showed increased O2 consumption at 5 mg/L and early post-ecdysial shrimp died in 3 hours due to greatly increased PCP uptake rates. Intermolt shrimp only increased O2 uptake or died at 10 to 20 mg/L NaPCP (65). In the grass shrimp, 1 mg/L causes ultrastructural changes in the gills, mitochondria,

gut, and hepatopancreas, during late ecdysis; these changes may lead to death. The new cuticle is very permeable and allows NaPCP into the hemolymph for transport throughout the organism (67). The rates of regrowth of lost limbs, or initiation of limb buds, have EC_{50}values ranging from 0.306 to 0.852 mg/L in grass shrimp exposed to NaPCP (74).

The blue crab, *Callinectes sapidus*, clears PCP within hours from the hemolymph to the hepatopancreatic tissues where it remains, some of it conjugated as esters (77). Hepatopancreatic enzymes such as fumarase, malate dehydrogenase, and succinic dehydrogenase are inhibited by PCP. Cytoplasmic enzymes affected are pyruvate kinase, glucose-6-phosphate dehydrogenase, and glutamate-pyruvate transaminase. Mitochondrial enzymes are more sensitive than those in the cytoplasm. Carbohydrate, protein, and lipid metabolism are affected, as is energy production and ion transport. Membrane bound enzymes are also affected (59). Planktonic larval development is affected by 76 µg/L of PCP at 26C in brackish water (202).

For the copepod, *Pseudodiaptomus coronatus*, the 96-h $EC_{50}$of NaPCP is 68.0 µg/L (78). Another copepod, *Nitocra spinipes*, had 96-h LC_{50} values of 21 mg/L for 4-MCP, and 270 µg/L for PCP, in static bioassays at pH 7.8 and brackish water (385). The isopod, *Mesidotea entomon*, subjected to 4-MCP in brackish water at pH 7.7, had 4 to 7 day LC_{50} values of 23 to 40.3 mg/L (373).

8.1.4 MARINE FISH

Table 8.1.4 gives some effects of chlorophenols on marine fish. There is about a one order-of-magnitude difference between the toxicity of PCP to young sockeye salmon, *Oncorhynchus nerka*, and the lesser toxicity of the unchlorinated parent phenol. The growth rate and food conversion efficiencies of sockeye salmon fry are affected by PCP at 2 µg/L (94). For sockeye salmon the 24-h EC50 is 1.7 mg/L for 2,4-DCP, 900 µg/L for 2,4,5-TCP, 1.1 mg/L for 2,4,6-TCP, 500 µg/L for 2,3,4,6-TTCP, and 300 µg/L for PCP (90).

In static bioassays, the 96-h LC_{50} values for 4-MCP, 2,4,5-TCP, and 2,3,5,6,-TTCP are 5.35, 1.66, and 1.9 mg/L, respectively, when tested with the sheepshead minnow, *Cyprinodon variegatus* (372, 393). With PCP there is a 60-day survival reduction in a continuous-flow bioassay at 88 µg/L (556). The lowest 96-h LC_{50} is 223 µg/L (118). Pro-larvae of the pinfish, *Lagodon rhomboides*, have a 96-h LC_{50} of 38 µg/L to PCP (119, 205, 555). The eel, *Anguilla anguilla*, was exposed to 0.1 mg/L PCP for 8 days in pH 8.1 seawater and 4 days in pH 7.1 freshwater. The observed changes in both cases indicated a hypermetabolic state with accelerated utilization of tissue energy reserves (667). The killifish, *Fundulus similis*, has a 96-h LC_{50} of >0.306 to NaPCP, and the 96-h LC_{50} for NaPCP and PCP is 0.112 mg/L in the mullet, *Mugil cephalis*.

8.1.5 SUMMARY FOR MARINE AQUATIC LIFE

Seaweeds are relatively insensitive to PCP, while the larval stages of molluscs, worms, and crustaceans are relatively sensitive. In crustaceans, the most sensitive stage is at ecdysis when there is no hard carapace and uptake is maximal but, even so crustaceans are not very sensitive. Mollusc larvae are among the most sensitive organisms in marine waters, with LC_{50} values in the 10s of micrograms range. Sockeye salmon show some chronic effects at concentrations as low as 2 µg/L of PCP where growth is affected.

8.2 Freshwater Life

8.2.1 COMMUNITIES

PCP

Biological structure and ecosystem processes were affected in outdoor streams at 48 µg/L of Na-PCP (206). Periphyton species ratios and growth responses were affected by 48 µg/L of PCP in streams (116). Ponds with higher primary productivity, especially those with high macrophyte densities, degraded PCP more readily than other less productive ponds, and afforded more protection for fish and other organisms than less productive ponds. These productive ponds had increased levels of TTCPs indicating PCP breakdown; however, the TTCPs were found in sediments, plant tissues and fish tissues, rather than in the water column (208).

8.2.2 FRESHWATER ALGAE

Table 8.2.2 gives some effects of chlorophenols on freshwater algae. For the unchlorinated parent compound, phenol, Chlorella pyrenoidosa was able to grow in 200 mg/L, but not in 500 mg/L (3). The equivalent concentrations are much lower for most chlorinated phenols.

2-MCP

The only reported NOEL response to 2-MCP is 10 mg/L with *Chlorella pyrenoidosa*. Photosynthesis suppression, as measured by oxygen production, occurred in *C. pyrenoidosa*; reduction was to 88% at 100 mg/L and 74% at 500 mg/L. These were steady state tests at 25C, 5% CO2, 72 hours, constant light and 1 g/L dry weight of algal cells (284). The lowest 96-h EC_{50} value is 70 mg/L in Selenastrum capricornutum (399); no LC_{50} values were found.

3-MCP

The only reported NOEL response to 3-MCP is 10 mg/L with *Chlorella pyrenoidosa*. Photosynthesis suppression, as measured by oxygen production, occurred in *C. pyrenoidosa*; reduction was to 82% at 100 mg/L but did not occur at 500 mg/L. The NOEL was estimated to be 10 mg/L. These were steady state tests at 25C, 5% CO2, 72 hours, constant light and 1 g/L (dry weight) of algal cells (284). The lowest 96-h EC_{50} value is 29 mg/L in Selenastrum capricornutum (399); no LC_{50} values were found.

4-MCP

The suppression of photosynthesis in *Chlorella pyrenoidosa*, as measured by oxygen production, was down to 84% at 100 mg/L and 27.4% at 500 mg/L. Test conditions were steady state, 25C, 5% CO2, 72 hours, constant light and 1 g/L dry weight of algal cells (284). NOEL values of 0.32 mg/L and 3.2 mg/L were reported for *Phaeodactylum tricornutum* and *Scenedesmus pannonicus*, respectively (371). The lowest 96-h EC_{50} value is 4.79 mg/L for growth in *Selenastrum capricornutum* (372); and the lowest 96-h LC_{50} is 9.6 mg/L in P. tricornutum (371).

2,4-DCP

The green alga, *Chlorella pyrenoidosa*, was able to grow in 8 mg/L 2,4-DCP, but not in 10 mg/L (3). At 100 mg/L there was complete destruction of chlorophyll in *C. pyrenoidosa*, and oxygen evolution dropped to 42%. At 50 mg/L the oxygen evolution was 56% of control (284). At 163 mg/L, nitrate and nitrite assimilation in *C. pyrenoidosa* is completely inhibited (500). The lowest EC value is 2 mg/L over 8 days in the green alga, *Microcystis aeruginosa* (383); no LC_{50} values were found.

2,3,4-TCP

There is one 96-h EC_{50} value reported; 2.0 mg/L for *Selenastrum capricornutum* (399); no other data were found.

2,3,5-TCP

The only datum found was an EC value of 10 mg/L for an unspecified green alga (220).

2,4,5-TCP

The trichal blue alga, *Phormidium*, was completely inhibited from spreading in culture by a 100 µg spot of 2,4,5-TCP applied to the culture medium (485). The only NOEL value is 1 mg/L for chlorophyll destruction in *Chlorella pyrenoidosa* (284); this was very close to the lowest 96-h EC_{50} of 1.22 for chlorophyll production in *Selenastrum capricornutum* (399).

2,4,6-TCP

Chlorella pyrenoidosa, a green alga, was able to grow in 100 µg/L of 2,4,6-TCP, but not in 1 mg/L according to one source (3); however, another source (284) indicates that 1 mg/L is the NOEL level for photosynthesis. Reported 72 to 96-h EC_{50} values are 3.5 mg/L in *Selenastrum capricornutum* (399) and 10 mg/L in *Chlorella vulgaris* (399) and *C. pyrenoidosa* (284). No EC_{50} data were found.

2,3,5,6-TTCP

The only data are reports of 96-h EC_{50} values in the 2.66 to 2.72 mg/L range for *Selenastrum capricornutum* (220, 451).

PCP

The algal community structure is disrupted, and some sensitive species are affected, at 1 µg/L of PCP (199). *Chlorella pyrenoidosa* has a 96-h EC_{50} for growth of 7 mg/L (85) but a 72-h EC_{100} of 7.5 µg/L for photosynthesis (284). Other species are affected at 2 µg/L (330). NaPCP at 15 mg/L prevents initiation of algal growth and stops existing algae after about 7 days; 20 mg/L stops existing algal growth immediately (163). NaPCP was initially toxic at 2.0 mg/L for 3 to 7 days for different algal species but by the end of a 21 day period any initial toxicity was overcome (330). In stable ponds in Rhodesia, 5 mg/L NaPCP had the most effect within 24 hours and *Spirogyra* recovered quickly, within 3 weeks (666). The 48-h LC_{65} for *Ankistrodesmus braunii* in laboratory culture was 6 to 7 mg/L NaPCP. After an 8-day recovery period the remaining cells multiplied normally again (8). There is an NOEL report of 2 mg/L for *Gomphonema parvulum*, a diatom, (330), but more data to indicate that there are effects below this

level at 1.8 mg/L, 80 µg/L, 290 µg/L, 410 µg/L, and 7.5 µg/L for several different species of fresh water algae (330, 85, 284). See Table 8.2.2 for details.

8.2.3 FUNGI

The yeast, *Saccharomyces cerevisiae*, is used for mutagenicity tests and is dealt with in Section 5.6. The fungi are dealt with in Section 7.1.1 in the Chapter on Terrestrial Life, rather than here under freshwater aquatic life.

8.2.4 BACTERIA

Bacteria are dealt with specifically in Section 7.1.2 under terrestrial life. Section 5.6 gives the results of mutagenicity tests which are often carried out on bacteria. The standard Ames assay uses *Salmonella typhimurium*, but many other bacteria are also used. Further data on the responses of bacteria to chlorophenols may be found in this section of the report and in Table 7.1.2. which lists many effects of chlorophenols on bacteria, including *Photobacterium phosphoreum*, which is commonly used in the Microtox assay.

8.2.5 AQUATIC PLANTS

The free-floating small duckweeds, *Lemna* sp., are common experimental aquatic plants since they are easily cultured. Their identification to the species level is difficult and most specific epithets found in non-taxonomic literature should be accepted tentatively. They appear to be relatively insensitive to chlorophenols compared to other aquatic organisms. Table 8.2.5.1 gives some effects of chlorophenols on aquatic plants.

4-MCP

The only effects are on *Lemna* sp. and the lowest EC value is 4.79 mg/L (220); other data indicate much higher 48-h EC_{50} values of 282 mg/L for chlorosis (372, 381).

2,4-DCP

The only data are short term or multi-week EC values for growth and chlorosis; the values are in the 5 to 283 mg/L range (3, 381).

2,3,6-TCP

There is one EC value of 5.92 mg/L for duckweed (220).

2,4,5-TCP

The 72-h EC_{50} for chlorosis in duckweed is 1.66 mg/L (372, 381).

2,4,6-TCP

The 2-week NOEL is reported as 100 µg/L for duckweed (3); chlorosis and growth effects are reported at 5 to 5.92 mg/L for duckweeds (3, 372, 381).

2,3,4,6-TTCP

In duckweeds, 2 to 3-day EC_{50} values of 603 to 1400 µg/L are reported for growth and chlorosis (220, 381).

PCP

When *Lemna minor* is exposed to 6 mg/L of PCP for 60 hours, the alanine aminotransferase activity drops to 10% of its starting value. The chlorophyll concentration begins to drop at 1 mg/L and plants are pale by 3 mg/L. Dark respiration increases up to 3 mg/L, then decreases to background at 6 mg/L. Table 8.2.5.2 gives the effects at various PCP concentrations (4). In another study the 48-h EC_{50}value for L. minor is found to be 189 µg/L for chlorosis (381). The water Hyacinth, *Eichhornia crassipes*, is tolerant of PCP or NaPCP. The appearance of the plant is affected at 4.6 or 5 mg/L, but 74 or 80 mg/L is required for a complete kill (332). The contact of NaPCP with *Myriophyllum* and *Phragmites* results in heavy localized damage to the foliage but after 6 to 8 weeks new shoots are formed and the plants recover (674). Canadian waterweed, *Elodea canadensis*, has a NOEL value of 230 µg/L, but 1 to 3-week EC values range from 380 to 1440 µg/L as measured by growth reduction (113).

8.2.6 AQUATIC INVERTEBRATES

Some effects of chlorophenols on aquatic invertebrates are given in the following series of tables:

Table 2.4-QSAR analyses on *Daphnia magna*
Table 8.2.6.1-aquatic insects
Table 8.2.6.2-freshwater molluscs
Table 8.2.6.3-freshwater protozoans and coelenterates
Table 8.2.6.4-freshwater worms
Table 8.2.6.5-freshwater crustaceans
Table 8.2.6.6-cladocerans other than *Daphnia magna*
Table 8.2.6.7-*D. magna*
Table 8.2.6.8-the snail *Lymnaea acuminata*
Table 8.2.6.9-effects of pH, temperature and PCP on the snail, *Physa gyrina*
Table 8.2.6.10- summary of Cladoceran data
Table 8.2.6.11-effects of PCP, temperature and duplicate tests on *Daphnia magna*
Table 8.2.6.12-effects of PCP, temperature and duplicate tests on *Daphnia pulex*

Communities

In a stable Rhodesian pond, 5 mg/L NaPCP caused the heaviest reduction in microfauna in the first 24 hours and the total population dropped from about 30 000 to 80/L after 10 days. Cladocera disappeared quickly and came back only slowly. Copepods and ostracods recovered quickly; the original population levels were back within 3 weeks (666).

Protozoans

Chronic data are available for several chlorophenols and the lowest ones are 68 mg/L for 2-MCP when tested with *Tetrahymena pyriformis* (244), 0.5 mg/L for 2,4-DCP using *Enterosiphon sulcatum*(42), and 0.68 mg/L for 2,4,5-TCP (237), 119 mg/L for 2,3,4,5-TTCP (260), 1.01 mg/L for 2,3,5,6-TTCP (244), and 0.15 mg/L for PCP (237), using *T. pyriformis*, a ciliate.

Coelenterates

The only datum is a 48-h LC50 value of 0.73 mg/L for PCP at 17C and 20C using *Hydra oligactis* (112, 197). There is one 21-day NOEL value of 0.032 mg/L NaPCP also using *Hydra oligactis* (333).

Leeches

Leeches bioconcentrate chlorophenols efficiently and, since they do not excrete it regularly, they are good integrators of fluctuating and sporadic chlorophenol levels over a period of time. The uptake rate is pH and temperature dependent (86).

Worms

Twenty-four hour chronic data of 10 mg/L for 2,4-DCP, 5 mg/L for 2,4,5-TCP, and 10 mg/L for 2,3,4,6-TTCP are available on liver flukes (388). The lowest 96-h LC50 NaPCP datum is 0.11 mg/L for the oligochaete worm, *Nais communis* (92); for PCP the lowest 96-h LC50 datum is 0.259 mg/L in the oligochaete, *Branchiura sowerbyi* (618), and the lowest 48-h LC50 datum is 0.13 mg/L in the planarian, *Dugesia lugubris* (197).

Insects

Insects are relatively insensitive to chlorophenols. The 48-h LC50 for 2,4,6-TCP is >13.5 mg/L for the chironomid, *Tanytarsus dissimilis* (245). The lowest PCP concentration having a 48-h LC50 effect is 0.11 mg/L in the midge, *Chironomus thummi* (197). Table 8.2.6.1 displays all the available data on the effects of chlorophenols, mostly PCP, on aquatic insects.

Crustaceans

The sensitivity of crustaceans varies with the stage in the molt cycle; maximum sensitivity occurs at, and just after, ecdysis, before the carapace has hardened (67, 68, 76). A concentration above 1 mg/L PCP affects the nerve impulse transmission in the abdominal motor axon of the Crayfish, *Astacus fluviatilis* (307). For 2,4-DCP the lowest 48-h chronic effects were found at 0.1 mg/L ; the lowest acute effects were 10-day LC14 values of 1 mg/L. The test organisms were crayfish, *Orconectes propinquus* and *O. immunis* (387). For 2,3,6-TCP, using the crayfish *Astacus fluviatilis*, the lowest 8-day LC50 value was 5.4 mg/L at pH 6.5; this rose to 19 mg/L at pH 7.5 (71). The lowest chronic PCP datum is 0.023 mg/L for survival of young *Gammarus fasciatus* (416). The lowest 96-h LC50 datum is 0.092 mg/L for *Gammarus pseudolimnaeus*, using Dowicide EC7 which was 88% PCP (79); for pure PCP the lowest 96-h LC50 datum is 0.22 mg/L using the shrimp, *Crangonyx pseudogracilis* (113).

Molluscs

Molluscs are particularly sensitive, especially to PCP, and the larval stages are the most susceptible; this is also true for other invertebrates with larval development (311). In the snail, Australorbis glabratus, 2 mg/L PCP causes an accumulation of acetate, pyruvate, lactate, and inorganic phosphate, indications that the Kreb cycle has been shut down. This results from poisoning of the oxidative phosphorylation system by PCP (62)

A useful set of data by Gupta et al., 1982 (97) is presented in Table 8.2.6.8, showing the effects of PCP on the snail, *Lymnaea acuminata*. Both Na-PCP and PCP were used and LC_{16}, LC_{50} and LC_{84}calculations were made, for 12-, 24-, 48-, 72-, and 96-hour exposure periods. In any time series block of data, the toxicity rises with increasing exposure time. Also, as expected, LC_{16} values are lower than LC_{50} and LC_{84} data, respectively, for any identical time period. Thirdly, with the exception of the 48- and 72-hour LC_{16} values, PCP was more toxic than Na-PCP. At the pH of 7.9, where this experiment was carried out, ionization of PCP should have been essentially complete and the availability of the two compounds equal. Correcting the doses to reflect the actual amount of active ingredient, PCP, by subtracting the weight of the sodium, only gives better agreement for the 72- and 96-hour values. Shorter exposure periods still indicate much less toxicity when given as Na-PCP. In this snail the ratios of the LC_{50}s of phenol to PCP vary from 802 to 923, over exposure periods of 12 to 96 hours. This is almost two orders-of-magnitude greater sensitivity to PCP than to phenol, which is greater than the one order-of-magnitude difference noted in section 8.2.4 for bacteria and section 8.1.4 for salmon. This verifies other work by VanDijk et al. (311) indicating the high relative sensitivity of molluscs to PCP.

Table 8.2.6.9 shows the effects of pH and temperature on the 96-h LC_{50} of PCP in the snail, *Physa gyrina* (113). The maximum effect of the PCP is at the optimum growth conditions tested of 24C and pH 8.1. Snails need high pH values in order for dissolved calcium to be readily available for shell growth, and higher temperatures promote high growth rates in these poikilotherms. Due to the mode of action of PCP on terminal respiration, effects will be more marked as growth rates increase. The lowest 96-h LC_{50} was 0.22 mg/L ; at pH 7.8, and a temperature of 4.2C, the 96-h LC_{50} rose to 1.38 mg/L.

Table 8.2.6.2 gives the effects of several chlorophenols on various species of molluscs. For 2-MCP, 4-MCP, 2,4-DCP, 2,4,5-TCP, and 2,3,4,6-TTCP, only 24-h LC_{100} data are available: the values are 10 mg/L, 10 mg/L, 10 mg/L, and 1.51 mg/L, respectively, for the limnaeid snails, *Pseudosuccinea columella* and *Fossaria cubensis* (388). There is one 96-h LC_{50} value of 5.5 mg/L for 2,4,6-TCP in the snail, *Aplexia hypnorum* (245).

There are more data for PCP. NOEL values of 0.05 mg/L, 0.111 mg/L, and 0.2 mg/L are reported, generally for reproductive effects in relatively hard water (85, 102, 112). The lowest chronic effects are reported at 0.05 and 0.09 mg/L (85, 215). The lowest acute effects reported are 96-h LC_{50} values of 0.16 to 0.18 mg/L PCP at 18C and pH 7.9 in hard water (97).

Cladocerans

There are abundant data on the cladocerans, mostly *Daphnia magna*. They are primarily IC_{50} or EC_{50} data; few are LC_{50} data. The ratio of acute to chronic toxicity endpoints varies widely, even within this relatively uniform group of organisms. It is 37 for *Ceriodaphnia reticulata* and 1 for *Sinocephalus vetulus* (113). Table 2.4 gives the results of an experiment on *Daphnia magna* by Devillers et al. in 1986 (57), testing all but two of the different congeners of chlorophenols under identical conditions; only 2,5-DCP and 2,3,4,6-TTCP were not included in this test series.

A series of tests with PCP using the cladoceran, *Sinocephalus vetulus*, at 25C and at a series of different pH levels shows that, as the pH rises from 7.3 to 8.3, the 48-h LC_{50} also rises from 160 µg/L to 364 µg/L (113). For several species of amphipods the toxicity of PCP is clearly shown to vary by about a factor of ten, over the range pH 6.5 to pH 8.5; acidic pH levels are more toxic (79). The PCP used was a commercial mixture, Dowicide EC7, which contained 88% PCP. The experimental conditions were a temperature of 22C, a hardness of 42 to 47 mg/L, alkalinity of 10 to 52 mg/L, a free CO2 level of 0.32 to 6.32 mg/L, dissolved oxygen >60% saturation at 6.6 to 8.7 mg/L, and 16 hours of light per day. Few experiments control, or report, this many experimental variables.

The series of experiments conducted by Adema et al. (85) illustrate the increase in toxicity as exposures increase from 24 hours to 21 days. Another series of experiments under identical conditions by Le Blanc (56) allow further comparisons of toxicities of the different chlorophenol congeners; however, fewer congeners were tested than in the experiment by Devillers (57). The same relatively non-toxic status for 2,4,6-TCP was found by both experiments. A much closer agreement between the ratios is found between the two experiments if the 24-h EC_{50} data of Le Blanc are compared to the 24-h IC_{50} data of Devillers (57). This shows that the length of exposure is a critical piece of data when comparing results between experiments.

A series of experiments were done by Lewis and Horning on *D. magna* and *D. pulex* which illustrate the variation between experimental runs, the effect of temperature differences, and the effect of the duration of the experiment when calculating the LC_{50} for NaPCP. These data are given in Tables 8.2.6.11 and 8.2.6.12 and show variation of up to 2.2 between the lowest and highest calculated LC_{50}.

Tables 2.4, 8.2.6.6, 8.2.6.7, 8.2.6.11 and 8.2.6.12 give data on the effects of chlorophenols on cladocerans. All of this information has been summarized in Table 8.2.6.10 which gives the lowest chronic, acute and NOEL data for cladocerans. The acute data are usually given as the 24-h or 48-h LC_{50}, and chronic data are usually the level which causes immobility. The lowest chronic effect level reported for PCP was 4.1 µg/L (113).

8.2.7 AMPHIBIANS

Very little work has been reported on aquatic vertebrate animals other than fish. What data there are indicate that the most sensitive organisms are the tadpole life-stages of amphibians. This is not surprising since they are small, fully submerged, growing and metamorphosing rapidly, have no impervious scales or exoskeleton, and have extensive gill surfaces exposed to the water for efficient uptake. Table 8.2.7 gives what few data there are available on amphibians. Tadpoles of the common frog, *Rana pipiens*, were tested with NaPCP. No tadpoles died at 0.6 mg/L over a three day period but there was a 6.3-h LC_{100} of 1 mg/L and a 1.3-h $LC_{100}$of 5 mg/L (69). No chronic data were reported. but one would expect such values to be less than 1 mg/L. Other reported 48-h LC_{50} data for amphibians are in the 0.2 to 0.3 mg/L range (112, 114, 323, 551), but no deaths were reported at 0.13 to 0.21 mg/L (112, 551).

8.2.8 FISH

The main uptake route for chlorophenols in fish is from the water via the gills, not from the diet (131). Toxicity of NaPCP in fish increases as the pH drops and the temperature rises (19, 168); water hardness has little effect on chlorophenol toxicity (19). Using goldfish, *Carassius auratus*, and bluegill

sunfish, *Lepomis macrochirus*, the effects of water hardness on the toxicity of PCP was investigated. Hardness levels of 13.0, 52.2, 208.7 and 365.2 mg/L as CaCO3 were tested at Ca/Mg ratios of 1 : 1 and 5 : 1. The tests were 96-h static bioassays and no significant effect of hardness was found (124). Yolk sac edema and skull deformations are common chronic effects with technical grade PCP, which contains dioxins and other impurities, but are rare with purified grades of PCP (259). The breakdown-product, pentachloroanisole (PCA) is much less toxic to fish, fungi, and bacteria than PCP (17). However, PCA is rapidly taken up by organisms and has a longer 1/2 life than PCP.

Salmonids are especially sensitive to PCP (311). Fish mortality is rapid at acutely toxic levels of PCP; thus 24-hour and 96-hour LC_{50}s are usually similar (251). When exposed to chlorinated effluents, the fathead minnow, *Pimephales promelas*, accumulated DCPs and TCPs (186).
Species, size, age, and genetic diversity are responsible for some observed differences in sensitivity to chlorophenols. PCP can be lethal in the 50 to 1000 µg/L range with chronic effects at 0.1 to 0.05 of the 96-h LC_{50} (211). Fish are killed or acutely affected, at 100 µg/L and growth and reproduction is affected at 2 µg/L (254).

There are a series of tables showing the effects of chlorophenols to fish:

Table 8.2.8.1-carp, *Alburnus*, *Aplocheilus*, *Cyprinus*, *Rutilus*, *Carassius*.
Table 8.2.8.2-miscellaneous species.
Table 8.2.8.3-*Lepomis macrochirus*, the bluegill sunfish.
Table 8.2.8.4-*Rasbora daniconius neilgeriensis*.
Table 8.2.8.5-*Pimephales promelas*, the fathead minnow.
Table 8.2.8.6-*Notopterus notopterus*.
Table 8.2.8.7-*Oncorhynchus* sp., salmon and trout.
Table 8.2.8.8-the guppy, *Poecilia reticulata* / *Lebistes reticulata*.
Table 8.2.8.9-*Ictalurus punctatus*, the channel catfish.
Table 8.2.8.10-*Salmo* sp., salmon and trout.
Table 8.2.8.11-*Notropis* sp.
Table 8.2.8.12-this table gives the lowest chronic and acute data for each chlorophenol congener, as extracted from Tables 8.2.8.1 to 8.2.8.11 of fish data.

The lowest acute response by a fish to PCP was 0.018 mg/L or 18 µg/L, while in frogs the lowest value was 0.207 mg/L. In the frog this was a 48-h LC_{50} value as compared to a 96-h LC50 value for the fish. One would expect the frog value to be lower if it had been tested and reported as a 96-h LC_{50} . The lowest fish chronic response to PCP was 0.66 µg/L. Using the more soluble Na or K salt of PCP results in a lower acute response level of 9.8 µg/L and a slightly higher chronic response level of 1.74 µg/L (69, 94, 126, 142, 148). Salmon and trout fry are quite sensitive to chlorophenols, more so than most other fish and animals, and more sensitive than frog tadpoles.

In fish, PCP concentrations are generally highest in liver, gills, gall bladder, and digestive tract, and lowest in muscle tissue (296, 143, 121). PCP absorbed by fish accumulates in various organs, but eventually ends up in the gall bladder and is ultimately secreted, as the beta-glucuronide or as the sulphate conjugate, by the liver (143).

Pre- or simultaneous treatment of rainbow trout with carbaryl, and then 0.25 or 0.50 mg/L PCP, produces a synergistic effect, and the 2-h LC_{50} for the trout and PCP decreases significantly (553).
MCPs

Using goldfish, *Carassius auratus*, and taking the product of concentration and survival time, the relative toxicities of the MCPs, compared to phenol were 1.15 for 2-MCP, 1.51 for 3-MCP and 1.89 for 4-MCP (392).

2-MCP

A group of fish species were exposed to 2-MCP for 48 hours and examined to determine the metabolites. These metabolites were 50 to 60% chlorophenylsulphate and 10 to 30% chlorophenylglucuronide in the aquarium water, as a result of urinary excretions. The bile contained 70 to 80% chlorophenylglucuronide and 10% chlorophenylsulphate. No dechlorination took place, as is common in mammals (158, 135). The fish species used were:

Rhodeus serviceus amarus -bitterling
Abramis brama -bream
Carassius carassius -carp, goldfish
Gobio gobio -gudgeon
Poecilia reticulata -guppy
Phoxinus phoxinus -minnow
Perca fluviatilis -perch
Rutilus rutilus -roach
Scardinius erythropthalmus -rudd
Gasterosteus aculeatus -stickleback
Tinca tinca -tench

These fish showed a sub-lethal response to 2-MCP at 3, 3, 10, 2, 3, 4, 4, 5, 7, 5, and 3 mg/L, respectively (158).

The lowest reported acute response for 2-MCP is 2.1 mg/L in rainbow trout, *Oncorhynchus mykiss*(611). This is a 96-h LC50 value at pH 7.7, 12C, in water with a hardness of 280 mg/L. The lowest chronic response is 3.0 mg/L in guppies, *Poecilia reticulata*. In the guppy, the LC_{50} rose from 7.1 to 13.5 mg/L, a ratio of 1.9, as the pH rose from 6.1 to 7.8, due to better uptake of the undissociated 2-MCP at the lower pH levels (363). See Tables 8.2.8.8 and 4.2.3.7 for pH effects on chlorophenol toxicity in the guppy. Fathead minnows, *Pimephales promelas*, in the embryo to larvae developmental stages, were reported not to be affected at concentrations up to 3.9 mg/L during long-term exposures (364). In fish the reported NOEL and no lethal effect values range from 3 to 10 mg/L, but no *Oncorhynchus* or *Salmo* species were tested.

3-MCP

The lowest reported acute response for 3-MCP is 2.9 mg/L at pH 7.7, 12C, in water with a hardness of 280 mg/L (611). The test fish was the rainbow trout, *Oncorhynchus mykiss*. The lowest chronic response was 10.0 mg/L in the common carp, *Cyprinus carpio*, where it caused malformed embryos (368). In fish the reported NOEL and no lethal effect values range from 1 to 1000 µg/L, but no *Oncorhynchus* or *Salmo* species were tested. In the guppy the LC_{50} rose from 6.4 to 7.9 mg/L, a ratio of 1.2, as the pH rose from 6.1 to 7.8, due to better uptake of the undissociated 2-MCP at the lower pH levels (363). See Tables 8.2.8.8 and 4.2.3.7 for pH effects on chlorophenol toxicity in the guppy. Data on 3-MCP are rare as it is not a commonly used chemical.

4-MCP

As shown in Tables 8.2.8.8 and 4.2.3.7 for the guppy, *Poecilia reticulata*, the toxicity of 4-MCP decreases from 6.3 to 9.0 mg/L as the pH rises from pH 5 to pH 8. The ratio of the toxicity at pH 8 to the toxicity at pH 5, is 1.4 for 4-MCP, and the toxicity of 4-MCP at pH 8 is 1/10 the toxicity of PCP at pH 8 (144). The lowest reported acute toxicity to 4-MCP is a 96-h LC_{50} of 3.8 mg/L at pH 7.6 to 8.3 in a static bioassay using juvenile fathead minnows, *Pimephales promelas* (394).

The lowest chronic response is a 48-h EC_{50} of 3.0 mg/L at pH 7 to 8, using the golden orfe, *Idus idus melanotis* (366). In fish the reported NOEL and no lethal effect values range from 2 to 3.2 mg/L, but no *Oncorhynchus* or *Salmo* species were tested.

2,3-DCP

No data were found on the effects of 2,3-DCP on fish.

2,4-DCP

The toxicity of 2,4-DCP to the guppy, *Poecilia reticulata*, decreases from 3.5 to 7.6 mg/L as the pH rises from pH 6 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 6 is 2.2, as shown in Tables 8.2.8.8 and 4.2.3.7 (144). The lowest reported chronic and acute toxicities to 2,4-DCP are both 0.07 mg/L in the rainbow trout, *Oncorhynchus mykiss*. The chronic response was an EC_{100} (220), and the acute response was a 23 to 27-d LC_{50} at 14C, pH 7.8, and water hardness of 200 mg/L; embryos and larvae were used (376). Several experiments were done on a number of *Oncorhynchus* sp. at different hardness levels, but pH was directly related to hardness so no clear distinction could be made between them and it is known that lower pH leads to greater toxicity (376).

2,5-DCP

No data were found on the effects of 2,5-DCP on fish.

2,6-DCP

The toxicity of 2,6-DCP to the guppy, *Poecilia reticulata*, decreases from 3.9 to 17.9 mg/L as the pH rises from pH 6 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 6 is 4.6; these data are given in Tables 8.2.8.8 and 4.2.3.7 (144). The lowest reported acute toxicity response to 2,6-DCP is a 96-h LC50 of 3.9 mg/L using the guppy, *Poecilia reticulata*; this was carried out at 26C, pH 6.0, and a water hardness of 90 mg/L (144). The lowest chronic response found was 5.0 mg/L in a 12-h static bioassay using larval lamprey, *Petromyzon marinus* (614).

3,4-DCP

There are few data on the effects of 3,4-DCP on fish. No chronic data were found but the lowest acute response, an LC_{100} of 5.0 mg/L, was reported for three species of fish in static bioassays (614). The conditions were 2.8C for 3 hours using the rainbow trout, *Oncorhynchus mykiss*, 17C for 3 hours using the bluegill sunfish, *Lepomis macrochirus*, and 12.8C for 11 hours using larval lamprey, *Petromyzon marinus*.

3,5-DCP

No data were found on the effects of 3,5-DCP on fish.

2,3,4-TCP

No data were found on the effects of 2,3,4-TCP on fish.

2,3,5-TCP

There were no chronic data found on the effects of 2,3,5-TCP on fish and acute data were rare, but the lowest effect was a 24-h LC_{50} value of 0.8 mg/L in Salmo trutta. The lethal concentration to the bluegill sunfish, *Lepomis macrochirus*, was reported to be 0.45 mg/L (220). The toxicity of 2,3,5-DCP to the guppy, *Poecilia reticulata*, decreases from 0.882 to 4.74 mg/L as the pH rises from pH 6.1 to pH 7.8. The ratio of the LC_{50} at pH 7.8 over that at pH 6.1 is 5.4; these data are given in Tables 8.2.8.8 and 4.2.3.7 (144).

2,3,6-TCP

There were few data on the effects of 2,3,6-TCP on fish. The lethal concentration to the bluegill sunfish, *Lepomis macrochirus*, was reported to be 0.32 mg/L . The lowest chronic effect, to the fathead minnow, *Pimephales promelas*, was reported at 0.72 mg/L (220). No good LC_{50} data were available to estimate acute effects.

2,4,5-TCP

The toxicity of 2,4,5-TCP to the guppy, *Poecilia reticulata*, decreases from 0.987 to 3.060 mg/L as the pH rises from pH 6 to pH 8 (see Tables 4.2.3.7 and 8.2.8.8). The ratio of the LC_{50} at pH 8 over that at pH 6 is 3.1 (144). The lowest reported acute effect level of 2,4,5-TCP to fish was a 96-h LC_{50} of 0.45 mg/L at pH 7.2, 22C and a water hardness of 40 mg/L using the bluegill sunfish, *Lepomis macrochirus* (390). The lowest chronic response reported occurred at 1.0 mg/L of Dowicide-2 as a source of 2,4,5-TCP. This was a 4-h static bioassay at 12.8C using rainbow trout, *Oncorhynchus mykiss* (614).

2,4,6-TCP

Rainbow trout, *Oncorhynchus mykiss*, were exposed to bleached kraft mill effluents for 2 weeks at pH 7 ± 0.5, 8 to 10C, 7 ppt salinity and 2.5% effluent in Baltic Sea Water. There was a 10-day half-life for clearance of the 2,4,6-TCP found in the livers once the exposure ended. Wild fish were caught near the discharge from the mill and the 2,4,6-TCP in the fatty portion of their livers was assayed. A 200 g perch, *Perca fluviatilis*, contained 2.7 µg/g ; 370 g and 600 g northern pike, *Esox lucius*, contained 0.4 µg /g and 0.5 µg/g (187).

The toxicity of 2,4,6-TCP to the guppy, *Poecilia reticulata*, decreases from 0.61 to 7.859 mg/L as the pH rises from pH 5 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 5 is 12.9 (see Tables 4.2.3.7 and 8.2.8.8) (144). The lowest reported chronic effects, on enzyme function occurred at 0.2 mg/L in a 96-h test using rainbow trout, *Oncorhynchus mykiss* (245). The lowest reported acute effect, a 96-h LC_{50} of

0.32 mg/L, was found at pH 7.2, 22C, and a water hardness of 40 mg/L using the bluegill sunfish, *Lepomis macrochirus* (390).

3,4,5-TCP

There are few data on the effects of 3,4,5-TCP on fish. No chronic data were found and the lowest acute value was a 7-d LC_{50} value of 1.14 mg/L for the guppy, *Poecilia reticulata* (419).

TCPs (unspecified)

There is a report on various salmon species, *Oncorhynchus*, and the northern squawfish, *Ptychocheilus oreganensis*, indicating that values of 1, 5, or 10 mg/L of TCP are the 0.5 to 4-h $EC_{100}$and LC_{100} level at 10C to 20C in a static bioassay (615). The chronic effect noted was equilibrium loss.

2,3,4,5-TTCP

The lowest reported acute effect of 2,3,4,5-TTCP on fish is a 96-h LC_{50} of 0.205 mg/L in a flow -through bioassay at pH 6.9 to 7.7 using 10 g rainbow trout, *Oncorhynchus mykiss* (261). The lowest chronic effect reported is a 96-h EC_{50} of 0.75 mg/L using fathead minnow fry, *Pimephales promelas*, in a static bioassay at pH 6.5 to 7.9, 22C, and water with a hardness of 32 to 99 mg/L (261). As shown in Tables 4.2.3.7 and 8.2.8.8, the toxicity of 2,3,4,5-TTCP to the guppy, *Poecilia reticulata*, decreases from 0.442 to 2.32 mg/L as the pH rises from pH 6.1 to pH 7.8. The ratio of the LC_{50} at pH 7.8 over that at pH 6.1 is 5.2 (144).

2,3,4,6-TTCP

As shown in Tables 4.2.3.7 and 8.2.8.8, the toxicity of 2,3,4,6-TTCP to the guppy, *Poecilia reticulata*, decreases from 0.348 to 3.665 mg/L as the pH rises from pH 6 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 6 is 10.53 (144). Juvenile trout exposed to 2% bleached kraft mill effluent at pH 6.7 and hardness 20 mg/L accumulated 2,3,4,6-TTCP in the bile. In one 30-day trial, 99% was conjugated. In a 10-day trial with 0.6% effluent, 69% of the 2,3,4,6-TTCP was conjugated as gluconurides in the bile. In a third trial with larger fish for 6 days, the bile conjugates were 81% gluconurides and 25% sulphates; plasma had 63% conjugated and the rest free (422). The lowest reported acute effect on fish is a 96-h LC_{50} of 0.085 mg/L in a static bioassay using 74% technical grade 2,3,4,6-TTCP on rainbow trout, *Oncorhynchus mykiss* (236). The conditions included pH 7.2, 12C, and a water hardness of 44 mg/L.

If one suspects the contaminants in the 74% purity material of having an effect, then the next lowest effect reported for pure 2,3,4,6-TTCP is a 96-h LC_{50} of 0.14 mg/L in a static bioassay using bluegill sunfish fry, *Lepomis macrochirus* (390). The conditions included pH 6.5 to 7.9, 22C, and a water hardness of 32 to 99 mg/L. No chronic effects data were found.

2,3,5,6-TTCP

There were no chronic data on fish for 2,3,5,6-TTCP and acute data were rare. The lowest acute value reported was a 96-h LC50 of 0.17 mg/L for young bluegill sunfish, *Lepomis macrochirus* (390, 459). The conditions were pH 6.7 to 7.8, 22C, and water hardness of 32 to 48 mg/L.

As shown in Tables 4.2.3.7 and 8.2.8.8, the toxicity of 2,3,5,6-TTCP to the guppy, *Poecilia reticulata*, decreases from 0.39 to 3.94 mg/L as the pH rises from pH 6.1 to pH 7.8. The ratio of the LC_{50} at pH 7.8 over that at pH 6.1 is 10.1 (144).

PCP

The lowest reported acute response to PCP by fish is a 96-h LC_{50} of 0.018 mg/L using rainbow trout fry, *Oncorhynchus mykiss* (148). The fry were 77 days old and the conditions were pH 7.2, 10C, and water hardness of 50 mg/L. The lowest chronic response, a reduced growth rate in rainbow trout fry at 15C, occurred at 0.66 µg/L (126). There is some difficulty with interpreting the data in this paper and it has been classed as a secondary reference and not used to set the guidelines. The next lowest value is a chronic growth effect in Sockeye salmon at 15ºC and pH 6.8 (94). The estimated threshold NOEL is 1.74 µg/L and the LOEL is 3.49 µg/L. This is the data used to set the PCP guideline. As shown in Tables 4.2.3.7 and 8.2.8.8, the toxicity of PCP to the guppy, *Poecilia reticulata*, decreases from 0.107 to 0.906 mg/L as the pH rises from pH 6 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 6 is 8.5 (144).

In Cichlids there is an increased feeding rate, but decreased growth rate, when exposed to 200 µg/L of PCP (150). This reduction in conversion efficiency is quantified at 30% in largemouth bass exposed to 50 µg/L of PCP (151). Shiners, *Notropis cornutus*, had feeding rates up 57% at 56 µg/L PCP and 65% at 180 µg/L. Simultaneously the conversion efficiencies dropped about 50% at 180 µg/L due to uncoupled oxidation and hence reduced ATP formation (149). Growth rates dropped 25% at the 180 µg/L rate and 320 µg/L was fatal. *Puntius ticto* ate as much, but gained less weight, under sub-lethal levels of NaPCP. Food conversion efficiencies began to drop at levels as low as 1.5 µg/L (133). Sockeye salmon, *Oncorhynchus nerka,* had reduced growth rate and food conversion efficiency at 15C when PCP levels exceeded 2 µg/L (94). Rainbow trout, *Oncorhynchus mykiss*, at 15C, suffer growth effects at 0.66 µg/L PCP, but not at 0.035 µg/L (203). Rainbow also accumulate PCP up to 5 times over controls on the same diet, when exposed for 115 days to 0.035 µg/L. There was no effect seen on growth or weight of the fish (126). Female rainbow exposed to 22 or 49 µg/L of 99% PCP for 18 days had lower oocyte viability (146).

In laboratory fish, the bluegill, *Lepomis macrochirus,* has the highest concentration of PCP in the bile, followed by liver, gills, and muscle (13). Experiments on *Notopterus notopterus* for 10, 20 or 30 days, at 13.6, 20.4, 40.8 and 60.2 µg/L of PCP, all showed effects on hepatic acid phosphatase, alkaline phosphatase, and succinic acid dehydrogenase. Liver activity was reduced over that in controls, mostly in hepatic acid phosphatase over 30 days (1).

The 1/2 life of PCP in gold fish, *Carassius auratus*, is 10 hours, while that of the unchlorinated parent compound, phenol, is less than 1 hour. In *Notopterus notopterus*, 15- and 30-day exposures to 8, 6 and 4 µg/L of NaPCP caused significant reductions in 5-nucleotidase activity, which is indicative of uncoupled oxidative phosphorylation. Kidney enzyme activity was not significantly different due to stimulation of the kidneys as they tried to compensate for lost efficiency elsewhere (145).

In the mullet-Rhinomugil corsula, carp-*Cyprinus carpio*, and cichlid-*Tilapia mossambica*, the metabolic rate rose at 100 µg/L of NaPCP in the water (138). As shown in Table 8.2.8.8 for the guppy, *Poecilia reticulata*, the toxicity of PCP decreases as the pH rises from pH 6 to pH 8. The ratio of the LC_{50} at pH 8 over that at pH 6 is 8.4 and the toxicity of PCP at pH 8 is 10 times that of 4-MCP at pH 8 (144).

There is a block of data by Mayer et al. in 1986 (111) on the bluegill, *Lepomis macrochirus*, in Table 8.2.8.3, which shows that 96-hour LC_{50}s for PCP are lower than 24 -hour LC_{50}s, and that harder water does afford some protection against PCP toxicity. In Table 8.2.8.4 there are data by Gupta (107), on *Rasbora daniconius neilgeriensis* showing that, for PCP, LC_{16} values are smaller than LC_{50}values, which are in turn smaller than LC_{84} values for any given exposure time; this is expected and indicates internal consistency in the data set. Furthermore, as exposure times increase the LC_{50}decreases, also an expected result. The no-effect level is about 1/7 of the 96-h LC_{16}, 1/15 of the 96-h LC_{50}, and 1/33 of the 96-h LC_{84}; this is approximately the same change, a factor of two, each time, and represents internal consistency in the data set. Since the LC_{16} and LC_{84} limits represent 1 standard deviation about the mean value of LC_{50}, and 1 standard deviation is 68% of the animals, there are 34% of the animals between the LC_{16} and the LC_{50} and between the LC_{50} and the LC_{84} . A standard factor for estimating no chronic effects from LC_{50} data is 1/20 or 0.05; these data indicate that 0.010/0.148 or 0.067 is an appropriate value for PCP.

In Table 8.2.8.5 there is a block of data on *Pimephales promelas*, the fathead minnow, from Hedtke et al., 1986 (113), which indicates that rising temperatures increase the toxicity of PCP. Data from Spelar et al., 1985 (79) show the increasing toxicity of PCP with decreasing pH.

In Table 8.2.8.6 a block of data on *Notopterus notopterus* from Gupta et al., 1982 (132) show that increasing fish size reduces toxicity to NaPCP and that with increasing exposure times the toxicity increases.

More data from Gupta et al., 1983 (142) show that higher temperatures increase toxicity and that increasing exposure time increases toxicity. The mean of 6 NOEL levels is 0.037 (range 0.032 to 0.043) of the 96-h LC_{50}; the values vary slightly for fish from different size classes and temperatures.

In goldfish, *Carassius auratus*, ambient PCP and pH levels affect the rate of uptake of PCP and the bioaccumulation factor, but ultimately, however long it takes, or under whatever conditions, fish begin to die when the body load reaches about 90 µg/g (547). Trout tissues do not methylate PCP, but the bile contains 250 µg/g of PCP glucuronide conjugate when the trout are subject to 26 µg/L PCP in the water (134, 141). Guppies, *Lebistes reticulatus*, previously acclimated to 1.0 and 0.1 mg/L NaPCP for 10 days, can survive 3 to 8 h in 5.0 mg/L NaPCP, which would be immediately lethal to un-acclimated fish (670). *Campostoma anomalum*, (silver-mouthed and blunt-nosed minnows), were able to detect and avoid NaPCP above 10 mg/L but not below 5 mg/L. Eggs of lake trout, *Cristivomen namaycush*, were more resistant than mature fish, but yolk sac fry were the most sensitive stage (69).

Steelhead trout, *Oncorhynchus mykiss*, were exposed to NaPCP at various life stages. When exposed to 0.3 mg/L post-fertilization, 100% mortality occurred within 1 week of fertilization; at 0.05 mg/L 100% mortality occurred within 24 hours of hatch. Alevin weight at hatch decreased and hatch was delayed. In 5-day tests, alevins died within 24 hours at 0.2 mg/L. Exposure between fertilization and yolk sac absorption caused 100% mortality at 0.04 mg/L, but little at 0.02 mg/L or 0.01 mg/L. However, if the dissolved oxygen was dropped to 5 mg O2/L, then 0.02 mg/L NaPCP was 100% lethal and at 3 mg O2/L, 0.01 mg/L NaPCP was 100% lethal. These NaPCP levels caused little mortality at normal O2 levels or if NaPCP was not present; the combination of NaPCP and low O2 was lethal.

NPCP and KPCP

The sodium salt is most commonly used but the potassium salt is used occasionally. The lowest reported acute effect is a 96-h LC_{50} of 9.8 µg/L using 9 cm long Notopterus notopterus (142). The experiment was carried out at 36C, pH 7.2, and 6.5 mg/L of dissolved oxygen. For chronic effects the lowest reported value is an EC50 of 1.74 µg/L for reduced growth rates of sockeye salmon, Oncorhynchus nerka (94).

8. 4 Guidelines from the Literature

Table 8.4.1 lists some non-organoleptic literature guidelines for chlorophenols with respect to aquatic life. It is evident that not all congeners have had guidelines set, that the reasons for setting guidelines vary, and that the guidelines levels vary markedly. Acute and chronic effects have both been used as end points. Time periods vary from instantaneous maxima to 96-h means. Some agencies set guidelines for specific congeners and others group the chlorophenols into homologues of the same number of chlorine atoms, isomers, and set a guideline for the homologous group. The 1987 CCREM and 1984 Ontario guidelines are for sums of chlorophenol isomers and not for individual chlorophenols. As is evident from Figure 2.4 there is considerable variation in toxicity within a group of isomers and we feel that individual chlorophenol guidelines should be set to reflect this variability. The alternative is to set guidelines for each isomer group low enough to protect against the most toxic member of the group; this would be excessively overprotective for some congeners. The BC Ministry of Environment has established site specific objectives for the sum of tri-, tetra-, and pentachlorophenols in fish, of 0.1 µg/g wet weight in the muscle tissue (536).

In the EPA-1986 criteria document, a recurring phrase is "toxicity occurs at concentrations as low as...and would occur at lower concentrations among species that are more sensitive than those tested". To get around this lack of data on more sensitive species, we have used the ratio of toxicity of PCP to that of the other congeners for *Daphnia*. This provides an estimate of the toxicity of the other congeners to the most sensitive species. There are no suitable data in the literature on which to base guidelines for most congeners, only calculated ratios between existing PCP data and the other congeners as determined by an experiment on *Daphnia*. There are also ratios inherent in the equations of Saarikoski et al. (144) derived from work on guppies and chlorophenols.

8.5 Recommended Guidelines

TOXICITY CRITERIA

Raw toxicity guidelines for the chlorophenols, calculated using the equations of Saarikoski et al. (144), are given in Table 8.5.1, in µg/L at 10 C between pH 5.6 and pH 8.4 . The guidelines at pH 7.2 and 10C are shown graphically in Figure 8.5.1. Table 8.5.1.1 gives the constants and variables needed to calculate these numbers. Considering the inherent variability of the values in Table 8.5.1, based on how they were generated, the implied precision does not seem warranted.

In addition, application of the guidelines would be unnecessarily complex with little if any concomitant increase in environmental protection. Guidelines were also calculated based on rainbow trout LC_{50}data. These guidelines are both based on acute data and are found in Table 8.6.6. The recommended maximum guidelines, given below and in Table 1.1.2 and Table 8.6.6 were derived from the chronic data of Webb and Brett (94) by calculating an NOEL from the LOEL. The derivation process is outlined in section 8.6. All these derivation processes lead to the same values for the guidelines.

Interim Aquatic Life Toxicity Guidelines ** for Chlorophenols.
(calculated in µg/L, at 10ºC * , from pH 5.7 to pH 9.2).

Chlorophenol Congeners	pH 5.7	pH 6.2	pH 6.7	pH 7.2	pH 7.7	pH 8.2	pH 8.7	pH 9.2
2-MCP	3.9	6.4	11	17	29	48	79	130
3-MCP	3.4	5.6	9.3	15	25	42	70	115
4-MCP	1.7	2.9	4.8	7.8	13	22	36	59
2,3-DCP	1.1	1.8	3.1	5.1	8.3	14	23	38
2,4-DCP	0.6	1.0	1.6	2.6	4,3	7.2	12	20
2,5-DCP	0.5	0.8	1.4	2.3	3.7	6.2	10	17
2,6-DCP	2.0	3.3	5.5	9.1	15	25	41	68
3,4-DCP	0.6	1.0	1.6	2.7	4.4	7.4	12	20
3,5-DCP	0.5	0.7	1.2	2.0	3.4	5.6	9.2	15
2,3,4-TCP	0.5	0.8	1.3	2.2	3.6	6.0	9.9	16
2,3,5-TCP	0.5	0.8	1.3	2.2	3.7	6.1	10	17
2,3,6-TCP	1.6	2.6	4.4	7.2	12	20	33	54
2,4,5-TCP	0.5	0.7	1.2	2.0	3.3	5.6	9.2	15
2,4,6-TCP	1.2	1.9	3.2	5.3	8.8	15	24	40
3,4,5-TCP	0.2	0.3	0.5	0.9	1.4	2.4	3.9	6.4
2,3,4,5-TTCP	0.4	0.6	1.0	1.7	2.8	4.7	7.8	13
2,3,4,6-TTCP	1.1	1.8	2.9	4.9	8.0	13	22	36
2,3,5,6-TTCP	0.5	0.8	1.3	2.2	3.6	6.1	10	17
2,3,4,5,6-PCP	0.2	0.3	0.5	0.7	1.2	2.0	3.4	5.5

* multiply the table values by 2 at 0ºC and by 0.5 at 20ºC.
** These guidelines are maximum values, rounded to 1 decimal.

Water Level Guidelines

The level of MCPs in the water should not exceed 0.1 µg/L.

The level of DCPs in the water should not exceed 0.2 µg/L.

The levels of TCPs, TTCPs and PCP in the water should not exceed the aquatic life toxicity guidelines as given above.

8.6 Rationale

TOXICITY CRITERIA

To help analyze the vast amount of data found in Tables 8.1.2 through 8.2.8.12, four summary tables were constructed. These tables, 8.6.2 to 8.6.5, list, for each chlorophenol congener, the lowest literature value found for freshwater and marine organisms and for chronic effects or acute LC_{50}s.

There are gaps in these tables, especially for marine life, where no experimental data exist for any life form for a given congener. As discussed elsewhere in this document, in section 5.8.1, the action of chlorophenols on all eukaryotic life is the same and the site of action is the mitochondrion. The ratios of the toxicity of the chlorophenols to the toxicity of PCP is primarily a function of their Ko/w values, but some QSAR relationships modify this response, as does the pKa value.

Choosing an Appropriate Organism and Data Set

Fish, generally salmonids, are usually the most sensitive aquatic organisms reported; however, few data are available on tadpoles, newts and salamanders which are also intimately exposed to the water, especially in their juvenile stages. In Table 8.6.2 most of the lowest reported LC_{50}s are for fish and only one is for a crayfish. For the tadpole of the frog, *Rana catesbiana*, the datum given was a 48-h LC_{50} of 0.207 mg/L. There is more reliable LC_{50} rainbow trout datum by Leeuwen et al. of 18 µg/L (148) found in Table 8.2.8.7. Niimi et al. (126) reported chronic effects of PCP on rainbow trout at 0.66 µg/L and an NOEL of 0.035 µg/L. This was determined to be secondary data and therefore not used to set the final guidelines. A paper by Webb and Brett (94) gives an LOEL of 3.49 µg/L for growth effects of PCP on young sockeye salmon and this was the value on which the final guidelines were set.

Table 8.6.1 is a collection of calculated or experimental no observed effect levels, (NOEL) and non-lethal levels of chlorophenols for aquatic life. It indicates the level at which no toxic effects were found for each congener and also indicates the range of values found by different authors for different species. There are no other toxicity data on other more sensitive species, and only incomplete data for many other species, for the other chlorophenol congeners. The toxicities of the other congeners would have to be estimated by using the ratio of toxicity between PCP and the other congeners in those few cases where complete, or near complete studies, were carried out. This process is outlined below.

SETTING TOXICITY RATIOS - PCP TO OTHER CHLOROPHENOLS

1-Estimating the 96-h LC_{50} to NOEL conversion factor

CCME has used 0.01 but a standard factor for estimating no chronic effects from 96-h LC_{50} data is 1/20 or 0.05. Only paired NOEL and 96-h LC_{50} data from the same PCP experiment are used in the calculations below. Table 8.2.8.6 contains a block of PCP data on the fish *Notopterus notopterus* from Gupta et al., 1982 (132) and 1983 (142). In µg/L, the paired NOEL and 96-h LC_{50} data are: 0.0011/0.032, 0.003/0.093, 0.0035/0.083, 0.0037/0.107, 0.0045/0.131, and 0.00043/0.0098.

The mean of these 6 ratios, NOEL levels/ the 96-h LC_{50} levels, is 0.037; the individual ratios are 0.034, 0.032,0.042,0.034,0.034 and 0.043, respectively. The values vary slightly for fish of different size classes

and different experimental temperatures. In Table 8.2.8.4 there are PCP data by Gupta in 1983 (107), on *Rasbora daniconius neilgeriensis* . These data indicate that 0.010/0.148 or 0.067 is an appropriate ratio for PCP. The mean of these 7 NOEL /96-h LC_{50} ratios is 0.041. These data were also discussed above in section 8.2.8 under PCP.

2-Daphnia magna data

To set guidelines for each of the chlorophenol congeners, starting from data sets with gaps in them and data of variable quality, the following procedures were followed. The most complete data set, that of Devilliers et al., 1986 (57) in Table 2.4 on *Daphnia magna*, was used as a starting point. It was used to establish the ratio of the guideline for any congener to that of PCP. The two gaps in this data set were filled by extrapolation using QSAR arguments from the data set itself, and by using calculated ratios from Saarikoski et al., 1982 (144). This process was documented, step-by-step, in Table 8.6.6 and is described below.

In Table 8.6.6, Column 1 gives the ratios of the toxicity of PCP to other chlorophenols for *Daphnia*, as determined from experiments by Devillers et al. (57). The most complete set of data on chlorophenol toxicity exists for PCP, so the lowest reported 96-h LC_{50} datum for PCP was chosen as the starting point for calculations. This was the 18 µg/L value for the rainbow trout as indicated above. This value was multiplied by the appropriate ratios to derive 96-h LC_{50} values for the other chlorophenols and the values given in Column 2. Data from Tables 8.2.8.4 and 8.2.8.6, as discussed above, indicate that for pentachlorophenol, the mean no-effect level appears to be about 0.041 of the 96-h LC_{50} value. This correction is made to Column 2 to yield Column 3, which should, in the absence of good experimental data, provide an adequate estimate of the lowest no-effect level of chlorophenols to rainbow trout. The guidelines developed by this method are only good at the pH of the experiment, 7.8 to 8.2, and can not be readily converted to other pH values; thus they are of limited value.

3-Guppy data

Using a series of equations developed by Saarikoski et al. (144) for guppies, one can derive guidelines for any given pH. When this is done at pH 8.0 and the results, column 6, Table 8.6.6, compared to the results, column 3, Table 8.6.6, generated using the data of Devilliers et al. (57) for *Daphnia magna*, it can be seen that there is reasonably good correspondence. The data of Saarikoski et al. (144) were developed using guppies, which are less sensitive than rainbow trout.

Experiments indicated that rainbow trout, *Oncorhynchus mykiss*, were about fifty times more sensitive to chlorophenols than guppies, *Poecilia reticulata*, for which the above pH relationships were determined (144). The lowest LC_{50} value for rainbow trout is 18 µg/L at 10C and a pH of 7.2. The water hardness was 50 mg/L and the animals were 77-d old fry. A correction factor is needed to convert the results of the equations, which are guppy LC_{50} values, to rainbow trout LC_{50} values. The procedure therefore was to calculate the guppy LC_{50} value at 26C and pH 7.2 using the equations in Saarikoski et al. (144).

This value was 1.38 mg/L. Converting it to a guppy LC_{50} value at 10C and pH 7.2 results in a value of 4.18 mg/L. Since the lowest rainbow trout LC50 value at 10C and pH 7.2 is 0.018 mg/L (148), a conversion factor of 0.00431 (0.018/4.18) needs to be applied to the equations derived from guppy data.

4-Corrections for pH and Temperature

Temperature and pH corrections need to be applied to the base guidelines calculated at 10C and a pH of 7.2. The temperature correction factor is not difficult to apply; the raw guidelines in Table 8.5.1 and interim guidelines in Table 8.6.6 are given for 10C which can be converted to any other desired temperature. However, the pH can not be simply converted. It has to be recalculated using an inconvenient equation and thus Table 8.5.1 was generated for the Saarikoski guidelines in the range pH 5.6 to pH 8.4 ; the equation is not valid outside this range, and tends to under and over estimate toxicity at low and high pH values, respectively.

A-Temperature Corrections

Calculated raw toxicity guidelines for the chlorophenols, as given in Table 8.5.1, are in µg/L at 10 C for pHs between 5.6 and 8.4 . The Saarikoski guidelines at pH 7.2 and 10C are shown graphically in Figure 8.5.1. The temperature effects were estimated using the data in Table 8.2.8.6 taken from Gupta et al., 1983 (142) using the fish, *Notopterus notopterus*. The ratio of toxicity at 16C to that at 36C was 11 at 24 hours, 9 at 48 hours, 7 at 72 hours and 6 at 96 hours. It declines with length of exposure and one assumes it would equilibrate at some value not far below 6 for long-term exposures. The normally accepted temperature effect on metabolic rates is a factor of 2 for a 10C change. Thus if the temperature rose from 10C to 20C the values would be divided by 2. This temperature dependence rate is one that is well established in physiological studies and should need no further substantiation.

Thus for the 20C difference in this experiment one would expect a change of 2 x 2 = 4, which is close to the 6 found at 96 hours, and 4 is probably a good estimate for long-term equilibrium conditions. Graphing these data indicate that a value of 4 should be reached within a week. For fish, Mayer et al. (111) found the following relationship for a 10C temperature change with all pollutants, except organophosphates:

$$\log \text{96-hour } LC_{50} \text{ (@T)} = \log \text{96-hour } LC_{50} + 0.4956 \text{ (@T+10)}.$$

This change is about a factor of 3 for a 96-hour period and is not far off the expected long-term value of 2 for a change of 10C. The temperature correction equation is given below:

$$\text{Guideline at YC} = (\text{Guideline at 10C}) \,.\, (2 - x)$$

where Y = the new temperature, and x = (Y- 10) / 10.

B-pH Corrections

Correction for different pH levels is more complex and guidelines at 10C for pHs in the pH 5.6 to pH 8.4 range are given in Table 8.5.1 for the raw Saarikoski guidelines. These would have to be considered interim guidelines until properly controlled experiments could provide a unified temperature and pH dependent regression equation for each chlorophenol. Table 8.5.1.1 gives the constants and variables needed to calculate these numbers. The full equation for calculating guidelines in µg/L, at any pH between about 5.6 and 8.4, and for any reasonable physiological temperature (1C to 36C), is given below : the guideline =

(GMW) (0.054) (1 / (antilog ((0.67 log P + 0.19 (10.05 - pKa) - 0.67) -

(log (4 (pH - pKa)+1)))) (1/2 ((T - 10) /10)) .

Values for the variables, GMW, logP, and pKa are given in Table 8.5.1.1 . T and pH are the desired temperature in C and pH, respectively. GMW is the molecular weight of the chlorophenol, logP is the logarithm (base 10) of the octanol/water partition coefficient, and pKa is the acid dissociation constant.

There is another pH conversion equation for PCP developed by the EPA which is easier to use and gives nearly the same results, the pH slope factor is very similar. It is probably the formula of choice for PCP but not necessarily for the other chlorophenols.

newconc. = eY

where Y = (ln (givenconc.) - 1.005 (given pH -new pH)) and newconc. is the concentration at the new pH and givenconc. is the concentration at the given pH.

Hardness does not seem to have a consistent, predictable effect on the toxicity of chlorophenols; however, in natural waters pH and hardness would normally be well correlated. There are some data for 4-MCP, 2,4,6-TCP and PCP over the range pH 5 to pH 8 in Table 8.2.8.8. There are also data on many of the other congeners over the pH range 6 to 8. The best set of data to determine the effects of pH on chlorophenol toxicity is that of Saarikoski et al., 1982 (144) for guppies which is found in Table 8.2.8.8. The Ko/w and the pKa of the chlorophenols are factors which affect pH-related toxicity, since it is the effect of pH on lipophilicity which is important. Toxicity is generally a function of how readily the chlorophenol molecule can gain access to the interior of the organism and this is determined primarily by the amount of dissociation of the chlorophenol molecule. At pH levels below the pKa, dissociation is almost nil and uptake is rapid; at higher pH levels, dissociation is virtually complete, uptake and toxicity are relatively low, and changes in uptake with a change in pH are relatively small. Most existing regression equations are only good in the approximate range pH 6.0 to pH 8.0. A regression equation was derived by Saarikoski et al. (144) using 8 of the 19 chlorophenols, which predicted toxicity at pH 7.0 as a function of the Ko/w. To find the toxicity at some other pH, in the range pH 6.0 to pH 8.0, the pKa is required.

5-Comparison of Guidelines Derived from LC_{50}, Chronic or NOEL Data

Niimi et al. (126) indicate, as shown in Table 8.2.8.7, that there are chronic effects of PCP on rainbow trout fry growth rates at 0.66 µg/L . This level is almost identical to the calculated chronic toxicity threshold of 0.7 µg/L as given in column 3 of Table 8.6.6. Whether one uses NOEL data from the literature, applies a 0.1 safety factor to the lowest literature chronic effect, or applies an acute-to-chronic ratio factor to the lowest LC_{50} data from the literature, the result is nearly the same. Inspection of Tables 8.6.2 to 8.6.5 indicates that the calculated guidelines in Column 5 are below any reported toxic effect on any organism for any chlorophenol congener. In addition, these guidelines are below guidelines set by other agencies (Table 8.4.1) for all chlorophenols. In Table 8.6.6, Column 6, calculated guidelines using the equations of Saarikoski et al. (144), based on guppies, are given and it is evident that there is reasonably good correspondence between these values and those in columns 3 and 5 which are derived from work by Devillers et al. (57) on *Daphnia* and Webb and Brett (94) on Sockeye Salmon. The values in columns 3, 5 and 6 were calculated for 10C and a pH of 7.2, since these were the conditions under which the rainbow trout LC_{50} data were generated. Since the guidelines derived using *Daphnia* data from

Devillers et al. (57) can not be applied to other pH values, while the guppy data from Saarikoski et al. (144) can be recalculated for any desired pH, and since there is good agreement between these two methods, the equations of Saarikoski et al. (144) would be better to generate guidelines from acute data.

6-Deriving Recommended Guidelines from the Raw Guidelines

Inspection of Table 8.5.1 shows that for some chlorophenols, mostly lower molecular weight ones, there is relatively little difference in the raw guidelines at low and high pH values. For other chlorophenols the change is quite small at low pH levels but becomes larger at higher pH levels. When one considers the inherent variability of the numbers in this table, based on how they were generated, such precision does not seem to be warranted. In addition, application of the guidelines would be unnecessarily complex with no concomitant increase in environmental protection. Guidelines were derived from the numbers in Table 8.5.1 by the following procedure.

The lowest value, at pH 5.6, was multiplied by 4 to establish an upper limit to a pH range:

- If the resulting number was greater than the Table 8.5.1 value for pH 8.4 then only one guideline was set for the chlorophenol; this guideline is applicable at any pH and was set at the mean between the Table 8.5.1 values at pH 5.6 and pH 8.4. Thus the guideline would not be more than a factor of 2 different from the Table 8.5.1 value at any pH.
- If the resulting number was less than the Table 8.5.1 value for pH 8.4, then several guidelines were necessary for that chlorophenol to reflect the larger change in toxicity with changing pH. For the range of pH 5.6 to the next higher pH after which the Table 8.5.1 value reaches 4 times the value at pH 5.6, the guideline was set at twice the Table 8.5.1 value at pH 5.6. Thus the guideline would not be greater than a factor of 2 different from the Table 8.5.1 value at any pH in the defined range. This process was then repeated, starting with the Table 8.5.1 value at the lowest pH in the next range.

No temperature corrections were made since the change from the guideline derived for 10C, would be less than a factor of two for any ambient temperatures encountered in BC.

7-Assumptions made in deriving guidelines in this manner

1. The ratio of toxicity of any given chlorophenol to that of PCP is the same, or nearly so, in species other than guppies, Saarikoski et al. (144), or Daphnia magna, Devillers et al. (57). Discussed under "Setting Toxicity Ratios - PCP to Other Chlorophenols ".
2. The pH response curve found for guppies, Saarikoski et al. (144), holds true for other species. Discussed under "pH Corrections".
3. The temperature dependence curve found for the fish Notopterus notopterus, Gupta et al. (142), holds true for other species. Discussed under "Temperature Corrections".
4. Temperature, pH or other corrections to calculated guidelines, which are less than a factor of two, are not justified due to the approximate nature of the numbers derived. Discussed under "Deriving Recommended Guidelines from the Raw Guidelines".

8-Derivation of the guidelines using the chronic data of Webb and Brett (94).

To determine the base value for PCP, from which the other chlorophenols are determined by ratio, the preferred CCME protocol is followed, using sockeye salmon growth data. The LOEL is 3.49 and a factor of 0.1 is applied to this to get an estimate of the NOEL of 0.35 at a pH of 6.8 and a temperature of 15ºC. These values were converted to 10ºC and a pH of 7.2 and are found in Table 8.6.6 in columns 4 (LOEL) and 5 (NOEL). The marine guideline is simply the same data calculated at a pH of 8.2.

9. IRRIGATION

No data were found documenting the effects of chlorophenols on irrigation uses of water. Terrestrial plants appear to be less sensitive to chlorophenols than aquatic organisms; any effects found were in the mg/L range. Water suitable for aquatic life or drinking should also be suitable for irrigation. See Section 7.2 and Table 7.2 for the few data that were found documenting the effects of chlorophenols on terrestrial plants. No guidelines were set for this use.

10. RECREATION

10.1 General

There are no data documenting the effects of chlorophenols on recreational uses of water. Human taste and odour thresholds for some chlorophenols in water are available, Tables 6.2.1 and 6.2.2, as are taste thresholds for some chlorophenols in fish meat, Table 8.3.1. No taste thresholds for crustacean or mollusc meat are published for the chlorophenols.

PRIMARY CONTACT RECREATION

Since swimming involves intimate contact of the face with the water, taste and odour thresholds for the chlorophenols should be met in water used for swimming. Since these thresholds are the critical factors determining drinking water guidelines, the drinking water guidelines would give adequate protection to waters used for swimming.

10.2 Recommended Guidelines

Water used for Primary Contact Recreation should meet Drinking Water Guidelines.

10.3 Rationale

The chlorophenols have objectionable tastes and/or odours above the drinking water guidelines. Such tastes and odours would also affect swimmers who have their faces immersed in water. The lowest reported odour thresholds for chlorophenols in water are 0.33 µg/L for MCPs and DCPs, 11 µg/L for TCPs, 600 µg/L for TTCPs and 857 µg/L for PCP (Table 6.2.2). These values are close to the aquatic life

guidelines for the smaller, less-substituted, more volatile MCPs and DCPs, but are very much higher than the aquatic life guidelines for the larger, saturated, less volatile PCP.

11. INDUSTRIAL

No data were found documenting the effects of chlorophenols on industrial uses of water. Due to taste and odour concerns, the food and beverage industries would have to use the drinking water guidelines, and the aquaculture industry would have to meet the aquatic life guidelines. A few other industries with a need for very high quality water, such as photofinishing, would likely have to use point of use treatment to keep chlorophenols below acceptable levels, if the local supply was not adequate.

12. RESEARCH NEEDS

The guidelines given for aquatic life are recommended as interim, except for PCP, due to the way in which they were estimated. What is needed is one comprehensive experiment which tests all the chlorophenols under the same set of conditions and generates a single regression equation for each chlorophenol based upon pH and temperature.

Nothing is known about the effects of chlorophenols on the industrial uses of water and some research should be undertaken in this field. Similarly, nothing appears to be known about effects on crop plants when chlorophenols are present in the irrigation water and a series of experiments should be conducted in this area.

Synergistic effects of chlorophenols with other organic compounds or with inorganics, especially those which also affect the mitochondrial enzymes, have apparently not been investigated. One would expect some effects in this area and research should be conducted to determine the severity of the response and which other compounds are important.

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Cserjesi et al. 1972 (17)
Cserjesi et al. 1975 (657)
Cunningham et al. 1986 (435)

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Davis et al. 1975 (281)
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Deichmann et al. 1948 (652)
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De Kreuk et al. 1981 (353)

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Dutton et al. 1986 (47)

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Ehrlich 1990 (747)
Elder et al. 1981 (593)
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Erickson et al. 1980 (562)
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Ernst et al. 1978 (642)
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Evans 1971 (441)
Exon et al. 1982 (739)
Exon 1984 (741)

Faas et al. 1979 (683)
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Fahrig et al. 1978 (563)
Falk et al. 1973 (640)
Fandry et al. 1989 (711)
Farquharson et al. 1958 (229)
Fetterolf 1964 (676)
Fiedler et al. 1990 (715)
Fielder 1982 (314)
Fields et al. 1966 (515)
Firestone 1977 (295)
Firestone et al. 1972 (37)
Fisher 1985 (5)
Fisher et al. 1986 (6)

Fleming 1946 (171)
Fogels et al. 1977 (211)
Folke et al. 1984 (533)
Fountaine et al. 1976 (299)
Forlin et al. 1978 (574)
Fox 1983 (685)
Fox 1978 (212)
Fox 1988 (176)
Fox et al. 1978 (59)
Fox et al. 1984 (291)
Fox et al. 1988 (177)
Freitag et al. 1982 (597)
Freiter 1979 (293)
Fudolej et al. 1978 (510)

Gaines 1969 (540)
Garrett 1980 (594)
Garrett et al. 1988 (289)
Gee et al. 1974 (534)
Geiger et al. 1988 (402)
Gelfand 1941 (163)
Gersdorff et al. 1940 (392)
Gilbert et al. 1990 (754)
Glaze et al. 1978 (595)
Gleason et al. 1976 (476)
Glickman et al. 1977 (134)
Goldstein et al. 1977 (35)
Goodnight 1942 (69)
Gotham et al. 1982 (764)
Goto et al. 1972 (484)
Goto et al. 1972 (482)
Graney et al. 1987 (252)
Grayson et al. 1979 (442)
Green 1978 (233)
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Guo et al. 1979 (679)
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Gurova et al. 1979 (679)

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Hall et al. 1985 (109)

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Harper et al. 1975 (32)
Harrison 1959 (323)
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Harvey et al. 1952 (213)
Hashimoto et al. 1982 (157)
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Hattula et al. 1981 (324)
Hattula et al. 1985 (529)
Hauch et al. 1980 (78)
Hawley 1977 (445)
Hedtke et al. 1985 (103)
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Heitmuller et al. 1981 (393)
Helling 1971 (507)
Hemmingsen 1960 (589)
Henderson 1992 (705)
Henderson et al. 1951 (167)
Herve et al. 1988 (718)
Hiatt et al. 1960 (210)
Hinkle 1973 (564)
Hirsch 1942 (332)
Hoak 1957 (463)
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Olivier et al. 1960 (215)
Omura et al. 1971 (339)

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Parker et al. 1980 (316)
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Pierce et al. 1978 (456)
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Pitter 1976 (185)

Pomazoreskaya 1978 (552)
Pruitt et al. 1977 (121)

Rao et al. 1978 (74)
Rao et al. 1981 (130)
Rapson et al. 1980 (430)
Rasanen et al. 1977 (231)
Reiner et al. 1977 (49)
Renner et al. 1983 (452)
Reteuna et al. 1986 (46)
Ribo 1987 (239)
Ribo et al. 1983 (367)
Roberts 1963 (560)
Robinson et al. 1983 (208)
Robson et al. 1969 (664)
Rogers et al. 1988 (203)
Rosenburg 1980 (491)
Rott et al. 1979 (21)
Rowe et al. 1982 (3)
Ruckdeschel et al. 1986 (432)
Rudd et al. 1956 (162)
Rudling 1970 (280)
Ruesink et al. 1975 (623)
Ryssov-Nielson 1975 (512)

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Saarikoski et al. 1982 (144)
Saarikoski et al. 1986 (153)
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Sax 1984 (426)
Schiff et al. 1961 (666)
Schimmel et al. 1978 (119)
Schipper 1961 (659)
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Schmidt 1992 (733)
Schmidt 1992 (734)
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Schultz 1983 (455)
Schultz 1986 (152)
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Schultz et al. 1986 (260)

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Sloof et al. 1983 (112)
Sloof et al. 1983 (333)
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Smith et al. 1991 (763)
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Spehar et al. 1985 (79)
Spencer 1957 (661)
Spokes et al. 1974 (439)
Springer 1957 (166)
Statham et al. 1975 (553)
Stehl et al. 1972 (296)
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Stofen 1974 (638)
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Thomas 1973 (370)
Thompson et al. 1986 (179)
Thursby et al. 1986 (2)
Thurston et al. 1985 (114)
Tomiyama et al. 1962 (72)
Trabalka et al. 1979 (368)
Tranvik et al. 1991 (700)
Trevors 1982 (308)
Trevors 1982 (609)
Trevors et al. 1981 (557)
Trevors et al. 1982 (519)
Triebig et al. 1981 (721)
Triebig et al. 1987 (722)
Trujillo et al. 1982 (10)
Turnbull et al. 1954 (633)
Turner et al. 1948 (164)
Tyler et al. 1974 (511)

Vallejo-Friere et al. 1954 (627)
Van Dyk et al. 1977 (73)
Van Horn 1943 (632)
Vasseur et al. 1986 (178)
Vaugh 1954 (668)
Veith 1980 (423)
Veith et al. 1979 (610)
Veith et al. 1979 (612)
Vela et al. 1976 (15)
Verma et al. 1982 (154)
Vermeer et al. 1974 (644)
Verscheuren 1977 (424)
Villanueva et al. 1975 (36)
Virtanen et al. 1982 (604)
Vizethum et al. 1979 (477)
Vogel et al. 1974 (286)

Walden et al. 1986 (714)
Walker 1954 (438)
Walker 1988 241)

Wan et al. 1990 (415)
Wan 1992 (719)
Ware 1988 (405)
Waters et al. 1982 (408)
Webb et al. 1973 (94)
Weber 1965 (70)
Weber et al. 1978 (643)
Weeden 1993 (729)
Weinbach 1954 (61)
Weinbach 1955 (60)
Weinbach 1956 (62)
Weinbach 1956 (64)
Weinbach et al. 1950 (75)
Weinbach et al. 1965 (63)
Wellens 1982 (396)
Welsh et al. 1987 (740)
Wegman et al. 1983 (349)
Whitley 1968 (334)
Williams 1982 (99)
Winner 1988 (417)
Woelke 1972 (120)
Wolf 1974 (470)
Wong et al. 1977 (221)
Wong et al. 1981 (20)
Wyllie et al. 1975 (573)

Xu 1996 (742)

Yasuhara et al. 1977 (338)
Yoshioka et al. 1978 (237)
Yount et al. 1986 (116)
Yunker 1981 (689)

Zischke et al. 1985 (102)
Zitko et al. 1974 (31)
Zoeteman 1975 (639)

Table 1.1.1 Summary: Water Quality Guidelines for Chlorophenols

Water Use	Guidelines (µg/L, max.)	
Raw Drinking Water		
aesthetic guidelines (taste and odor)	MCPs *	0.1
	DCPs*	0.3
	TCPs*	2
	TTCPs*	1
	PCP	30
toxicity guidelines	2,4-DCP	900
	2,4,6-TCP	5
	2,3,4,6-TTCP	100
	PCP	60
Wildlife and Livestock Water		
aesthetic guidelines	use raw drinking water aesthetic guidelines	
toxicity guidelines	MCPs	1600
	DCPs	4000
	TCPs	1800
	TTCPs	3500
	PCPs	1500
Aquatic Life: Fresh/Marine/Estuarine		
toxicity guidelines for aquatic life	all CPs	use Table 2
Irrigation	none set	
Recreation	use raw drinking water	
Industrial		

food processing	use raw drinking water aesthetic guidelines
other industrial uses	none set

Table 1.1.2 Interim Aquatic Life Toxicity Guidelines ** for Chlorophenols.

(calculated in µg/L, at 10°C * , from pH 5.7 to pH 9.2).

chlorophenol congeners	pH 5.7	pH 6.2	pH 6.7	pH 7.2	pH 7.7	pH 8.2	pH 8.7	pH 9.2
2-MCP	3.85	6.35	10.5	17.4	28.7	47.9	79.0	130
3-MCP	3.39	5.60	9.32	15.4	25.3	42.2	69.6	115
4-MCP	1.73	2.85	4.75	7.83	12.9	21.5	35.5	58.5
2,3-DCP	1.11	1.84	3.06	5.05	8.32	13.9	22.9	37.7
2,4-DCP	0.58	0.95	1.58	2.61	4,30	7.16	11.8	19.5
2,5-DCP	0.50	0.82	1.36	2.25	3.70	6.17	10.2	16.8
2,6-DCP	2.00	3.31	5.51	9.09	15.0	25.0	41.2	67.8
3,4-DCP	0.59	0.98	1.63	2.69	4.43	7.39	12.2	20.1
3,5-DCP	0.45	0.74	1.23	2.03	3.35	5.58	9.21	15.2
2,3,4-TCP	0.48	0.79	1.32	2.18	3.59	5.99	9.88	16.3
2,3,5-TCP	0.49	0.81	1.34	2.22	3.65	6.09	10.0	16.6
2,3,6-TCP	1.58	2.61	4.35	7.18	11.8	19.7	32.5	53.6
2,4,5-TCP	0.45	0.74	1.23	2.02	3.34	5.56	9.17	15.1
2,4,6-TCP	1.17	1.94	3.23	5.32	8.77	14.6	24.1	39.7
3,4,5-TCP	0.19	0.31	0.52	0.86	1.41	2.35	3.88	6.40
2,3,4,5-TTCP	0.38	0.62	1.04	1.71	2.83	4.71	7.77	12.8

2,3,4,6-TTCP	1.07	1.76	2.94	4.85	7.99	13.3	22.0	36.2
2,3,5,6-TTCP	0.49	0.80	1.34	2.21	3.64	6.07	10.0	16.5
2,3,4,5,6-PCP	0.16	0.27	0.45	0.74	1.22	2.03	3.35	5.52

* multiply the table values by 2 at 0°C and by 0.5 at 20°C.
** These guidelines are maximum values, rounded to 3 significant figures.

Table 1.1.3 Taste Guidelines for Chlorophenols in Fish Muscle.

Chlorophenol	Guidelines (µg/g) *
2-MCP	10
3-MCP	20
4-MCP	40
2,3-DCP	80
2,4-DCP	0.2
2,5-DCP	20
2,6-DCP	30
2,4,6-TCP	50
2,3,4,5,6-PCP	20

* These are based on the wet weight of muscle tissue and are interim maximum values.

Table 1.3 Relative Toxicity of the Chlorophenols in Several Biological Test Systems:

PCP=1000. Test Systems A-P, [References] and Summary Statistics.

Congeners		A	B	C	D	E	F	G	H	I
		[57]	[144]	[135]	[56]	[381]	[273]	[315]	[229]	[260]
2-MCP		42	40	0.01	262			4	244	17
3-MCP		48		0.03					156	
4-MCP		94	55	0.02	166	4	18		200	
2,3-DCP		146								
2,4-DCP		283	113	0.05	262	17	36	40	112	31
2,5-DCP										
2,6-DCP		81	82						134	
3,4-DCP		275								
3,5-DCP		364								
2,3,4-TCP		339								
2,3,5-TCP		333								
2,3,6-TCP		103							178	
2,4,5-TCP		365	245	0.69	252	114	158	456	156	
2,4,6-TCP		139	270	0.62	113	32	27	77	200	26
3,4,5-TCP		862								
2,3,4,5-TTCP		431								585
2,3,4,6-TTCP			459		2345	313	360	585	422	
2,3,5,6-TTCP		334			1193					
2,3,4,5,6-PCP		1000	1000	1000	1000	1000	1000	1000	1000	1000 1000

Table 1.3 (continued)

Congeners		K	L	M	N	O	P		arith.
		[381]	[651]	[651]	[130]	[130]	[90]	N	mean

2-MCP			81	19				10	72
3-MCP			117	400				5	144
4-MCP		6	66	30				10	64
2,3-DCP								1	146
2,4-DCP		35	147	54	980	204	118	16	155
2,5-DCP			147	213				2	180
2,6-DCP							50	4	87
3,4-DCP								1	275
3,5-DCP								1	364
2,3,4-TCP								1	339
2,3,5-TCP							250	2	292
2,3,6-TCP								2	141
2,4,5-TCP			225	158	2232	688	222	13	406
2,4,6-TCP		43	160	49	633	363	182	16	151
3,4,5-TCP								1	862
2,3,4,5-TTCP					2907	1189		5	1342
2,3,4,6-TTCP		375	450	81	676	543	400	12	548
2,3,5,6-TTCP					610	376		4	628
2,3,4,5,6-PCP		1000	1000	1000	1000	1000	1000	16	1000 1000

Table 2.1.1 The Chlorophenols: Formulae, Names and Molecular Weights.

Abbreviations	Weights	Names	Formulae
P	89.12	Phenol	C_6H_6O
CPs	(n =1 to 5)	Chlorophenols	$C_6H_{(6-n)}Cl_nO$

MCPs	128.56	Monochlorophenols	$C_6H_5Cl\ O$
2-MCP		2-monochlorophenol	
3-MCP		3-monochlorophenol	
4-MCP		4-monochlorophenol	
DCPs	163.00	Dichlorophenols	$C_6H_4Cl_2O$
2,3-DCP		2,3-dichlorophenol	
2,4-DCP		2,4-dichlorophenol	
2,5-DCP		2,5-dichlorophenol	
2,6-DCP		2,6-dichlorophenol	
3,4-DCP		3,4-dichlorophenol	
3,5-DCP		3,5-dichlorophenol	
TCPs	197.45	Trichlorophenols	$C_6H_3Cl_3O$
2,3,4-TCP		2,3,4-trichlorophenol	
2,3,5-TCP		2,3,5-trichlorophenol	
2,3,6-TCP		2,3,6-trichlorophenol	
2,4,5-TCP		2,4,5-trichlorophenol	
2,4,6-TCP		2,4,6-trichlorophenol	
3,4,5-TCP		3,4,5-trichlorophenol	
TTCPs	231.98	Tetrachlorophenols	$C_6H_2Cl_4O$
2,3,4,5-TTCP		2,3,4,5-tetrachlorophenol	
2,3,4,6-TTCP		2,3,4,6-tetrachlorophenol	
2,3,5,6-TTCP		2,3,5,6-tetrachlorophenol	
2,3,4,5,6-PCP	266.34	2,3,4,5,6-pentachlorophenol	C_6HCl_5O
2,3,4,5,6-NaPCP	288.33	2,3,4,5,6-sodium-pentachlorophenate	NaC_6Cl_5O
2,3,4,5,6-KPCP	304.43	2,3,4,5,6-potassium-pentachlorophenate	KC_6Cl_5O

Table 2.1.2 List of Alternate names for the Chlorophenols, as found in the literature (655, and other references).

2-MCP

2-chlorophenol
6-chlorophenol *
6-MCP *
6-monochlorophenol *
O-chlorophenol
O-CP
O-MCP
O-monochlorophenol
orthochlorophenol
orthomonochlorophenol
phenol, 2-chloro-
phenol, 2-monochloro
phenol, 6-chloro- *
phenol, 6-monochloro- *
phenol, O-chloro-
phenol, O-monochloro

3-MCP

3-chlorophenol
5-chlorophenol *
5-MCP *
5-monochlorophenol *
M-chlorophenol
M-CP
M-MCP
M-monochlorophenol
metachlorophenol

metamonochlorophenol
phenol, 3-chloro-
phenol, 3-monochloro
phenol, 5-chloro- *
phenol, 5-monochloro- *
phenol, M-chloro-
phenol, M-monochloro

4-MCP

4-chlorophenol
P-chlorophenol
P-CP
P-MCP
P-monochlorophenol
parachlorophenol
paramonochlorophenol
phenol, 4-chloro-
phenol, 4-monochloro-
phenol, P-chloro-
phenol, P-monochloro-

2,3-DCP

5, 6 -DCP *
5, 6 -dichlorophenol *
phenol, 2, 3-dichloro-
phenol, 5, 6-dichloro- *

2,4-DCP

4,6 -DCP *
4,6 -dichlorophenol *
phenol, 2,4-dichloro-
phenol, 4,6-dichloro- *

2,5-DCP

3,6 -DCP *
3,6 -dichlorophenol *
phenol, 2,5-dichloro
phenol, 3,6-dichloro *

2,6-DCP

phenol, 2,6-dichloro-

3,4-DCP

4,5 -DCP *
4,5 -dichlorophenol *
phenol, 3,4-dichloro-
phenol, 4,5-dichloro- *

3,5-DCP

phenol, 3,5-dichloro-

2,3,4-TCP

4,5,6 -TCP *
4,5,6 -trichlorophenol *
phenol, 2,3,4-trichloro-
phenol, 4,5,6-trichloro- *

2,3,5-TCP

3,5,6 -TCP *
3,5,6 -trichlorophenol *
phenol, 2,3,5-trichloro-
phenol, 3,5,6-trichloro- *

2,3,6-TCP

2,5,6 -TCP *
2,5,6 -trichlorophenol *

phenol, 2,3,6-trichloro-
phenol, 2,5,6-trichloro- *

2,4,5-TCP

3,4,6-TCP *
3,4,6-trichlorophenol *
Collunosol
Dowicide B
Nurelle
Omal
phenol, 2,4,5-trichloro-
phenol, 3,4,6-trichloro- *
Preventol I

2,4,6-TCP

2,4,6-T
Dowicide 25
NCI-CO2904
Omal
Phenachlor
phenol, 2,4,6-trichloro-
Trichlorofenol

3,4,5-TCP

phenol, 3,4,5-trichloro-

2,3,4,5-TTCP

2,3,4,5-TCP
2,3,4,5-TeCP
3,4,5,6-TCP *
3,4,5,6-TeCP *
3,4,5,6-tetrachlorophenol *
3,4,5,6-TTCP *

phenol, 2,3,4,5-tetrachloro-
phenol, 3,4,5,6-tetrachloro- *

2,3,4,6-TTCP

2,3,4,5-TeCP *
2,3,4,6-TCP
2,3,4,6-TeCP
2,4,5,6-TCP *
2,4,5,6-tetrachlorophenol *
2,4,5,6-TTCP *
Dowicide 6
phenol, 2,3,4,6-tetrachloro-
phenol, 2,4,5,6-tetrachloro- *
TCP

2,3,5,6-TTCP

2,3,5,6-TCP
phenol, 2,3,5,6-tetrachloro-

Na-PCP (and similar names for the rarer K salts)

penta-ate
pentachlorophenate sodium
pentachlorophenol sodium salt
pentachlorophenoxy sodium
pentaphenate
phenol pentachloro-sodium derivative monohydrate
sodium PCP
sodium pentachlorophenate
sodium pentachlorophenoxide

2,3,4,5,6-PCP (or just PCP since there is only 1)

A13-00134
Caswell No. 641
Chem-tol

Chlon
Chlorophen
Crystogilal
Dow Pentachlorophenol D P -2 Antimicrobial
Dowicide 7
Dowicide EC-7
Dowicide-7
Dowicide-G
Dura Treet II
Duratox
EP-30
Forpen-50 Wood Preservative
Fungifen
Glazd Penta
Grundier Arbezol
Lauxtox
Lauxtox A
Liropen
NCI-C54933
NCI-C55378
NCI-C56655
Ontrack WE Herbicide
Ortho Triox Liquid Vegetation Killer
Osmose Wood Preserving Compound
PCP chlorophen
Penchloral
Penchlorol
Penta
Penta Concentrate
Penta Ready
Penta WR
Penta-kil
Pentachlor-phenol
Pentachlorofenol
Pentachlorofenola
Pentachlorofenolo
Pentachlorophenate

pentachlorophenol
Pentacon
Pentanol
Pentasol
Penwar
Peratox
Permacide
Permagard
Permasan
Permatox
Permite
phenol, 2,3,4,5,6-pentachloro-
Priltox
Santobrite
Santophen
Santophen-20
Sinituho
Term-i-trol
Thompson's Wood Fix
@ Vulcan Block Penta
@ Vulcan GlazD Penta Tech. Grade Pentachlorophenol
Watershed Wood Preservative
Weedone
@ Wood-Treat Technical Penta (PPI brand)
Woodtreat

* illegitimate numbering of the chlorine positions
@ the only currently (1996) registered products in Canada.

Table 2.3 Characteristics of the Chlorophenols.

Congeners	CAS	RTECS	MISA	in	Solubility in Water
	Reg. #	Reg. #	Reg. #	use	

2-MCP	95-57-8	SK2625000		yes	28 g/L @ 20C
3-MCP	108-43-0	SK2450000		yes	26 g/L @ 20C 27 g/L @ 15C @ pH 5.1
4-MCP	106-48-9	SK2800000		yes	27 g/L @ 20C
2,3-DCP	576-24-9			no	
2,4-DCP	120-83-2	SK8575000		yes	4.5-4.6 g/L @ 20C 4.5 g/L @ 25C 6.2 g/L @ 25C @ pH 5.1
2,5-DCP	583-78-8			no	
2,6-DCP	87-65-0	SK8750000	553.00	no	
3,4-DCP	95-77-2			no	
3,5-DCP	591-35-5			no	
2,3,4-TCP	15950-66-0			no	22.1 mg/L
2,3,5-TCP	933-78-8		1427.50	no	22 mg/L
2,3,6-TCP	933-75-5			no	
2,4,5-TCP	95-95-4	SN1400000		yes	948 mg/L @ 25C @ pH 5.1 <2000 mg/L @ 25C
2,4,6-TCP	88-06-2	SN1575000		yes	434 mg/L @ 25C @ pH 5.1 800-900 mg/L @ 25C 2430 mg/L @ 96C

3,4,5-TCP	609-19-8		1432.00	no	
2,3,4,5-TTCP	4901-15-3	SM9200000	1358.00	no	
2,3,4,6-TTCP	58-90-2	SM9275000		yes	100 mg/L @ 25C 183 mg/L @ 25C @ pH 5.1
2,3,5,6-TTCP	935-95-5	SM9450000		no	
2,3,4,5,6-PCP	87-86-5	SM6300000		yes	5 mg/L @ 0C 9.6 mg/L @ 25C @ pH 5.1 12 mg/L @ 15C 14 mg/L @ 20C 20 mg/L @ 30C 35 mg/L @ 50C
Na-PCP	131-52-2			yes	4 g/L @ pH 8.0

Table 2.3 (continued)

Congeners	pKa	boiling point	melting point	log10 Ko/w	
	range	range, C	range, C	range	
2-MCP	8.3-8.6	175	9	2.12-2.17	1.26 @ 20 C 1.26 @ 25 C
3-MCP	8.8-9.1	214	33-34	2.48-2.50	1.25 @ 25 C
4-MCP	9.1-9.4	217-219	42-44	2.35-2.44	1.27 @ 30 C

2,3-DCP	6.4-7.8	206	57-58	3.15-3.19	
2,4-DCP	7.5-8.1	210	45	2.75-3.30	
2,5-DCP	6.4-7.5	211	58-59	3.20-3.24	
2,6-DCP	6.7-7.8	219	68	2.57-2.86	
3,4-DCP	7.4-8.7	253-254	65-68	3.13-3.44	
3,5-DCP	6.9-8.3	233	68	2.57-3.56	
2,3,4-TCP	6.5-7.7	sublimes	77-84	3.49-4.07	
2,3,5-TCP	6.8-7.4	248-255	57-62	3.84-4.56	
2,3,6-TCP	6.0-7.1	246 (272)	58	3.88	
2,4,5-TCP	7.0-7.7	sublimes	67-70	3.72-4.10	
2,4,6-TCP	6.0-7.4	243-249	68-70	3.62-4.05	1.49 @ 75 C
3,4,5-TCP	7.7-7.8	271-277	101	4.01-4.39	
2,3,4,5-TTCP	6.2-7.0	sublimes	116-117 95-98	4.21-5.16	
2,3,4,6-TTCP	5.3-6.6	150	69-70	4.10-4.81	1.60 @ 60 C
2,3,5,6-TTCP	5.2-5.5	188	114-116	3.88-4.92	

2,3,4,5,6-PCP	4.7-4.9	293-311	174-191	5.01-5.86	1.98 @ 22 C

Table 2.4 QSAR Analyses of the Relative Toxicity of the Chlorophenols.

Using data on ***Daphnia magna***.

Chlorophenol congeners	**24-h IC_{50} (mg/L)**		**Ratio of CP/PCP**
	range	mean	
2-MCP	16.6-19.3	17.95	23.62
3-MCP	13.8-17.75	15.78	20.76
4-MCP	5.79-10.34	8.07	10.62
2,3-DCP	4.09-6.30	5.19	6.83
2,4-DCP	2.48-2.89	2.68	3.53
2,5-DCP	-	**4.50 ***	**5.92 ***
2,6-DCP	8.69-10.08	9.38	12.34
3,4-DCP	2.55-2.98	2.77	3.64
3,5-DCP	1.85-2.33	2.09	2.75
2,3,4-TCP	2.00-2.48	2.24	2.95
2,3,5-TCP	2.06-2.50	2.28	3.00
2,3,6-TCP	6.25-8.52	7.38	9.71
2,4,5-TCP	1.88-2.29	2.08	2.74
2,4,6-TCP	4.93-6.01	5.47	7.20
3,4,5-TCP	0.82-0.93	0.88	1.16
2,3,4,5-TTCP	1.52-1.98	1.76	2.32
2,3,4,6-TTCP	-	**2.70 ***	**3.55 ***
2,3,5,6-TTCP	1.87-2.66	2.27	2.99

2,3,4,5,6-PCP	0.62-0.89	0.76	1.00

This is an experiment by Devillers and Chambon in 1986 (57). The ***Daphnia magna*** were all >72 hours old. The water hardness was 200 mg/L as CaCO3,
dissolved oxygen >2.27 mg/L, pH 7.8 to 8.2 and the temperature 20C.
* 2,5-TCP and 2,3,4,6-TTCP were not tested; these two were estimated using QSAR arguments.

Table 3.2 Some Commercially Registered Products Containing Chlorophenols.

use	type	PRODUCT NAME	% OF EACH CHLOROPHENOL
C	SO	Dowicide 2 Antimicrobial	2,4,5-DCP (95.0)
C	SN	Biocide 207	Na-TCP (17.3), NaPCP (14.1)
C	SN	Biocide 209	Na-TCP 927.8, Na-PCP (10.0)
C	SO	Dowicide 8 Antimicrobial	Na-TCP (85.0)
C	SO	49-167 Tetrachlorophenol	TTCP (94.0)
C	SN	Permatox 180	K-TTCP (28.3)
C	SN	Alchem 4135 Fungicide Sap Stain Inhibitor	Na-TTCP (24.0) also TBTT (2.3)
C	SN	ML-21 Liquid for Control of Bacteria and Fungi	Na-PCP (14.1), Na-TTCP (17.3)
C	SN	Permatox 100 Liquid Fungicide Concentrate	Na-TTCP (22.8)
C	SN	Chapco SSC Concentrate Liquid Fungicide for Lumber and Timber	Na-TTCP (22.8)
C	SN	Diatox	Na-TTCP (19.4), Na-PCP (4.8)
C	SN	VW and R Guardsman Stain Control Woodbrite 24	Na-TTCP (16.3), Na-PCP (7.7) also Borax (2.0)

C	SU	18-600 Woodsheath Cherry Brown 10.1 IG	Na-TTCP (6.9)
C	SU	18-528 Woodsheath Seabrite - 10.0 IC	Na-TTCP (14.2)
C	SN	18-706 Tetra Concentrate 18.0 IG	Na-TTCP (21.9)
C	SU	18-708 Woodsheath Clear 10.1 IC	Na-TTCP (13.6)
D	SN	Behr Wood Preservative NO. 91	PCP (5.0)
D	SN	Moorewood Penta Wood Preservative Clear 456-00	PCP (4.8)
D	SN	Bulldog Grip Wood Preservative Clear	PCP (4.8)
D	SN	Mastercraft Clear Wood Preservative and Sealer	PCP (2.9)
C	SN	Penta-Mix Wood Preservative	PCP (5.0)
C	SN	Penta Preservative Concentrate 1 to 10 Wood Preservative in Soil Poison	PCP (36.3), TTCP (5.0)
C	SN	Chapman Penta WR Concentrate 1-5	PCP (22.2), TTCP
use	**type**	**PRODUCT NAME**	**% OF EACH CHLOROPHENOL**
C	PA	Pol-Nu Pak Ground-Line Pole Treatment Bandage	PCP (8.8), TTCP (1.2)
C	PA	Pol-Nu Penta Preservative Grease for Ground-Line Treatment	PCP (8.8), TTCP (1.2)
C	PA	Timpreg Pak Pol-Nu Type Preservative Grease	PCP (8.8), TTCP (1.2) also Creosote (15) and NaF (15)
C	PA	Timpreg Pol-Nu Type Preservative Grease	PCP (8.8), TTCP (1.2), also Creosote (15.0) and NaF (15.0)
C	PA	Timpreg B Pol-Nu Type Wood Grease	PCP (8.8), TTCP (1.2) also borax (15.5)
C	PA	Timpreg B (Special) Wood	PCP (8.8), TTCP (1.2) also

		Preservative Grease	Creosote (15.5) and borax (15.5)
C	EC	PQ-10 Liquid Fungicide for Lumber and Timber	PCP (17.6), TTCP (2.4) also Copper-8-quinolinolate
C	SN	Goodyear Penta Wood Preserver	PCP (5.0)
C	SN	Goodyear Wood Preserver Black	PCP (5.0)
C	SN	Cuprinol Penta NO.2	PCP (4.8)
C	SN	Cuprinol Penta NO.2 (WR)	PCP (4.8)
C	SN	Kleene-Phene Disinfectant	PCP (0.5), TTCP (0.06), also O-benzyl-p-chlorophenol
C	SN	CC Pentol Wood Preservative for Field Cuts	PCP (5.0)
D	SN	Super Solignum 10-10 Clear Wood Preservative	PCP (4.8)
D	SN	Super Solignum Wood Preservative	
		- Stain 10-22, Cedar	PCP (3.1)
		- Stain 10-21, Redwood	PCP (3.1)
		- Stain 10-62, Brunswick Green	PCP (3.1)
		- Stain 10-23, Mahogany	PCP (3.1)
		- Stain 10-14, Walnut	PCP (3.1)
		- Stain 10-16, Teakwood	PCP (3.1)
		- Stain 10-15, Black	PCP (3.1)
		- Stain 10-200, Bungalo White	PCP (3.1)
		- Stain 10-68, Straw	PCP (3.1)
		- Stain 10-66, Drift Wood	PCP (3.1)
		- Stain 10-63, Dark Brown	PCP (3.1)
use	**type**	**PRODUCT NAME**	**% OF EACH CHLOROPHENOL**
C	SN	Wood Preservative Clear	PCP (4.9)

D	SN	Protox Clear (Clair)	PCP (5.0)
D	SN	Woodsol Paintable Penta Clear	PCP (4.8)
D	SN	Wood Preservative Clear	PCP (5.0)
C	GR	Domtar Pentachlorophenol - Industrial Wood Preservative	PCP (96.0)
C	GR	Dowicide EC-7 Antimicrobial	PCP (88.0), TTCP (12.0)
D	SN	Later's Pentachlorophenol SN Ready-to-Use Wood Preservative	PCP (5.0)
C	SN	Later's Pentachlorophenol Wood Preservative 1-10 Liquid Concentrate	PCP (40.0)
S	SN	Laurentide Paint Wood Preservative Clear G-14	PCP (4.8)
D	SN	Rez Penta Wood Preservative Clear	PCP (5.0)
D	SN	Rez Penta Wood Preservative Green	PCP (5.0)
D	SN	Tim-Ber-Lox Fungicide Wood Preservative Green 4410	PCP (4.8)
D	SN	Tim-Ber-Lox Fungicidal Wood Preservative Clear 4413	PCP (4.8)
D	SN	Pentox Penta Green Wood Preservative	PCP (5.0)
D	SN	Pentox Wood Preservative Brown	PCP (3.9)
C	SN	Pentox 1+10 Penta	PCP (40.0)
C	SN	24-12 Wood Preservative Solution	PCP (5.0)
C	SU	Osmose Osmoplastic Wood Preserving Compound	PCP (2.2), also NaF (43.7) Creosote (20.0), Dinitrophenol (2.0) and K-dichromate (3.1)
C	PA	Osmoplastic-B Wood Preservative Compound	PCP (10.0), also Creosote (15.0) and Borax (15.0)

C	SN	Penta Preservative 1-10	PCP 36.3), TTCP (5.0)
D	SN	Penta-Phenol Clear Paintable Wood Preservative and Primer-Sealer	PCP (4.8)
C	GR	RCL49-162 Pentachlorophenol for Manufacturing Purposes Only	PCP (96.0)
use	**type**	**PRODUCT NAME**	**% OF EACH CHLOROPHENOL**
D	SN	Woodlife Liquid Water Repellent Wood Preservative	PCP (4.8)
D	SN	Roz-Tox Clear Wood Preservative and Sealer	PCP (2.9)
C	PP	Sanitized Van Interior Aerosol	PCP (0.1)
C	PP	United Van Lines Sanitized Van Interior Spray	PCP (0.1)
C	SN	Stangard Penta Wood Preservative Concentrate 1-10	PCP (41.0)
C	SN	Stangard Paintable Penta Clear Wood Preservative	PCP (5.0)
C	SN	Stangard Penta WR Wood Preservative Concentrate 1-4	PCP (21.0)
C	SN	Stangard Penta WR Water Repellent Wood Preservative	PCP (5.0)
C	SN	Stangard Penta Green Wood Preservative	PCP (5.0)
D	SN	Stangard Paintable Penta Clear Wood Preservative	PCP (5.0)
D	SN	Stangard Penta WR Water Repellent Wood Preservative	PCP (5.0)
D	SN	Stangard Penta Green Wood Preservative	PCP (5.0)
C	SU	18-116R Wood Sealer T-678 Gold	PCP (4.4)

C	PA	Stangard Penta Grease 10 Groundline Wood Preservative	PCP (10.0)
C	SN	Horntox Clear Wood Preservative	PCP (0.06), also zinc (2.0)
C	SN	Horntox Green Wood Preservative	PCP (0.06), also copper (2.0)
C	SN	Nevarot Water Repellent Wood Preservative	PCP (4.8)
C	SN	Pole topper Fluid Wood Preservative	PCP (8.8), TTCP (1.2)
C	PA	Osmoband Wood Preservative Bandage	PCP (8.8), TTCP (1.2), also Creosote (15.0) and NaF (20.0)
C	SN	PCP 1 to 10 Concentrate Wood Preservative	PCP (35.9), TTCP (4.2)
use	type	PRODUCT NAME	% OF EACH CHLOROPHENOL
C	SO	Uniroyal 17039 Pentachlorophenol	PCP (96.0)
C	SN	Guardsman Penta Preservative 1-10	PCP (43.0)
C	SN	Guardsman Penta Preservative	PCP (4.3)
C	EC	Mystox LSE Bacteriostatic and Fungistatic Additive	PCP (0.5), also fatty acid esters of PCP (25.0)
D	SN	Woodlife Liquid Water Repellent Wood Preservative	PCP (5.0)
C	SN	Woodlife Liquid Water Repellent Wood Preservative	PCP (5.0)
C	SN	Woodlife 3:1 Concentrate Wood Preservative	PCP (16.3)
C	SN	Guardsman Penta. Preservative 1-10	PCP (43.0)
C	SN	Guardsman Penta Preservative	PCP (4.3)
C	EC	Mystox LSE Bacteriostatic and Fungistatic Additive	PCP (0.5), also fatty acids esters of PCP (25.0)

C	SU	18-116R Wood Sealen T-678 Gold	PCP (4.4)
D	SN	Woodlife Liquid Water Repellent Wood Preservative	PCP (5.0)
C	SN	Woodlife Liquid Water Repellent Wood Preservative	PCP (5.0)
C	SN	Woodlife 3:1 Concentrate Wood Preservative	PCP (16.3)
C	SN	Betz Slimicide A-9	Na-PCP (27.6), Na-TCP (9.1)
C	SG	Chapman Permatox 10-S	Na-PCP (36.0), also borax (57.0)
C	GR	Napclor-S Antimicrobial	Na-PCP (90.0)
D	SN	Dearcide 712 Liquid Cooling Water Microbistat	N-PCP (32.0), Na-TCP (8.0)
S	SG	Dowicide G-ST Antimicrobial	Na-PCP (90.0), also NaOH (1.5)
C	SN	Chem-Aqua 400	Na-PCP (5.0)
P	SN	Ready-To-Use Moss Stop	Na-PCP (3.6)
C	PE	Slimicide Formula Y-100 Pellets	Na-PCP (90.0)
C	SN	Sanitized Brand SPI	Na-PCP (10.0)
C	SN	Sanfax Pinfax Liquid Disinfectant	Na-PCP (0.6), also Na-O-benzyl-P-chlorophenate (1.1)
C	IF	Patox Pole Treating Wrap Type 1	Na-PCP (8.5), Na-TTCP (1.0) also Creosote (11.0), NaF (37.1) and K-borate (12.5)

Abbreviations for use and type codes for Table 3.2.
C - commercial, P - domestic, EC - Emulsifiable concentrate, GR - granular, IF - impregnated fabric, PA - paste, PE - pellet, PP - pressurized, SO - solid, SG - soluble granule, SN - solution, SU - suspension, TBTT - tributyltin, PCP - pentachlorophenol, TCP - trichlorophenols, TTCP - tetrachlorophenols, Na - sodium, K - potassium, NaF - sodium fluoride, NaOH - sodium hydroxide.

Table 3.3.1 Analyses of 2,4-DCP and 2,4-D Acid Samples for Chlorinated

Phenolic Impurities.

Contaminant	% of 2,4-DCP Sample *		
	maximum	minimum	mean (n=10)
2,4-DCP	98.30	83.90	92.24
2,6-DCP	9.18	1.40	4.48
2,4,6-TCP	3.23	0.53	4.24
2-MCP	3.52	0.10	1.09
4-MCP	1.19	0.05	0.46
2,4-DC-6-methyl P	0.34	ND	0.07
3-MCP	ND	ND	ND
Contaminant	**% of 2,4-D Acid Sample ***		
	maximum	minimum	mean n=13
2,4-DCP	1.45	0.004	0.19
2,6-DCP	0.048	0.001	0.01
2,4,6-DCP	0.14	0.001	0.02
2-MCP	0.004	0.0004	0.001
4-MCP	0.005	0.0004	0.001
2,4-DC-6-methyl P	ND	ND	ND
3-MCP	ND	ND	ND

* ND = non detectable

Table 3.3.2 The Concentrations of Chlorophenols found in Commercial Products

REFERENCE	GRADE/FORMULATION	CONC. (g/L)	CHLOROPHENOL
5	C14 labelled, pure	980	PCP
5	Reagent	970	PCP
6	C14 labelled, pure	950	PCP
38	Dowicide EC-7	904	PCP
38	Dowicide EC-7	104	TTCPs
38	Dowicide EC-7	<1	TCPs
45	Fungicide	<990	PCP
49	Commercial	750	PCP
79	Technical, Dowicide EC-7	880	PCP
98	Pure	990	PCP
98, 99	Technical	860	PCP
102	Dowicide EC-7, lot	49	2,3,4,6-TTCP
102	12109-M	9	2,3,4,5-TTCP
111	Technical	960	PCP
111	Liquid	900	Na-PCP
113	Dowicide EC-7	937	PCP
113	Dowicide EC-7	49	2,3,4,6-TTCP
113	Dowicide EC-7	9	2,3,4,5-TTCP
117	Dowicide G-ST	790	Na-PCP
118	Dowicide G	790	Na-PCP
127	Purified	990	PCP
127	Dowicide EC-7, Technical	910	PCP
140	Santobrite	>900	Na-PCP
147	Purified	990+	PCP
147	Ultrapurified	990+	PCP
206	Dowicide EC-7	937	PCP

208 *	Dowicide G-ST, Lot 90921d	80	2,3,4,6-TTCP
236	Dowicide EC-7	880	PCP
236	Technical	960	PCP
236	Technical	860	PCP
236	Technical	740	2,3,4,6-TTCP
236	Liquid	900	Na-PCP

* this is an antimicrobial.

Table 3.3.3 Contaminants Present in Commercial PCP Products

REFERENCE	GRADE/FORMULATION	mg/kg *	CONTAMINANT
38	Dowicide EC-7	400	hexa-CB
		3.4	hexa-CDBF
		1.8	hepta-CDBF
		1.0	octa-CDBF
102	Dowicide EC-7, lot 12109-M	270	hexa-CB
113	Dowicide EC-7	270	hexa-CB
127	Technical mixture	28	hexa-CB
		600	hepta-CPP
		14000	octa-CPP
		7500	nona-CPP
		14.5	octa-CDBF
	Purified (99%)	2.4	hepta-CPP
		150	octa-CPP
		250	nona-CPP

	Dowicide EC-7	75	hexa-CPP
		1.9	hepta-CPP
		2.8	octa-CPP
		7	nona-CPP
		0.37	hexa-CDPO
		80	hepta-CDPO
		1.6	octa-CDPO
		0.37	nona-CDPO
147	Purified	100	hepta-CPP
		1700	octa-CPP
	Ultrapurified	45	hepa-CPP
		13	octa-CPP
		414	nona-CPP

* 1000 mg/kg = 0.1%.
CB - chlorobenzene, CPP - chlorophenoxyphenol, CDBF - chlorodibenzofuran,
CDPO - chlorodiphenyloxide

Table 3.3.4 Dioxins Present as Impurities in Commercial PCP Products.

REFERENCE	GRADE/FORMULATION	mg/kg *	DIOXIN SPECIES
35	Commercial PCP	8	HCDDs
		520	HPCDDs
		1380	OCDD
36	Technical PCP	42	HCDDs
		24	HPCDDs

		11	OCDD
37	Commercial PCP	0.17-39.0	HCDDs
38	Dowicide EC-7	1	HCDDs
		6.5	HPCDDs
		15	OCDD
		<0.05	2,3,7,8-TTCDD
102	Dowicide EC-7, lot	2	HCDDs
	#12109-M	40	OCDD
113	Dowicide EC-7	2	HCDDs
		40	OCDD
127	Dowicide EC-7	0.11	HPCDDs
		1.0	OCDD
127	Technical (mixture)	115	HPCDs
		2350	OCDD
147	Purified	25	HCDDs

* 1000 mg/kg = 0.1%.
HPCDD (2 isomers) heptachlorinated, OCDD (1 isomer) octachlorinated, TTCDD (22 isomers) tetrachlorinated, HCDD (10 isomers) hexachlorinated dibenzo-p-dioxins.

Table 3.3.5 Analyses of polychlorinated phenols for Dioxins and Furans-1977.

Contaminant or compound	Concentration mg/L or %		
	Tech M	Comm-D(7)	Comm-D(EC-7)
PCP	84.6%	88.4%	89.8%
TTCPs	3.0%	4.4%	10.1%

TCPs	-	<0.1%	<0.1%
Chlorinated phenoxyphenols	-	<6.2%	-
octa-p-dioxins	1380	2500	15.0
hepta-p-dioxins	520	125	6.5
hexa-p-dioxins	8	4	1.0
octa-furan	260	80	<1
hepta-furan	400	80	1.8
hexa-furan	90	30	<1
penta-furan	40	-	-
tetra-furan	<4	-	-

From Reference 8.
Tech-M. = Monsanto, Technical PCP. No longer produced.
Comm-D(7). = Dow, Commercial Dowicide 7, 9522A.
Comm-D(EC-7). = Dow, Commercial Dowicide EC-7.

Table 3.5.1 Chlorophenol Levels Detected in Fraser River Water.

CHLOROPHENOL	# OF SITES	RANGE (ng/L)
2,3,4-TCP	1	2.4
3,4,5-TCP	1	0.46
2,3,5,6-TCP	3	0.12-0.75
2,4-DCP	7	2.5-7.5
2,4,6-TCP	8	13-22
2,3,5-TCP	8	3.8-6.6
2,3,4,6-TTCP	8	2.3-133.0
PCP	8	2.0-56.0

From References 269 and 720.

Table 3.5.2 PCP, TTCP and TCP levels in Water and Sediment at SW BC Sites in 1978.

SITES	SEDIMENT µg/kg			WATER µg/LRATIO:		RATIO: SEDIMENT/WATER	
	PCP	TTCP	TCP	PCP	TTCP	PCP	TTCP
MCI *	ND	ND	ND	ND	ND		
F2	35.0	28.0	ND	0.28	0.10	125	280
F3	10.8	27.4	2.3	0.25	1.0	43	27
F4	18.1	21.9	ND	<0.05	0.30	>362	73
F5	5.0	10.0	ND	<0.05	0.20	>100	50
F mean	17.2	21.8		<0.16	0.15	>157	107.5
M1	34.7	39.8	ND	0.75	1.3	46	31
M6	52.8	98.7	52.1	2.4	5.2	22	19
M7	106.6	272.1	91.0	<0.01	0.06	>10660	4535
M8	16.0	19.5	ND	<0.05	0.09	>320	2167
M9	42.0	65.4	ND	<0.05	0.06	>840	1090
M10	13.1	22.8	ND	3.1	3.3	4.2	6.9
M11	187.9	157.3	37.3	7.3	0.22	0.26	715
M mean	64.7	96.5		<0.20	<1.7	>1699	1223

From Reference 87. ND - non detectable, M - marine, F - freshwater.

MCI * Control, Roberts Bank north of the Coal Port Causeway
F2 Fraser River at Coquitlam, D/S of Pitt River (Crown Zellerbach)
F3 Fraser River at Coquitlam, D/S of Pitt River (Domtar)

F4 Fraser River, Burnaby side, opposite east end of Lulu Island
(Canadian White Pine)
F5 Fraser River Estuary, D/S Swing bridge, SE corner of Lulu Island
(Laters Chemical)
M1 Burrard Inlet, west of Lynn Creek on North Shore
(Seaboard Terminals)
M6 Squamish Estuary, Channel east of Main river channel (Empire Mills)
M7 Victoria Harbour at outfall U/S Johnson Street Bridge (BCFP)
M8 Cowichan Estuary, Channel near Outfall (Doman's)
M9 Nanaimo Harbour, near outfalls and deep sea docks
(Domans and CIPA Lumber)
M10 Nanaimo Estuary, booming grounds near outfall
(Mayo Forest Products)
M11 Port Alberni Harbour, near outfalls
(M & B, Alberni Pacific and Somass Division)

Table 3.5.3 PCP and TTCP Contamination of Canadian Foods.

FOOD	SAMPLES	PCP (µg/kg, µg/L)	TTCPs (µg/kg, µg/L)
Carrots	5	trace, 4, 8, 13	2,5
Turnips	2	3, trace	trace
Raw Milk	6	1, 1, 2, 2, 2, 5	trace
Cabbage	1	1	-
Beets	1	4	1
Potato (peels)	2	157, 12500	150,2160
Potato (pulp)	2	5, 6	1, 14
Potato (whole)	61	279, 2710 a mean of 8.3 for 44 samples between 1 and 58,	472 a mean of 14.3 for 9 samples between 1 and 45,

		trace (14 samples)	trace (41 samples),

From Reference 8. Those samples not specified in the table had non-detectable values.

Table 3.5.4 Chlorophenols Detected in Fraser River Biota

(mean µg/kg wet weight of animal).

congeners	Shrimp ***Crangon***				Staghorn sculpin			
Site No.	1	1	1	4	1	1	4	6
Sample No.	1	2	3	1	1	2	1	1
individuals	15	15	12	17	1	12	1	12
g wet weight	9.792	6.413	4.605	3.772	13.342	4.793	20.775	4.213
2,6-DCP	-	-	-	-	-	-	-	-
3,5-DCP	-	-	-	-	-	-	-	-
3,4-DCP	-	-	-	-	-	-	0.59	-
2,3,4-TCP	-	-	-	-	-	-	0.22	-
2,3,5-TCP	-	-	-	-	-	-	-	-
2,3,6-TCP	-	-	-	-	-	-	-	-
2,4,5-TCP	-	-	-	-	-	-	-	-
2,4,6-TCP	-	-	-	-	0.46	-	1.4	2.4
3,4,5-TCP	-	-	-	-	-	-	3.1	-
2,3,4,5-TTCP	-	-	-	-	-	-	-	-
2,3,4,6-TTCP	1.2	2.3	2.9	3.7	6.3	18.3	16.6	49
2,3,5,6-	-	-	-	-	-	-	-	-

TTCP								
2,3,4,5,6-PCP	2.2	3.3	6.5	3.2	8.8	21.3	19.6	79

congeners	Smelt		Squawfish		Prickly sculpin			St. Fl.
Site No.	4	4	1	13	6	13	15	15
Sample No.	1	2	1	1	1	1	1	1
individuals	lots	6	1	1	2	3	19	1
g wet weight	13.13	2.273	160.41	159.85	26.834	1.268	5.99	23.255
2,6-DCP	-	-	-	-	-	-	-	53
3,5-DCP	-	-	-	-	-	-	-	-
3,4-DCP	-	-	73.3	69	-	69	-	61
2,3,4-TCP	-	-	0.10	0.29	-	0.29	0.41	1.4
2,3,5-TCP	-	-	0.28	0.10	-	0.10	-	0.36
2,3,6-TCP	-	-	0.21	0.78	-	0.78	0.38	1.8
2,4,5-TCP	-	-	-	-	-	-	-	-
2,4,6-TCP	0.52	-	1.4	1.3	2.4	1.3	2.4	1.4
3,4,5-TCP	-	-	-	-	-	-	-	1.2
2,3,4,5-TTCP	-	-	0.41	-	-	-	-	0.15
2,3,4,6-TTCP	9.1	15.7	11	2.6	49	2.6	5.2	2
2,3,5,6-TTCP	-	-	-	-	-	-	-	-
2,3,4,5,6-PCP	8.8	13.4	14.5	3.9	79	3.9	6.2	2.7

From References 269 and 720.
Table 3.5.4 (continued)

congeners	Starry flounder							
Site No.	1	1	4	6	6	13	13	13
Sample No.	1	2	1	1	2	1	2	3
individuals	1	8	8	4	6	1	2	1
g wet weight	23.147	20.896	7.978	7.291	8.215	5.56	14.31	27.368
2,6-DCP	-	-	-	-	-	-	-	56
3,5-DCP	-	-	-	-	-	-	-	-
3,4-DCP	-	-	-	-	-	-	12	24
2,3,4-TCP	0.20	-	-	-	-	-	1.6	0.22
2,3,5-TCP	-	-	-	-	-	-	0.48	0.28
2,3,6-TCP	-	-	-	-	-	-	1.0	0.33
2,4,5-TCP	0.22	-	-	-	-	-	0.62	0.30
2,4,6-TCP	0.94	0.80	2.3	0.63	0.97	-	2.2	1.5
3,4,5-TCP	-	-	-	-	-	-		0.76
2,3,4,5-TTCP	0.04	-	-	-	-	-	0.67	0.36
2,3,4,6-TTCP	8.6	10.7	13	15	13	4	8.2	5.2
2,3,5,6-TTCP	-	-	-	-	-	-	0.42	0.29
2,3,4,5,6-PCP	15.5	13.1	29.9	22	18	4.1	12	9.2

congeners	Peamouth chub						
Site No.	4	6	6	13	13	13	15
Sample No.	1	1	2	1	2	3	1
individuals	5	1	1	11	8	1	23
g wet	3.447	46.21	57.12	16.822	7.729	24.973	13.701

weight							
2,6-DCP	-	20	-	-	-	-	-
3,5-DCP	-	41	-	-	-	-	-
3,4-DCP	-	241	85	-	-	-	-
2,3,4-TCP	-	49	0.59	0.28	0.80	3.4	1.3
2,3,5-TCP	-	-	0.13	0.17	0.46	1.7	0.22
2,3,6-TCP	-	1.8	3.8	0.21	0.63	0.56	0.95
2,4,5-TCP	-	5.6	0.20	0.22	0.33	0.64	-
2,4,6-TCP	2.9	12	1.3	2.8	1.9	4.5	2.1
3,4,5-TCP	-	0.59	-	-	-	16	-
2,3,4,5-TTCP	-	0.26	-	0.17	0.67	1.5	0.35
2,3,4,6-TTCP	30.8	4.8	8.4	6.9	8.7	8.6	5.2
2,3,5,6-TTCP	-	-	-	0.26	-	-	-
2,3,4,5,6-PCP	40.3	3.5	8.4	9.9	12	6.6	9.9

From References 269 and 720.

Table 3.6.1 Summary of Chlorophenol Data from the Fraser Estuary, 1973-1987.

Dichlorophenols

sample type	n<MDC	n>MDC	mean>MDC	std. dev.	max.
sediment	9	0	-	-	-
water	109	14	0.018	0.039	0.0152

fish-muscle	49	69	67.6	131	736
fish-liver	19	27	44.8	38.2	207
invertebrates	4	0	-	-	-

Trichlorophenols

sample type	n<MDC	n>MDC	mean>MDC	std. dev.	max.
sediment	139	16	7.39	3.8	15.1
water	49	99	0.024	0.021	0.116
fish-muscle	174	88	58.1	193	1442
fish-liver	43	12	19.8	9.55	34.5
invertebrates	10	17	225	468	2000

Tetrachlorophenols

sample type	n<MDC	n>MDC	mean>MDC	std. dev.	max.
sediment	116	132	11.0	14.5	90
water	32	174	0.705	1.66	14.8
fish-muscle	65	292	95.7	269	2522
fish-liver	30	61	67.0	91.9	520
invertebrates	25	27	388	702	3000

Pentachlorophenol

sample type	n<MDC	n>MDC	mean>MDC	std. dev.	max.
sediment	128	129	12.6	19.5	107
water	40	166	0.159	0.326	2.71
fish-muscle	54	311	134	344	3200

fish-liver	24	67	105	167	1030
invertebrates	24	28	392	907	4200

Table 3.6.1 (continued)

Total Chlorophenols

sample type	n<MDC	n>MDC	mean>MDC	std. dev.	max.
sediment	120	137	23.2	29.9	180
water	28	178	0.852	1.94	17.5
fish-muscle	55	315	252	699	6239
fish-liver	24	69	182	246	1550
invertebrates	21	31	816	1830	9200

From Reference 693. Data in µg/L (water) or µg/g (dry for sediments) and (wet for organisms). MDC = minimum detectable concentration; this varies with the sample and the chlorophenol since this is composite data from many sources and analytical techniques.

Table 4.1 Fate of Chlorophenols in Water

Process	MCPs	DCPs	TCPs	TTCPs	PCP
1/2 life in water	<1 to 26 days	>6 days	>9 to 35 days	>3.5 months	<3.5 months if aerobic
1/2 life in sediment				years if organic	years if organic
1/2 life in fish tissue	2 days	2 days	10 days	10 days	10 days
biological	214 in	occurs	2,4,5-	20-221 in fish	10-15000

concentration factor	bluegill sunfish	in marine biota and crops	TCP 170-1900 in fish. 2,4,6-TCP 51-442 in plants. 115 to 12180 in fish and 3000 in inverts.	muscles 40-8590 in fish liver. 1000 in leeches 1250 in ***Cladophora***	in fish, liver high and muscle low, lower still in algae and inverts.

Table 4.1 (continued)

Process	MCPs	DCPs	TCPs	TTCPs	PCP
loss by evaporation or by volatilization	not important	not important	not important	not important	of little importance in shallow water at pH 5, not important over pH 7.
hydrolysis and oxidation	not important	not important	not important	not important	not important
photolysis	unknown in nature, occurs in the laboratory	unlikely to be significant in nature	occurs in nature but significance is not known	unlikely to be of significance in nature	important in clear, shallow, neutral to alkaline waters.
sorption	there is a tendency	may be important	observed in lake and	observed in	important, observed in

	to attach to organic particles	in organic sediments	river sediments	river and lake sediments	acidic and in contamin-ated sites
biological degradation	occurs in the laboratory the rate in nature is not known.	rate depends on water quality and on prior bacterial exposure	reported in water, soil and bacterial cultures, likelier in stagnant waters.	reported in soil and bacterial cultures, slower in aquatic systems.	occurs in culture and in nature, best at higher temperatures and when aerobic.

From Reference 220.

Table 4.1.5.1 Adsorption of Chlorophenols on Bentonite Organic Clays

Chlorophenol congener	pKa	Bentone 24		Bentone 18C	
		pH	% adsorbed *	pH	% adsorbed*
2-MCP	8.48	7.8	77	7.6	15
3-MCP	9.02	7.7	81	7.6	13
4-MCP	9.38	8.0	49	7.7	6
2,4-DCP	7.85	7.7	96	7.5	38
2,5-DCP	7.35	7.9	94	7.5	38
2,6-DCP	6.80	7.8	79	7.4	32
3,4-DCP	8.39	7.9	97	7.6	34
3,5-DCP	7.92	7.9	96	7.6	42
2,4,5-TCP	7.74	7.6	98	7.4	64
2,4,6-TCP	6.42	7.5	92	7.4	55

From Reference 509. * after 45 to 48 hours in the dark at 20C.

Table 4.1.5.2 Partitioning of TCPs and TTCPs in the Lower Rhine River.

congener	Sediment, N = 17, (µg/kg)			Ratio (max) Sed./Water	Water, N=13 (µg/L)		
	Samples	Max	Median		Samples	Max	Median
TCPs							
2,3,4-	3	0.8	0.7	20	1	0.04	-
2,3,5-	17	11.0	2.4	39	5	0.28	-
2,3,6-	0	-	-	-	6	0.36	-
2,4,5-	17	15.0	6.4	47	10	0.32	0.15
2,4,6-	16	3.7	1.9	5	13	0.74	0.13
3,4,5-	14	19.0	1.2	61	7	0.31	0.05
TTCPs							
2,3,4,5-	17	8.9	0.9	445	3	0.02	-
2,3,4,6-	17	4.9	1.7	25	12	0.20	0.07
2,3,5,6-	16	2.8	1.4	35	7	0.08	0.01

From Reference 349. Sediment samples are expressed as dry weight. Only the samples with detectable levels of TCP or TTCP are listed in this table.

Table 4.2.1 Chlorophenol Degradation Studies in Soil and Sludge.

Degradation of 3-MCP and 2,4,6-TCP.

pH of the	chlorophenol species	

sludge	3-MCP	2,4,6-TCP	From Reference 184. The percent degradation of 200 mg/L of 2,4,6- TCP and 3-MCP where degradation is defined as ring opening. It takes longer to achieve the formation of free chloride ion. Degradation is in sewage sludge at 27C after 3 days of aeration, and is a function of pH.
6.1	20		
6.5		100	
7.0	40	100	
8.0	0		
8.5	0	100	
8.8		100	

Degradation of 2-MCP.

pH of the	temperature, C				
sludge	20	25	27	30	From Reference 184. The percent degradation of 200 mg/L of 2-MCP where degradation is defined as ring opening. It takes longer to achieve the formation of free chloride ion. Degradation is in sewage sludge after one days aeration and is a function of pH and temperature.
6.5	33		88		
6.8		74		57	
7.3	27		89		
7.9	20		91		
8.4		22		6	
9.3		3		0	

Degradation of some Chlorophenols in Sewage Sludge.

compound	% loss (184)	days	% loss (185)	mg COD/gm/h
phenol			98.5	80.0
2-MCP	100	4	95.6	25.0
3-MCP	100	3		
4-MCP	100	3	96.0	11.0
2,4-DCP	100	5	98.0	10.5

2,5-DCP	16	4		
2,4,6-TCP	75	3		
Na-PCP	0	4		

From References 184 (% degradation) and 185 (COD production). Maximum %
degradation of 100 mg/L over the specified time period, and the rate of COD production. Degradation is defined as ring opening; it takes longer to achieve free chloride ion formation.
Table 4.2.1 (continued)

Degradation of 2,4-DCP and 2,4,6-TCP in Sludge.

initial conc. in mg/L	pH 7.0, 26C		
	2,4-DCP	2,4,6-TCP	From references 184 and 185 % degradation after 2 days of treatment under the specified conditions of pH, initial concentration. and temperature. Degradation is defined as ring opening, it takes longer to achieve free chloride ion.
50	80	100	
100	75	100	
200	40	60	
400	55	0	

The % degradation of MCPs by pre-acclimated sludge in 6 hours.

mg/L	2 MCP	3 MCP	4 MCP
1	100	100	100
10	97	40	80
100	20	0	16

from Reference 440. 1 mg/L 2 MCP is 100% degraded in 3 hours.

The % degradation of Chlorophenols by pre-acclimated sludge.

mg/L	2,4-DCP 2 days	2,4-DCP 5 days	2,5-DCP 4 days	2,4,6-TCP 3 hours	2,4,6-TCP 10 days	PCP 4 days
50	80	-	-	-	-	-
100	75	100	52	70	-	0
200	40	-	-	-	-	-
300	-	-	-	-	95	-
400	55	-	-	-	-	-

from References 184, 473 and 536.

Aerobic Degradation of an initial concentration of 100 mg/L of Chlorophenols in the Soil (from reference 18).

Chlorophenol Congeners	Sterile Soil		Non-Sterile Soil	
	% breakdown	days	% breakdown	days
2-MCP	67	40	70	0.5-1.0
3-MCP	31	160	70	80-160
4-MCP	5	20	70	1-2
2,4-DCP	31	40	70	7-20

Table 4.2.3.1 Calculated Chlorophenol Bioconcentration Factors.

congener	log Ko/w (table 2.2)	Ref. 378	* Ref. 109	Ref. 612	Ref. 610	Ref. 34	Geo. mean	Arith. mean
phenol	1.47	1.41	4.60	7.70	2.40	8.30	3.83	4.95
2-MCP	2.16	6.90	34.0	25.8	13.7	19.7	14.8	12.0

3-MCP	2.50	15.1	91.5	46.8	26.6	30.1	27.4	29.7
4-MCP	2.38	11.5	64.5	37.9	21.0	25.9	22.1	101.3
2,3-DCP	3.19	74.1	683	157	103	71.3	96.1	82.8
2,4-DCP	3.08	57.5	496	129	82.8	62.1	78.6	98.3
2,5-DCP	3.20	75.9	703	159	105	72.2	97.8	186
2,6-DCP	2.74	26.3	184	71.2	42.6	40.7	42.4	45.2
3,4-DCP	3.25	85.1	813	174	116	76.8	107	113
3,5-DCP	3.52	159	1790	279	196	108	175	186
2,3,4-TCP	3.80	302	4040	455	339	153	291	312
2,3,5-TCP	4.10	603	9670	769	610	222	501	551
2,3,6-TCP	3.88	363	5100	523	396	169	336	363
2,4,5-TCP	3.76	275	3590	424	313	145	270	289
2,4,6-TCP	3.86	347	4810	505	381	165	324	350
3,4,5-TCP	4.25	851	15000	1000	818	277	663	737
2,3,4,5-TTCP	4.95	4270	115000	3400	3220	641	2340	2878
2,3,4,6-TTCP	4.35	1070	20000	1190	994	303	787	889
2,3,5,6-TTCP	4.90	3800	99400	3120	2920	602	2137	2611
2,3,4,5,6-PCP	5.20	7590	238000	5270	5250	876	3683	4747

BC = biological concentration factor. P = Ko/w = octanol/water partition coefficient.
* The high values of the calculated BCF from Ref. 109, calculated for 12C and pH 7.0, are not included in the means which are thus based on four values.
Ref. 387. :log BCF = log P-1.32
Ref. 109. :log BCF = 1.265 log P-1.201
Ref. 612. :log BCF = 0.76 log P-0.23
Ref. 34. :log BCF = 0.542 log P+0.124
Ref. 610. :log BCF = 0.85 log P-0.70

Table 4.2.3.2. PCP Tissue levels and Bioaccumulation Factors Relative to the Sediments.

Site	CMM	CPM	CAL	CAM	LAL	LAM	MAC	PP	PT
mean micrograms per kilogram in the tissues									
C-1							3		
1-M		3			24	3			
2-F			140	40				<1	
3-F			600	14				<1	
4-F			300	74	100	3			3
5-F			3	12	470	5			
6-M	<1				35	3			
7-M	3	7			210	13			
8-M	16				3	3			
9-M	3								
10-M	17				2100	3	<1		
11-	8				640	84			

M									
mean bioaccumulation factors relative to the sediments									
C-1									
1-M		0.08			0.69	0.8			
2-F			4	1.1				0.03	
3-F			56	1.3				0.09	
4-F			17	4.1	5.5	0.18			0.18
5-F			0.6	2.4	94	1.0			
6-M	0.02				0.66	0.06			
7-M	0.03	0.7			2.0	0.12			
8-M	1.0				0.18	0.18			
9-M	0.06								
10-M	1.3				160	0.23	0.08		
11-M	0.04				3.4	0.45			

From Reference 87. See also the footnotes following Table 4.2.3.3.

CMM-crab, ***Cancer magister***, muscle
CPM-crab, ***Cancer productus***, muscle
CAL-prickly sculpin, ***Cottus asper***, liver
CAM-prickly sculpin, ***Cottus asper***, muscle
MAC-clam, ***Macoma balthica***, whole animal
PP-crayfish, ***Pacifactacus lenuisculus***, pincers
PT-crayfish, ***Pacifactacus lenuisculus***, tail
LAL-staghorn sculpin, ***Leptocottus armatus***, liver
LAM-staghorn sculpin, ***Leptocottus armatus***, muscle

Table 4.2.3.3. TTCP Tissue levels and Bioaccumulation Factors Relative to the Sediments.

Site	CMM	CPM	CAL	CAM	LAL	LAM	MAC	PP	PT
mean micrograms per kilogram in the tissues									
C-1							3		
1-M		6			69	6			
2-F			89	100				<1	
3-F			320	80				3	
4-F			96	10	74	3			3
5-F			82	5	480	8			
6-M	8				63	9			
7-M	3	3			470	8			
8-M	20				29	10			
9-M	6.6								
10-M	5				1600	8	12		
11-M	3				430	34			
mean bioaccumulation factors relative to the sediments									
C-1									
1-M		0.15.			1.7	0.15			
2-F			3.2	3.6				0.04	
3-F			12	2.9				0.12	
4-F			4.4	0.5	3.4	0.12			0.12
5-F			8.2	0.5	48	1.31			
6-M	0.08				0.64	0.09			
7-M	0.012	0.012			1.7	0.03			
8-M	1.0				1.5	0.51			
9-M	0.1								
10-M	0.21				70	0.35	0.50		

11-M	0.158				2.7	0.22			

See also the footnotes following Table 4.2.3.2.
Table 4.2.3.2 (continued)

C-1: Control, Roberts Bank north of the coal port causeway.
1-M: Marine, Burrard Inlet west of Lynn Creek on the North Shore at Seaboard Terminals.
2-F: Freshwater, Fraser River at Coquitlam, D/S of the Pitt River at Crown Zellerbach.
3-F: Freshwater, Fraser River at Coquitlam, D/S of the Pitt River at Domtar.
4-F: Freshwater, Fraser River, Burnaby side, opposite the east end of Lulu Island at Canadian White Pine.
5-F: Freshwater, Fraser River Estuary, D/S of the swing bridge at the SE corner of Lulu Island at Laters Chemicals.
6-M: Marine, Squamish Estuary, east of the main river channel at Empire Mills.
7-M: Marine, Victoria Harbour at the outfall U/S of the Johnson St. Bridge at BCFP.
8-M: Marine, Cowichan Estuary, channel near outfall and deepsea docks at Nanaimo.
9-M: Marine, Nanaimo Harbour, near outfalls and deepsea docks by Domans and CIPA Lumber.
10-M: Marine, Nanaimo Estuary, booming grounds near outfall at Mayo Forest Products.
11-M: Marine, Port Alberni Harbour, near outfalls at C M & B., Alberni Pacific and Somass Divisions.
From Reference 87.

Table 4.2.3.4 Bioaccumulation Factors for PCP Relative to Water.

B. C. F.	Species, tissues and conditions	Refs.

	Fish (whole body- mean value is 872)	
2	***Carassius auratus***, goldfish, 0.1 mg/L, pH 10	547
5	***Oncorhynchus mykiss***, rainbow trout, 0.035 µg/L, 115 days	126
5	***Cyprinodon variegatus***, sheepshead minnow, 0.118 mg/L, 151 days	556
10	***Lepomis macrochirus***, bluegill sunfish	121
12	***Carassius auratus***, goldfish, 0.1 mg/L, pH 9.0	547
13	***Cyprinodon variegatus***, sheepshead minnow, 151 days	205
24	***Carassius auratus***, goldfish, 0.1 mg/L, pH 8.0	547
27	***Cyprinodon variegatus***, sheepshead minnow, 0.389 mg/L,151 days	556
30	***Fundulus similis***, killifish, 4 days exposure	205
34	***Cyprinodon variegatus***, sheepshead minnow, 28 days	205
34	***Esox lucius***, pike	274
36	***Lebistes reticulata***, guppy, 3µg/L	536
38	***Mugil cephalus***, striped mullet, 4 days	205
53	***Fundulus similis***, killifish, 57-610 µg/L, 5 days	10
60	***Leuciscus rutilus***, roach	274
67	fish, 0.3 µg/L	536
100	***Salmo trutta***, brown trout	90
120	***Carassius auratus***, goldfish, 0.1 mg/L, pH 6.7	547
131	***Carassius auratus***, goldfish, 0.1 mg/L, pH	547

	5.5	
132	fish	582
132	***Gambusia affinis***, mosquito fish	12
165	***Carassius auratus***, goldfish, 0.1 mg/L, 12 hours	579
200	***Oncorhynchus mykiss***, rainbow trout, 0.035 µg/L, 115 days	126
240	***Oncorhynchus mykiss***, rainbow trout, 0.66 µg/L, 115 days	126
250	***Oncorhynchus mykiss***, rainbow trout	126
296	***Gambusia affinis***, mosquito fish	12
300	flounder	272
380	***Platichthys stellatus***, starry flounder	269
480	***Carassius auratus***, goldfish	273
500	***Poecilia reticulata***, guppy, pH 6	144
580	***Carassius auratus***, goldfish, 0.2 mg/L	204
580	***Carassius auratus***, goldfish, 0.2 mg/L	473
650	***Fundulus similis***, killifish	272

Table 4.2.3.4 (continued)

B. C. F.	Species, tissues and conditions	Refs.
770	***Pimephales promelas***, fathead minnow	610
900	***Carassius auratus***, goldfish, 0.1 mg/L, 3 days	204
900	***Carassius auratus***, goldfish, 0.1 mg/L, 3 days	473
1000	***Carassius auratus***, goldfish, 0.1 mg/L, 5 days	143
1000	***Carassius auratus***, goldfish, 0.1 mg/L, 5	204

	days	
1000	***Carassius auratus***, goldfish, 0.1 mg/L, 5 days	473
1140	***Leuciscus idus melanotis***, golden orfe, 42µg/L, 3 days	597
1640	fish	262
1640	***Leptocottus armatus***, staghorn sculpin	269
1640	***Cottus asper***, prickly sculpin	269
9100	***Perca flavescens***, perch	291
12376	***Carassius auratus***, goldfish, 0.2 mg/L, 24 hours	136
15000	***Ictalurus nebulosus***, catfish	291
	Fish (muscle-mean value is 103)	
4	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 16 days	121
4	***Oncorhynchus mykiss***, rainbow trout, 0.25 mg/L, 24 hours	577
13	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	13
13	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	121
13	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	205
39	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	134
39	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	141
74	***Cottus asper***, prickly sculpin	87
84	***Leptocottus armatus***, staghorn sculpin	87
500	bass, 15 µg/L	13
500	bass, 15 µg/L	200

	Fish (gills-mean value is 537)	
50	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 16 days	121
60	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	13
60	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	121
1500	bass	13
1500	bass	300
	Fish (bile-mean value is 40025)	
51	***Oncorhynchus mykiss***, rainbow trout, 12 C, pH 7.3	137
80000	bass	13
80000	bass	300
	Fish (digestive tract-mean value is 170)	
130	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 16 days	121
210	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	13
210	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	121

Table 4.2.3.4 (continued)

B. C. F.	Species, tissues and conditions	Refs.
	Fish (liver-mean value is 1708)	
64	***Oncorhynchus mykiss***, rainbow trout, 0.25 mg/L, 24 hours	577
230	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 16 days	121

350	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	13
350	***Lepomis macrochirus***, bluegill sunfish, 0.1 mg/L, 8 days	121
600	***Cottus asper***, prickly sculpin	87
615	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	134
615	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	141
2100	***Leptocottus armatus***, staghorn sculpin	87
8000	bass	13
8000	bass	300
	Fish (fat-mean value is 128)	
24	***Oncorhynchus mykiss***, rainbow trout, 0.25 mg/L, 24 hours	577
231	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	134
231	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	141
	Fish (plasma-mean value is 138)	
26	***Oncorhynchus mykiss***, rainbow trout, 0.25 mg/L, 24 hours	577
250	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	134
250	***Oncorhynchus mykiss***, rainbow trout, 26 µg/L, 24 hours	141
	Fish (gall bladder-mean value is 1454)	
213	***Carassius auratus***, goldfish, 200 µg/L, 5 hours	136
2695	***Carassius auratus***, goldfish, 200 µg/L, 24 hours	136

	Crustaceans (mean value is 4.5)	
1	***Pacifactacus lenuisculus***, crayfish, pincer	87
1.7	***Palaemonetes pugio***, grass shrimp, 4 days	205
5	***Pacifactacus lenuisculus***, crayfish, tail	87
6.5	***Palaemonetes pugio***, grass shrimp, 2 mg/L, intermolt	578
7	***Cancer productus***, crab, muscle	87
17	***Cancer magister***, crab, muscle	87
	Molluscs (mean value is 96)	
5	***Macoma balthica***, clam, whole body	87
20	***Tapes philippinarum***, carpet shell, 1 day	205
21	***Physa***, sp., snail	12
21	snail	582
33	snail, 2 mg/L, 24 hours	473
41	***Crassostrea virginica***, oyster, 25 µg/L, 28 days	119
41	***Crassostrea virginica***, oyster, 25 µg/L, 28 days	205

Table 4.2.3.4 (continued)

B. C. F.	Species, tissues and conditions	Refs.
50	snail, 2 mg/L, 30 hours	473
78	***Crassostrea virginica***, oyster, 2.5 µg/L, 28 days	119
78	***Crassostrea virginica***, oyster, 2.5 µg/L, 28 days	205
304	***Mytilus edulis***, mussel, 8 days	205

390	***Mytilus edulis***, mussel, 2.5 µg/L	205
390	***Mytilus edulis***, mussel, 2.5 µg/L	682
	Miscellaneous animals (mean value is 828)	
20	***Culex pipiens***, mosquito	12
26	mosquito	582
60	chironomid	291
205	***Daphnia magna***	12
205	***Daphnia magna***	582
3830	***Lanice conchilega***, polychaete worm, 8 days	205
3830	***Lanice conchilega***, polychaete worm, 8 days	682
	Plants and algae (mean value is 334)	
5	***Oedegonium cardiacum***, alga	12
5	alga	582
9.2	oats, 0.2 mg/L	596
400	***Cladophora***, sp., alga	291
1250	***Chlorella fusca***, alga, 5 µg/L,24 hours	597

only bioaccumulation factors of 1 or greater are given in this table.

Table 4.2.3.5 Bioaccumulation Factors Relative to Water for Chlorophenols except PCP.

B. C. F.	Species, tissues and conditions	Refs.
	2-MCP	

6.4	***Carassius auratus***, goldfish, 20 mg/L, 24 hours	273
214	***Lepomis macrochirus***, bluegill sunfish, 9 µg/L, 28 days	379
214	***Lepomis macrochirus***, bluegill sunfish, 9 µg/L, 28 days	423
	4-MCP	
10.1	***Carassius auratus***, goldfish, 20 mg/L, 24 hours	273
	2,4-DCP	
9.2	oat seedling, 0.2 mg/L,14 days	513
10	***Salmo trutta***, brown trout, 20 mg/L, 24 hours	90, 714
34	***Carassius auratus***, goldfish, 20 mg/L, 24 hours	273
	2,3,5-TCP	
12	***Salmo trutta***, brown trout, 0.8 mg/L, 24 hours	90
	2,4,5-TCP	
170	***Poecilia reticulata***, guppy, pH 6, 26C	144
1800	***Pimephales promelas***, fathead minnow, 49.3 µg/L, pH 7.5, 28 days, 22C	605
1900	***Pimephales promelas***, fathead minnow, 4.8 µg/L, pH 7.5, 28 days,22C	605
	2,4,6-TCP	
26.9	***Oncorhynchus mykiss***, rainbow trout, pH 7.3,12C	137
51	***Chlorella fusca***, 50 µg/L, 24 hours, 22C	597
250	***Leuciscus idus melanotis***, golden orfe, pH 7, 30 µg/L, 23C	460
310	***Leuciscus idus melanotis***, golden orfe, 3	597

	days, 30 µg/L, 22C	
580	***Chlorella fusca***, alga, pH 7, 49 µg/L, 24 hours, 23C	460
1000	***Echinodorus***, sp., aquatic plant, 0.5 µg/L	604
1020	***Poecilia reticulata***, guppy, fry, 0.5 µg/L	604
1720	***Oedegonium***, sp., alga, 0.5 µg/L	604
3020	***Lymnaea***, sp., snail, no shell, 0.5 µg/L	604
4460	***Elodea***, sp., aquatic plant, 0.5 µg/L	604
7000	***Poecilia reticulata***, guppy, male, 0.5 µg/L	604
12180	***Poecilia reticulata***, guppy, female, 0.5 µg/L	604
20269	marine worm	642

Table 4.2.3.5 (continued)

BCF	Species, tissues and conditions	Refs.
	TTCPs	
5	***Pacifactacus lenuisculus***, crayfish, pincer	87
5	***Pacifactacus lenuisculus***, crayfish, tail	87
6	***Cancer productus***, crab, muscle	87
12	***Macoma balthica***, whole clam	87
20	***Cancer magister***, crab, muscle	87
34	***Leptocottus armatus***, staghorn sculpin, muscle	87
100	***Cottus asper***, prickly sculpin, muscle	87
320	***Cottus asper***, prickly sculpin, liver	87
1600	***Leptocottus armatus,*** staghorn sculpin, liver	87
	2,3,4,5-TTCP	

17625	marine worm	642
	2,3,4,6-TTCP	
	Fish	
25.5	***Oncorhynchus mykiss***, rainbow trout, pH 7.3,12C, bile	137
30	***Oncorhynchus mykiss***, rainbow trout, (range 30-130)	532
93	***Carassius auratus***, goldfish, 0.8 mg/L	273
100	***Platichthys stellatus***, starry flounder	269
150	***Esox lucius***, pike	274
200	***Leuciscus rutilus***, roach	274
200	***Poecilia reticulata***, guppy, pH 6, 24 hours	144
440	***Leptocottus armatus***, staghorn sculpin	269
440	***Cottus asper***, prickly sculpin	269
450	***Salmo trutta***, brown trout, muscle, 5C	90
450	***Amploplites ruprestris***, rock bass, 20 ng/L	255
	Molluscs	
60	***Mytilus edulis***, mussel, 6-8 ng/L	533
834	***Physa***, sp., snail, 3-6 ng/L, (range 667-1000)	255
2900	***Ferrissia***, sp., limpet, 4 ng/L	255
3750	Sphaeriidae, clam, 4 ng/L	255
	Insects	
250	***Agalus***, sp., diving beetle, 4 ng/L	255
1500	Ephemeroptera, mayfly larva, 3-6 ng/L, (range 1330-1670)	255
3360	Hydropsychida, caddis fly larva, 3-6 ng/L, (range 833-8250)	255
6540	Tipulidae, cranefly larva, 3-4 ng/L, (range	255

	330-12750)	
9000	***Nigronia***, sp., fishfly larva, 6 ng/L	255
10900	Zygoptera, damselfly larva, 4-6 ng/L, (range 5500-19000)	255
18550	Anisoptera, dragonfly larva, 4-6 ng/L, (range 2300-34800)	255
26500	***Pycnopsyche***, sp., caddis fly, 1-4 ng/L, (range 23000-30000)	255

Table 4.2.3.5 (continued)

BCF	Species, tissues and conditions	Refs.
	Leeches	
9590	***Glossiphonia complanata***, 7-35 ng/L, (range 451-22300)	255
10000	leeches, (mixture of 2,3,4,6- and 2,3,5,6-TTCP)	291
13900	***Helobdella stagnalis***, 7-35 ng/L, (range 2260-20000)	255
52200	***Dina dubia***, 1-39 ng/L, (range 3000-122000)	255
89600	***Erpobdella punctata***, 1-15 ng/L, (range 10100-146000)	255
	Miscellaneous organisms	
250	***Orconectes propinquus***, crayfish, 20 ng/L	255
1250	***Cladophora***, sp., alga,(mix of 2,3,4,6- and 2,3,5,6-TTCP)	291
25300	Oligochaete worm, 3-4 ng/L, (range 19250-41300)	255
	2,3,5,6-TTCP	
	Miscellaneous organisms	

1250	***Cladophora***, sp., alga, (mix of 2,3,5,6- and 2,3,4,6-TTCP)	291
10000	leeches, (mixture of 2,3,5,6- and 2,3,4,6-TTCP)	291
	Fish (muscle-mean value is 119)	
21	sunfish	456
53	catfish	456
79	sunfish	456
219	bass	456
223	catfish	456
	Fish (liver-mean value is 3162)	
171	sunfish	456
970	sunfish	456
1000	catfish	456
5000	bass	456
8670	catfish	456

only bioaccumulation factors of 1 or greater are given in this table.

Table 4.2.3.6 Bioaccumulation Factors for Chlorophenols Relative to the Sediments.

Species and Tissues	Bioaccumulation factor	
	PCP	TTCPs
Cancer magister, crab, muscle	0.01-1.3	0.006-1.0
Cancer productus, crab, muscle	0.03-0.14	0.004-0.15
Leptocottus armatus, staghorn sculpin, muscle	0.02-1.0	0.03-1.31

Leptocottus armatus, staghorn sculpin, livers	0.06-160	0.64-70
Pacifactacus lenuisculus, crayfish, pincers	0.03-0.09	0.04-0.18
Pacifactacus lenuisculus, crayfish, tail	0.06-0.30	0.04-0.20
Macoma balthica, clam, whole body	0.08	0.5
Cottus asper, prickly sculpin, muscle	1.1-4.1	0.5-3.6
Cottus asper, prickly sculpin, liver	0.2-56.0	3.2-12.0

Table 4.2.3.7 The Effect of pH on Chlorophenol Toxicity in guppies, *Lebistes reticulata*.

Chlorophenol congeners	pH 5.0	pH 6.0 to pH 6.1	pH 7.0 to pH 7.3	pH 7.8 to pH 8.0	ratio of: pH 8/pH 6
2-MCP		7.1	11.2, 13.8 *	13.5	1.9
3-MCP		6.4	6.4	7.9	1.2
4-MCP	6.3 *	7.8 *	8.5 *	9.0 *	1.1
2,3-DCP					
2,4-DCP		3.3, 3.5 *	4.2, 5.5 *	5.9, 7.6 *	2.0
2,5-DCP					
2,6-DCP		3.9 *	7.8 *	17.9 *	4.6
3,4-DCP					

3,5-DCP					
2,3,4-TCP					
2,3,5-TCP		0.9	1.6	4.7	5.2
2,3,6-TCP					
2,4,5-TCP		1.0 *	1.2 *	3.1 *	3.1
2,4,6-TCP	0.6 *	0.9 *	2.3 *	7.9 *	
3,4,5-TCP					
2,3,4,5-TTCP		0.4	0.8	2.3	5.8
2,3,4,6-TTCP		0.3 *	1.1 *	3.7 *	12.3
2,3,5,6-TTCP		0.4	1.4	3.9	9.8
2,3,4,5,6-PCP	0.04 *	0.11 *	0.45 *	0.91 *	8.3

The numbers in the body of the table are the 14-d, or * 96-h LC_{50} values in mg/L, taken from references 363 and 144 *, respectively.

Table 4.2.3.8 Ratios of Chlorophenols in Water and Rainbow Trout Tissues.

Chlorophenol congeners in the mixture	Ratios after 6 days				
	water	Fish		K o/w	K o/w X (Water)
		Bile	Plasma		
2,4,6-TCP	33	29	5	2	3
2,3,4,6-TTCP	48	39	31	9	19
2,3,4,5,6-PCP	19	32	64	89	78

From Reference 137.
250-450 g fish, pH 7.3-7.4, 11-12C, water hardness 80 mg/L as $CaCO_3$. The actual levels in the water, in µg/L, were;14, 50 and 104 for the TCP, TTCP

and PCP, respectively. The actual levels in the fish, in µg/mL, were 4, 26 and 53 in the plasma, and 1344.5, 1809.8 and 1473.7 in the bile.

Table 4.2.3.9 PCP Uptake Rates in Rainbow Trout, *Oncorhynchus mykiss*, Tissues.

Tissue	Tissue levels of PCP in µg/g or µg/L after the specified times in hours.						
	0 h	1 h	2 h	4 h	8 h	16 h	24 h
Liver		7	9	9	13	14	16
Blood		1	3	4	5	5	6.5
Fat			4		4	8	6
Muscle		0.5	0.5	0.5	1	1	1

From References 134 and 141.
The fish were subjected to a static concentration of PCP at 26 µg/L for the specified times. The values are means of at least 6 fish in 2 duplicate experiments.

Table 4.2.3.10 PCP Elimination Rates in Rainbow Trout, *Oncorhynchus mykiss,* Tissues.

Tissue	Tissue levels of PCP in µg/g or µg/L after the indicated time.						
	0 h	1 h	2 h	4 h	8 h	16 h	24 h
Liver	6.5	5.5	5	4	3	2	1.5
Blood	2.5	2	2	1.5	1	0	0
Fat	1	1	1	2	1	1	1
Muscle	1	1	1	1	1.5	0.5	1

From References 134 and 141.
The fish were subjected to a static concentration of PCP at 26 µg/L for 24 hours, then put in clean running water for the specified times. These data are from a new set of experiments; thus the starting values are not the same as the terminal values of Table 4.2.3.9. The values are means of at least 6 fish in 2 duplicate experiments.

Table 4.2.3.11 PCP Uptake Rates in Bluegill Sunfish, *Lepomis macrochirus*, Tissues.

Tissue	Levels of PCP in µg/g wet weight after the indicated time.				
	1 day	2 days	4 days	8 days	16 days
muscle	0.3	0.3	0.5	1.3	0.4
gill			2.7	6.0	5
liver			35.0	35.0	23.0
digestive tract			9.0	21.0	13.0
combined *			9.0	14.0	8.0

From Reference 121.
* -combined level of gills, liver and digestive tract.
The fish were subjected to a static concentration of PCP at 0.1 mg/L for the specified times.

Table 4.2.3.12 PCP Elimination Rates in Bluegill Sunfish, *Lepomis macrochirus*, Tissues.

Tissue	Levels of PCP in µg/g wet weight after the specified time.				
	1 day	2 days	4 days	8 days	16 days

muscle			0.06	0.03	0.03
gill		0.15	0.16	0.13	0.08
liver		4.0	0.7	4.0	0.6
digestive tract			2.0	0.3	0.13
combined *			2.5	0.8	0.2

From Reference 121.
* -combined level of gills, liver and digestive tract. The fish were subjected to recovery in clean water for the specified times after the accumulation regime shown in Table 4.2.3.11.

Table 4.2.3.13 PCP Accumulation in a Fish, Shrimp and Oyster from flowing seawater, over 96 hours.

Species	water in µg/L	tissues in mg/kg	bioaccumulation factor
Mugil cephalotis,	46.0	0.29	6.3
fish	88.0	6.7	78.8
	157.0	8.8	56.1
Palaemonetes pugio,	32.0	0.05	1.6
shrimp	54.0	0.10	1.9
	76.0	0.24	3.0
	249.0	0.43	1.7
Crassostrea virginica,	2.8 26.0	0.18 0.86	64.3 33.1

oyster			

From Reference 683.

Table 4.2.4.1 Depuration 1/2 Lives (DP50) of Chlorophenols in Animals.

Chlorophenol	Hours	Species	References
2-MCP	<24	***Lepomis macrochirus***, whole sunfish,	379
2,4,5-TCP	12	***Pimephales promelas***, fathead minnow	605
PCP	5	trout, liver	134, 141
PCP	7	trout, blood	134, 141
PCP	7	trout, muscle	134, 141
PCP	7	trout, heart	134, 141
PCP	10	***Carassius auratus***, whole goldfish, (DP80 is 20 hours)	204
PCP	10	trout, gills	134, 141
PCP	13	male rat, whole body	277
PCP	15	rat, blood	464
PCP	17	female rat, whole body	277
PCP	23	trout, fat	134, 141
PCP	24	mice, whole body	407
PCP	<24	fish, whole body	205
PCP	41	male rhesus monkey, whole body	570
PCP	72	male rhesus monkey, plasma	570

PCP	78	***Macaca mulatta***, monkey, plasma	464
PCP	84	female rhesus monkey, plasma	570
PCP	92	female rhesus monkey, whole body	570
PCP	113	***Fundulus similis***, whole body	10
PCP	168	trout, whole body	131, 577

Table 5.0 Cytotoxicity, Fetotoxicity/Embryotoxicity and Teratogenicity of the Chlorophenols.

congener	concentration or dose	refs.	species and effects
CYTOTOXICITY			
4-MCP	250 mg/L	448 502 503	***Vicia faba*** root cells had decreased mitotic index, anaphase bridges, cytomixis, lagging chromosomes, and disturbed meta- and ana-telophases
2,4-DCP	16.3 g/l	448 502 503	application as a spray to ***Vicia faba*** buds resulted in meiotic alterations of chromosome stickiness, lagging chromosomes and anaphase bridges
2,4-DCP	62.5 mg/L	448, 502	***Vicia faba*** root cells developed meiotic

		503	alterations of chromosome stickiness, lagging chromosomes, anaphase bridges, chromosome disintegration, cytomixis, and disturbed prophase and anaphase.
PCP	174 ng/L, 87 ng/L 43.5 ng/L	448, 502 503	***Vicia faba*** root cells developed a decreased mitotic index, anaphase bridges, cytomixis, lagging chromosomes, and disturbed meta- and ana-telophase
FETOTOXICITY/ EMBRYOTOXICITY			
2,4,5-TCP	9.0 mg/kg	480	pregnant mice receiving oral doses during days 6-15 of gestation showed increased rates of resorption and embryo mortality.
2,3,4,6-TTCP	10 mg/kg/d	322	oral doses to female rats on day 21 of gestation had no effect
2,3,4,6-TTCP	30 mg/kg/d	322	oral doses to female rats on day 21 of gestation produced delayed ossification of skull bones in 17% of the young.
2,3,4,6-TTCP	25 mg/kg/d	531	there was no effect on female rats or their embryos.
2,3,4,6-	100-200	531	caused reduced maternal

TTCP	mg/kg/d		weight gain and 3-4% pre-implantation losses

congener	concentration or dose	refs.	species and effects
2,3,4,5,6-PCP	26-30 mg/kg	38 244, 288 322	causes reduced numbers of offspring, neonatal survival rates and weanling growth rates in rats
2,3,4,5,6-PCP	30 mg/kg/d	737 738	NOAEL for rabbit and rat embryos
2,3,4,5,6-PCP	0.34-60 mg/kg	558, 564 565 566 567	causes fetotoxicty and maternal toxicity in rats, mice and hamsters
2,3,4,5,6-PCP	80 mg/kg/d	738	caused resorptions, reduced live litter size and fetal body weights and malformations (gastroschisis, hydrocephaly, diagramatic hernia, kidney dilation, extra rib pairs, delayed ossification) in rats
TERATOGENICITY			
2,4,5-TCP	9.0 mg/kg	480	oral doses at days 6-15 of gestation in mice does not lead to any observed teratogenic effects.
PCP	30 mg/kg/d	737 738	NOAEL for oral doses during days 6 to 18 of gestation in rabbits and days 6 to 15 of gestation in rats.

PCP	30 mg/kg/d	227 288	given 2 months prior to and right through to lactation causes no teratogenic effects.

Table 5.6 Ames Mutagenicity Assays using *Salmonella typhimurium*

(strains TA98, TA100, TA1535, TA1537 and TA1538)

chlorophenol congener		µg/plate of the chlorophenol								
	***	0.1	0.5	1.0	5.0	50	100	200	500	1000
4-MCP								neg. **		
2,3-DCP	neg.									
2,4-DCP			neg.		neg.	neg.			neg. *	
2,5-DCP			neg.		neg.	neg.			neg. *	
2,6-DCP			neg.		neg.	neg.			neg. *	
3,4-DCP			neg.		neg.	neg.			neg. *	
3,5-DCP			neg.		neg.	neg.			neg. *	
2,3,4-TCP		neg.		neg.			neg.			neg.
2,3,5-TCP		neg.	neg.	neg.	neg.	neg.	neg.		neg.	neg.
2,3,6-TCP	neg.									
2,4,5-TCP			neg.		neg.	neg.			neg. *	
2,4,6-TCP		neg.	neg.	neg.	neg.	neg.	neg.		neg.	neg.

									*	
2,3,4,6-TTCP			neg.		neg.	neg.			neg. *	
2,3,4,5,6-PCP	neg.									

* toxic but not mutagenic. ** slight increase in only one strain. *** 2,3-DCP, 2,3,6-TCP and PCP are reported as negative in the Ames mutagenicity test but no details of the concentrations used are available to put into this table. PCP was also negative to all *Salmonella* stains at up to 10 mg/plate and *E. coli* negative to PCP at up to 5 mg/plate.

Table 5.8.1 The Product of the LC_{100} and the time it takes to kill fish with PCP.

LC_{100} in mg/L	***Notropis umbratilis*** (redside shiner)		***Fundulus notatus*** (blackstripe topminnow)	
	minutes to die	(LC_{100}) X (time)	minutes to die	(LC_{100}) X (time)
5.0	16	80	90	450
4.0	20	80	100	400
3.0	30	90	150	450
2.0	40	80	225	450
1.5	50	75	285	427
1.0	100	100	435	435
0.8	125	100	585	468
0.6	160	96		
mean		87.6		440
stnd. dev.		10.1		21.9

From Reference 69.

Table 6.2.1 Taste Thresholds for Chlorophenols in Drinking Water.

Chlorophenol	Thresholds in µg/L., (refs.), - @ temperature in C
2-MCP	**0.1** (335, 364, 366)-21; 2 (183), 50 (436)-40
3-MCP	**0.1** (335, 366, 372)-21; 50 (436)-40
4-MCP	**0.1** (335, 366, 372)-21; 50 (436)-40; >1000 (183)
2,3-DCP	**0.04** (366)
2,4-DCP	**0.3** (335, 366, 372); 2 (182), 8 (183)
2,5-DCP	0.5 (366, 372)
2,6-DCP	**0.2** (366, 372); 2 (183, 424); 3 (183)
3,4-DCP	**0.3** (366, 372)
2,3,6-TCP	**0.5** (366)
2,4,5-TCP	**1.0** (366, 372)
2,4,6-TCP	**2** (366, 372); >1000 (372)
2,3,4,6-TTCP	**1** (366, 424)
2,3,4,5,6-PCP	**30** (205, 366)

The lowest taste or odour value for each chlorophenol in Tables 6.2.1 and 6.2.2 is
in bold face.

Table 6.2.2 Odour Thresholds for Chlorophenols in Drinking Water

congeners	Thresholds in µg/L., (refs.), - @ temperature

	in C
2-MCP	0.33 (364, 463)-20; 0.33 (364, 463)-30; 1 (436)-60; 2 (183, 364)-25; 2.5 (364)-60; 4 (183); 5 (436)-20
3-MCP	1 (436)-60; 5 (436)-20; 50 (366, 372)-21; 200 (436)-30
4-MCP	1 (436)-60; 5 (436)-20; 33 (463)-30; 60 (366)-21; 250 (183)-25; >1000 (183)
2,3-DCP	30 (366)-21
2,4-DCP	0.65 (463)-30; 2 (183, 495)-25; 8 (183); 40 (366, 495)-21; 100 (372)
2,5-DCP	**0.33** (463)-30; 0.45 (424); 2 (639); 30 (366, 372)-21; 33 (372)-30
2,6-DCP	2 (183); 3 (183, 362)-25; 200 (366)-21
3,4-DCP	100 (366, 372)-21
2,3,6-TCP	300 (366)-21
2,4,5-TCP	11 (183, 333, 372, 463)-25; 200 (366)-21
2,4,6-TCP	100 (372, 463)-30; 300 (366, 372)-21; 1000 (372)-25; >1000 (183, 463)-25
2,3,4,6-TTCP	600 (366, 424)-21; 857 (309)-30; 900 (424, 463)-30; 47000 (309)-60
2,3,4,5,6-PCP	857 (463)-30; 1600 (205, 366)-20

Table 6.3 Literature Guidelines for Chlorophenols in Drinking Water.

Guidelines	Source, Type of Guideline or Effect	References

	Phenolics	
1.0 µg/L	IJC-1974	677
	Sum of all Chlorophenols	
2.0 µg/L	Canada, H&W-1978, BC-1982	328, 329
	2-MCP	
0.1 µg/L	Chemosphere: 15-1986 taste, US EPA-1979, 1980 & 1986 organoleptic, criterion	335, 364, 672, 696
1.0 µg/L	WHO-1984 MAC, odour & taste	225
	3-MCP	
0.1 µg/L	Chemosphere: 15-1986 taste, US EPA-1979,1980 & 1987 criterion	335, 672, 694, 695
	4-MCP	
0.1 µg/L	Chemosphere: 15-1986 taste, US EPA-1979, 1980, 1986 &1987 criterion	335, 672, 694, 695, 696
1.0 µg/L	WHO-1984 MAC, odour & taste	225
	2,3-DCP	
0.04 µg/L	US EPA-1979,1980,1986 & 1987 criterion	672, 694, 695, 696
2.0 µg/L	USSR-1973 organoleptic	638
	2,4-DCP	
0.1 µg/L	Chemosphere: 15-1986 potability limit	335
0.3 µg/L	Canada-1987 aesthetics, Chemosphere: 15-1986 taste, US EPA-1979, 1980, 1986 & 1987 criterion	110, 335, 672, 694, 695, 696
1.0 µg/L	WHO-1984 taste & odour, Chemosphere: 15-1986 taste	225, 335
2.0 µg/L	USSR-1973 organoleptic	638

10.0 µg/L	WHO-1984 MAC	225
900 µg/L	Canada-1987 MAC	110
3000 µg/L	WHO-1984 toxicity	225
3090 µg/L	US EPA-1980, 1986 & 1987 toxicity	694, 695, 696
	2,5-DCP	
0.5 µg/L	US EPA-1979, 1980, 1986 & 1987 criterion	672, 694, 695, 696
2.0 µg/L	USSR-1973 organoleptic	638
	2,6-TCP	
0.2 µg/L	US EPA-1979, 1980, 1986 & 1987 criterion	672, 694, 695, 696
1.0 µg/L	WHO-1984 MAC, taste	225
2.0 µg/L	USSR-1973 organoleptic	638
10.0 µg/L	WHO-1984 odour	225

Table 6.3 (continued)

Guidelines	Source, Type of Guideline or Effect	References
	3,4-DCP	
0.3 µg/L	US EPA-1979, 1980, 1986 & 1987 criterion	672, 694, 695, 696
2.0 µg/L	USSR-1973 organoleptic	638
	TCPs	
0.4 µg/L	USSR-1970 organoleptic	424
35 µg/day	IJC-1986 ADI per adult	227
	2,3,6-TCP	
0.4 µg/L	USSR-1973 organoleptic	638
	2,4,5-TCP	
0.1 µg/L	WHO-1984 MAC	225

0.4 µg/L	USSR-1973 organoleptic	638
1.0 µg/L	WHO-1984 taste, US EPA-1979, 1980, 1986 & 1987 criterion	225, 672, 694, 695, 696
100 µg/L	WHO-1984 odour, US EPA-1980 organoleptic	225, 372
2600 µg/L	WHO-1984 toxicity, US EPA-1980, 1980, 1986 & 1987 public health	225, 372, 694, 695, 696
7000 µg/kg	Chemosphere: 14-1985 ADI, adult	226
	2,4,6-TCP	
0.1 µg/L	WHO-1984 aesthetics	225
0.12 µg/L	US EPA-1980, 1986 & 1987 carcinogenicity lifetime risk increase of 10-7	694, 695, 696
0.4 µg/L	USSR-1973 organoleptic	638
1.0 µg/L	WHO-1984 MAC, taste	225
1.2 µg/L	US EPA-1980, 1986 & 1987 carcinogenicity lifetime risk increase of 10-6	694, 695, 696
2.0 µg/L	US EPA-1979 criterion, Canada-1987 & 1989 aesthetic, US EPA-1980, 1980, 1986 & 1987 organoleptic	110, 373, 672, 690, 694, 695, 696
5.0 µg/L	Canada-1987 and 1989 MAC	110, 690
10.0 µg/L	WHO-1984 MAC	225
12 µg/L	US EPA-1980, 1986 & 1987 carcinogenicity lifetime risk increase of 10-5	694, 695, 696
12.0 µg/L	WHO-1984 carcinogenicity	225
62.0 µg/L	WHO-1984 ADI	225

100 µg/L	WHO-1984 odour	225

Table 6.3 (continued)

Guidelines	Source, Type of Guideline or Effect	References
	2,3,4,6-TTCP	
0.1 µg/L	WHO-1984 (MAC)	225
1.0 µg/L	Canada-1987 & 1989 aesthetics, WHO-1984 taste, US EPA-1979, 1980, 1986 & 1987 criterion	110, 225, 672, 690, 694, 695, 696
100 µg/L	Canada-1987 & 1989 MAC	110, 690
1000 µg/L	WHO-1984 odour	225
	2,3,4,5,6-PCP	
1.0 µg/L	WHO-1984 MAC	225
2.0 µg/L	Canada-1978 MAC	264
3.0 µg/L	WHO-1984 ADI	225
3.0 µg/L	US NAS-1977 ADI, adult	224
10.0 µg/L	WHO-1984 MAC	225, 327
21.0 µg/L	WHO-1984 toxicity, US NAS-1977 NAE	224, 225
30.0 µg/L	Canada-1987 & 1989 aesthetics, US EPA-1988 taste, US EPA-1980, 1980 & 1986 organoleptic	110, 405, 414, 690, 694, 696
60 µg/L	Canada-1987 & 1989 MAC	110, 690
100 µg/L	WHO-1984 taste	225
220 µg/L	US EPA-1986 MAC	266
300 µg/L	USSR-1973 organoleptic	638
680 µg/L	US EPA-1984 MAC	105
1000 µg/L	WHO-1984 odour	225

1010 µg/L	US EPA-1980 toxicity	414
1600 µg/L	US EPA-1988 odour	405
	NaPCP	
5000 µg/L	USSR-1973 organoleptic	638

Table 7.1 The Toxicity of some Commercial Chlorophenol Preparations to Pulp and Paper Fungi and Bacteria.

agent (active ingredients)	***Aerobacter aerogenes***	***Bacillus mycoides***	***Aspergillis niger***	***Penicillium expansum***
Biocide Dis-124 (polychlorinated)	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 chlorophenols)	200	25	55	550
Nalco 21M (NaPCP)	200	5	45	40
Nalco 21S (2,4,5-NaTCP,NaPCP)	25	5	35	55
Nalco 201 (chlorinated phenols)	50	9	35	65
Santobrite (NaPCP)	250	4	35	30

The body of the Table gives the mg/L of active ingredient needed to cause **complete** inhibition of growth.

Table 7.1.1 The Effect of Chlorophenols on Fungal Growth.

congener	mg/L	species/conditions	effect	ref.
4-MCP	47.6	***Tricoderma viride***, 25C	40-h EC_{50}	381
2,4-MCP	8.6	***Tricoderma viride***, 25C	40-h EC_{50}	381
2,3,4-TCP	5.9-24.7	16 species of fungi	EC_{100}	432
2,3,5-TCP	2.96-11.8	16 species of fungi	EC_{100}	432
2,3,5-TCP	25.0	***Aspergillis niger***, 22C	EC_{100}	431
2,4,6-TCP	6.9	***Tricoderma viride***, 25C	40-h EC_{50}	381
2,3,4,5-TTCP	1.74-13.9	16 species of fungi	EC_{100}	432
2,3,4,6-TTCP	6.96-23.2	16 species of fungi	EC_{100}	432
2,3,4,6-TTCP	0.8	***Tricoderma viride***, 25C	40-h EC_{50}	381
2,3,5,6-TTCP	29-464	16 species of fungi	EC_{100}	432
2,3,4,5-TTCP	6.96	***Aspergillis fumigatus***	EC_{100}	452
2,3,4,5-TTCP	0.696	***Microsporon canis***	EC_{100}	452
2,3,4,5-TTCP	23.2	***Trichophyton rubrum***	EC_{100}	452
2,3,4,5-TTCP	23.2	***Candida albicans***	EC_{100}	452

2,3,4,5-TTCP	2.32	***Trichophyton mentagraphytes***	EC_{100}	452
2,3,4,6-TTCP	23.2	***Aspergillis fumigatus***	EC_{100}	452
2,3,4,6-TTCP	2.3	***Microsporon canis***	EC_{100}	452
2,3,4,6-TTCP	23.2	***Trichophyton rubrum***	EC_{100}	452
2,3,4,6-TTCP	23.2	***Candida albicans***	EC_{100}	452
2,3,4,6-TTCP	6.96	***Trichophyton mentagraphytes***	EC_{100}	452
2,3,5,6-TTCP	69.6	***Aspergillis fumigatus***	EC_{100}	452
2,3,5,6-TTCP	6.96	***Microsporon canis***	EC_{100}	452
2,3,5,6-TTCP	23.2	***Trichophyton rubrum***	EC_{100}	452
2,3,5,6-TTCP	23.2	***Candida albicans***	EC_{100}	452
2,3,5,6-TTCP	23.2	***Trichophyton mentagraphytes***	EC_{100}	452
2,3,4,5,6-PCP	1.3	***Aspergillis niger***, glucose metabolism effects	EC_{100}	569

Table 7.1.2 The Effects of Chlorophenols on Bacteria.

chlorophenol	mg/L	species/conditions/test parameters	effect	ref.

		Spirillum volutans		
3,5-DCP	5.0	mobility	IC_{50}	47
		Bacillus cereus (resazurin assay)		
2,3,4,5,6-PCP	3.9	range 2.5-5.2 depends on carrier	EC_{50}	179
2,3,4,5,6-PCP	9.5	range 8.0-11.0 depends on	EC_{50}	179
		Pseudomonas fluorescens		
2,3,4,5,6-PCP	8.6	at maximum stationary phase	LC_{50}	557
2,3,4,5,6-PCP	10		1-h LC_0	557
2,3,4,5,6-PCP	18	at early log phase	LC_{50}	557
2,3,4,5,6-PCP	25		1-h LC64	557
2,3,4,5,6-PCP	29	at mid log phase	LC_{50}	557
2,3,4,5,6-PCP	50		1-h LC_{84}	557
2,3,4,5,6-PCP	75		1-h LC_{100}	557
		sewage or activated sludge cultures		
2-MCP	380	sewage, reduction in respiration rate	IC_{50}	196
4-MCP	178	sewage, reduction in respiration rate	IC_{50}	196

2,4-DCP	49.5	sewage, reduction in respiration rate	IC_{50}	196
2,4-DCP	50	sludge, TTC dehydrogenase test	EC	512
2,4-DCP	500	sludge, TTC dehydrogenase test	EC_{50}	512
3,4-DCP	74	from sewage, 22C	1-h IC_{50}	47
3,5-DCP	4.7	reduction in respiration rate	3-h EC_{50}	44
3,5-DCP	7.42	mixed culture, reduction in respiration	IC_{50}	46
3,5-DCP	11	reduction in respiration rate	3-h EC_{50}	44
3,5-DCP	11.94	mixed culture, reduction in respiration	IC_{50}	46
3,5-DCP	12.5	from sewage, reduction in respiration	IC_{50}	196
3,5-DCP	18	sludge, reduction in respiration	3-h EC_{50}	238
2,4,5-TCP	8.3	sludge, reduction in respiration	3-h EC_{50}	237
2,4,5-TCP	8.3	sludge, reduction in respiration	3-h EC_{50}	238
TCP	60	inhibition of gas production, lab. static bioassay in fresh water	EC_{50}	461

Table 7.1.2 (continued)

chlorophenol	mg/L	species/conditions/test parameters	effect	ref.
PCP	2.5	sludge, reduction in	3-h IC_{50}	196

		respiration rate		
PCP	20	sludge, reduction in growth rate	3-M EC_{50}	45
PCP	21	sludge, reduction in respiration	3-h EC_{50}	237
PCP	21	sludge, reduction in respiration	3-h EC_{50}	238
PCP	36	activated sludge, reduction in growth	1-W EC_{50}	45
PCP	>40	sludge, reduction in nitrification	EC_{50}	45
PCP	49	sludge, reduction in respiration	1-M EC_{50}	45
PCP	61	sludge, reduction in respiration	1-W EC_{50}	45
PCP	134	sludge, reduction in respiration	3-M EC_{50}	45
		Photobacterium phosphoreum **(microtox)**		
2-MCP	14.7	reduced light output	15-m EC_{50}	240
2-MCP	6.8	reduced light output, static bioassay	30-m EC_{50}	367
3-MCP	14.1	reduced light output, static bioassay	30-m EC_{50}	367
2,3-DCP	4.6	reduced light output	15-m EC_{50}	240
2,4-DCP	5.5	reduced light output, static bioassay	30-m EC_{50}	367
3,5-DCP	4.15	reduced light output, 15C	5-m IC_{50}	46
3,5-DCP	4.24	reduced light output, 15C	10-m	46

			IC_{50}	
3,5-DCP	4.47	reduced light output, 15C	5-m IC_{50}	46
3,5-DCP	4.70	reduced light output, 15C	30-m IC_{50}	46
2,3,4-TCP	1.1	reduced light output	15-m EC_{50}	240
2,3,4-TCP	1.1	reduced light output, distilled water	15-m EC_{50}	435
2,3,4-TCP	1.25	reduced light output	30-m EC_{50}	367
2,3,4-TCP	1.4	reduced light output, natural water #3	15-m EC_{50}	435
2,3,4-TCP	1.4	reduced light output, natural water #5	15-m EC_{50}	435
2,3,4-TCP	1.61	reduced light output	15-m EC_{50}	367
2,3,4-TCP	1.7	reduced light output, natural water #2	5-m EC_{50}	435
2,3,4-TCP	1.76	reduced light output	15-m EC_{50}	240
2,3,4-TCP	1.8	reduced light output, natural water #1	15-m EC_{50}	435
2,3,4-TCP	2.2	reduced light output, natural water #4	15-m EC_{50}	435
2,3,5-TCP	1.11	reduced light output, static bioassay	30-m EC_{50}	367
2,3,5-TCP	1.37	reduced light output, static bioassay	15-m EC_{50}	367
2,3,5-TCP	1.76	reduced light output, static bioassay	5-m EC_{50}	367

Table 7.1.2 (continued)

chlorophenol	mg/L	species/conditions/test parameters	effect	ref.
2,3,4,5-TTCP	0.176	reduced light output	30-m EC_{50}	367
2,3,4,5-TTCP	0.207	reduced light output	15-m EC_{50}	367
2,3,4,5-TTCP	0.335	reduced light output	5-m EC_{50}	367
2,3,4,5-TTCP	0.4	reduced light output	15-m EC_{50}	240
2,3,4,5-TTCP	0.4	reduced light output, de-ionized water	15-m EC_{50}	435
2,3,4,5-TTCP	0.5	reduced light output, natural water #1	15-m EC_{50}	435
2,3,4,5-TTCP	0.6	reduced light output, natural water #4	15-m EC_{50}	435
2,3,4,5-TTCP	0.7	reduced light output, natural water #5	15-m EC_{50}	435
2,3,4,5-TTCP	1.5	reduced light output, natural water #2	15-m EC_{50}	435
2,3,4,5-TTCP	1.7	reduced light output, natural water #3	15-m EC_{50}	435
2,3,4,6-TTCP	1.27	reduced light output	30-m EC_{50}	367
2,3,4,6-TTCP	1.46	reduced light output	15-m EC_{50}	367
2,3,4,6-TTCP	1.88	reduced light output	5-m EC_{50}	367
2,3,5,6-TTCP	2.21	reduced light output	30-m EC_{50}	367
2,3,5,6-TTCP	2.54	reduced light output	15-m EC_{50}	367

2,3,56-TTCP	2.78	reduced light output	5-m EC_{50}	367
2,3,4,5,6-PCP	0.48	reduced light output, 15C	30-m EC_{50}	181
2,3,4,5,6-PCP	0.61	reduced light output, 15C	15-m EC_{50}	181
2,3,4,5,6-PCP	0.65	reduced light output, 15C, pH 6.0	10-m EC_{50}	178
2,3,4,5,6-PCP	0.72	reduced light output, 15C, pH 6.5	10-m EC_{50}	178
2,3,4,5,6-PCP	0.79	reduced light output, 15C, pH 7.0	10-m EC_{50}	178
2,3,4,5,6-PCP	0.8	reduced light output, 20C	10-m EC_{50}	178
2,3,4,5,6-PCP	0.8	reduced light output, 20C	15-m EC_{50}	178
2,3,4,5,6-PCP	0.8	reduced light output, 20C	30-m EC_{50}	178
2,3,4,5,6-PCP	0.8	reduced light output, 15C	30-m EC_{50}	178
2,3,4,5,6-PCP	0.82	reduced light output, 15C, pH 7.5	10-m EC_{50}	178
2,3,4,5,6-PCP	0.9	reduced light output, 15C	15-m EC_{50}	178
2,3,4,5,6-PCP	1.0	reduced light output, 15C	10-m EC_{50}	178
2,3,4,5,6-PCP	1.0	reduced light output, 15C	15-m EC_{50}	182
2,3,4,5,6-PCP	1.03	reduced light output, 15C	5-m EC_{50}	181
2,3,4,5,6-PCP	1.1	reduced light output, 20C	5-m EC_{50}	178

2,3,4,5,6-PCP	1.14	reduced rate of ATP formation	EC_{50}	239
2,3,4,5,6-PCP	1.5	reduced light output, 15C	5-m EC_{50}	178
2,3,4,5,6-PCP	2.23	reduced photosynthesis	EC_{50}	239
2,3,4,5,6-PCP	3.51	reduced growth rate	EC_{50}	239

Table 7.1.2 (continued)

chlorophenol	mg/L	species/conditions/test parameters	effect	ref.
		dairy cultures (kefir, butter, cheese and yogurt)		
2,4,5-TCP	100	growth inhibition	EC_{100}	486
2,3,4,5,6-PCP	100	growth inhibition	EC_{100}	486
		Pseudomonas sp.		
2,4-DCP	25	growth inhibition	EC_{100}	511
		Bacillus subtilis		
2,3,4,5,6-PCP	1.3	inhibition of proline and glycine uptake	EC_{100}	568

Table 7.2 The Effect of Chlorophenols in Water on the Germination of Radish and Sorghum Seeds.

chlorophenol	EC_{50} in mg/L, Ref. 651

congeners	radish ***Raphanus sativus***	sorghum ***Sorghum sudanense***
2-MCP	88.7	279
3-MCP	61.8	13.5
4-MCP	107.7	180
2,4-DCP	49.1	100
2,5-DCP	49.1	25.4
2,4,5-TCP	32	34.2
2,4,6-TCP	45	110.2
2,3,4,6-TTCP	16	67
2,3,4,5,6-PCP	7.2	5.4

Table 7.5.1 The Effects of Pentachlorophenol on Mammals

mg/L	Species	Conditions	Effect	Ref.
		total quantity is unknown		
12.5	man	oral and dermal, daily	EC	545
51	calf	Ca salt in drinking water	5-wk EC	167
60	calf	Na salt in drinking water	7-wk EC	167
106.5	man	dermal application, hands	2h.EC	545
1325	cows	oral in kerosene	LC_{100}	661
10000	man	dermal application	EC	162

mg dose	Species	Conditions	Effects	Ref.
4	monkey	rhesus, Na salt	NOEL	167
10	dog	daily in diet	4-mon LD_0	162
3200	calf	in 40 gallons of water	4-d NOEL	167
18000	man	70 kg	LD_{100}	163
	rats			
10	daily in diet		4 mon LD_0	162
20	injection, causes temperature rise		1.5-2.0C	544
	rabbits			
22-23	intravenous		1.5-h LD_{100}	541
40-50	dermal in pine oil		9-h LD_{100}	541
60-70	dermal in fuel oil		1.5-h LD_{100}	541
70-85	subcutaneous in olive oil		3-h LD_{100}	541
70-90	oral in fuel oil		2-h LD_{100}	541
90-100	dermal in fuel oil		1.5-h LD_{100}	541
100	subcutaneous		7-h LD_{100}	541
100-130	oral in olive oil		10-h LD_{100}	541
110-130	dermal in Dione oil		5-h LD_{100}	541
130-170	dermal in fuel oil		6-h LD_{100}	541
250	dermal		3-h LD_{100}	541
250-	oral		3-h LD_{100}	541

300			
350	dermal in olive oil	LD_{100}	654

Table 7.5.1 (continued)

mg/kg	Species	Conditions	Effect	Ref.
3.5	dog	chronic, oral, capsules	NOEL	752
27-210	many Sp.	oral, solvent & purity vary	LD_{50}	199
35-50	calves		11-d LD_{50}	323
140	calf	oral	LD_{50}	655
100	guinea pig	oral	LD_{50}	287
120	sheep	oral	LD_{50}	655
150-200	dog	oral	LD_{50}	287
168	hamsters	Syrian, both sexes, oral	LD_{50}	543
257	man	oral	LD_{100}	163
294	gerbils	female, oral in PPG	LD_{50}	450
mg/kg	Conditions		Effect	Ref.
	rats			
0.34	reproduction study		NOEL	558
0.4	tech. grade, reproduction study, ovum implantation		LD_{20}	558
3	reproductive study		NOEL	199
3	daily, reproductive study		NOEL	38

3	oral, daily, pre- and post-mating	62-d NOEL	539
3.4	reprod. study, 12 % pup loss in 1st 2 weeks	LD12	558
4	tech. grade, reprod. study, fetal weight loss	EC	558
5	pregnant female, oral in corn oil, daily	NOEL	288
5	female, fetal effects study	NOEL	199
15	pregnant female, oral in corn oil, daily	EC	288
25	growth and blood effects study	NOEL	287
27	oral	LD_{50}	225
27	oral in fuel oil	3-h LD_{50}	541
27.3	oral in fuel oil	LD_{50}	542
27-210	solvent dependent, least in water	LD_{50}	199
30	daily, 1 generation reproduction study	EC	38
30	oral, daily, pre- and post-mating, fewer pups	62-d EC	539
30	pregnant, oral in corn oil, daily, weight loss	EC	288
34	rats, reproductive study 20% pup weight loss	LD13	558
40	tech. grade, reprod. study, 20% fetal wt. loss	EC	558
50	pregnant, oral in corn oil, daily weight loss	fetal LD_{100}	288
50-350	females more sensitive than males	LD_{50}	232

50-350	females more sensitive than males	LD_{50}	315
56	male, intraperitoneal	LD_{50}	652
65	oral at 3-4 days old	LD_{50}	539
66	Na salt given subcutaneously	2-h LD_{50}	541

Table 7.5.1 (continued)

mg/kg	Conditions	Effect	Ref.
77.9	oral in olive oil	LD_{50}	592
78	oral in olive oil	3-h LD_{50}	541
78	oral	LD_{50}	162
78	oral	LD_{50}	165
78	oral	LD_{50}	166
90	subcutaneous	LD_{50}	541
135	female, commercial grade	LD_{50}	288
135	female, oral	LD_{50}	526
146	male,	LD_{50}	540
146	oral	LD_{50}	161
150	adults, oral	LD_{50}	539
175	female	LD_{50}	540
205	male, commercial grade	LD_{50}	288
205	male, oral	LD_{50}	526
210	Na salt orally	2-h LD_{50}	541
250-300	oral	LD_{50}	541
276	intraperitoneal	LD_{50}	229
320	adult males, oral, tech. grade in pine oil	LD_{50}	540
330	females, oral, tech. grade in pine	LD_{50}	540

	oil		
	mice (female C57, except for Ref. 287)		
32	oral in 40% ethanol	LD_{50}	450
36	oral in 40% ethanol	LD_{50}	450
59	intraperitoneal in propylene glycol	LD_{50}	450
74	oral in 40% ethanol	LD_{50}	450
120-140	oral	LD_{50}	287
150	oral in propylene glycol	LD_{50}	450

Table 7.5.2 The Effects of Chlorophenols on Mammals and Birds

mg/kg	Species	Conditions	Effect	Ref.
		2-MCP		
120	rabbit	intravenous, minimum dose	LD	364
230	male rat	intraperitoneal	LD_{50}	229
230	rat	intraperitoneal, minimum dose	LD	364
440	blue fox		LD_{50}	364
670	rat	oral	LD_{50}	364
800	guinea pig	subcutaneous, minimum dose	LD	364
950	rat	subcutaneous	LD_{50}	364
950	rabbit	subcutaneous	LD_{50}	364

2230	rat	oral in olive oil	LD_{50}	652
3160	male rat	subcutaneous	LD_{50}	652
		3-MCP		
335	male rat	intraperitoneal	LD_{50}	372
335	male rat	intraperitoneal	LD_{50}	229
697	rat	oral in olive oil	LD_{50}	372
1730	rat	subcutaneous	LD_{50}	372
1865	male rat	oral in olive oil	LD_{50}	652
4630	male rat	subcutaneous in olive oil	LD_{50}	652
		4-MCP		
250	rat	intraperitoneal	LD_{50}	372
281	rat	intraperitoneal	LD_{50}	229
500	rat	oral	LD_{50}	372
500	rat	oral	LD_{50}	653
660	rat	oral in olive oil	LD_{50}	372
660	rat	oral in olive oil	LD_{50}	652
860	mouse	oral	LD_{50}	372
1030	rat	subcutaneous in olive oil	LD_{50}	372
1030	rat	subcutaneous in olive oil	LD_{50}	652
1500	rat	dermal	LD_{50}	372
1500	rat	dermal	LD_{50}	653
		MCPs		
250	rats		LD_{50}	315
250	rats		LD_{50}	232

Table 7.5.2 (continued)

mg/kg	Species	Conditions	Effect	Ref.
		2,4-DCP		
9	sheep and cattle	muscle and fat tissue levels	28-d NOEL	315
9	sheep and cattle	liver and kidney tissue levels	28-d EC-high	315
30	sheep and cattle	muscle and fat tissue levels	28-d NOEL	315
30	sheep and cattle	liver and kidney tissue levels	28-d EC-high	315
45	mouse	oral, daily	6-mon NOEL	312
60	sheep and cattle	muscle and fat tissue levels	28-d NOEL	315
60	sheep and cattle	liver and kidney tissue levels	28-d EC-high	315
100	mouse	oral, daily	6-mon NOEL	312
150	pigeons	oral; 91% eliminated in 5 days	not lethal	497
230	mouse	oral, daily	6-mon EC	312
430	rat	intraperitoneal	LD_{50}	229
1600	mouse	oral	LD_{50}	312
1630	mice, both sexes	oral	LD_{50}	312
1720	rat	in fuel oil	LD_{50}	652
1730	rat	subcutaneous	LD_{50}	496
3670	male rat	oral	LD_{50}	312
4500	female rat	oral	LD_{50}	312
		2,6-DCP		
5.5	rat livers	mitochondrial	EC50	425

		enzymes		
390	rat	intraperitoneal	LD_{50}	372
390	male rat	intraperitoneal	LD_{50}	229
1730	rat	subcutaneous	LD_{50}	426
2940	rat	oral	LD_{50}	426
		DCPs		
250	rats		LD_{50}	315
250	rats		LD_{50}	232
		2,3,6-TCP		
308	male rat	intraperitoneal	LD_{50}	229
		2,4,5-TCP		
1	rabbit	oral, 20 doses in 28 days	28-d NOEL	313
1	rats, both sexes	oral	98-d NOEL	313
1	man	oral, urine and fecal residue levels	NOEL	489

Table 7.5.2 (continued)

mg/kg	Species	Conditions	Effect	Ref.
3	rats, both sexes	oral	98-d NOEL	313
10	rabbit	oral, 20 doses in 28 days	28-d NOEL	313
10	rats, both sexes	oral	98-d NOEL	313
10	cows	dietary level; milk and cream residue levels	3-wk <0.05 mg/kg	464
30	cows	dietary level; milk and	3-wk	464

		cream residue levels	<0.05 mg/kg	
30	rat	oral,18 doses in 24 days	24-d NOEL	313
30	rat	oral, daily,	17-wk NOEL	478
30	rats, both sexes	oral	98-d EC	313
100	rat	oral,18 doses in 24 days	24-d NOEL	313
100	rabbit	oral, 20 doses in 28 days	28-d EC	313
100	rats, both sexes	oral	98-d EC	313
300	rat	oral,18 doses in 24 days	24-d NOEL	313
355	male rat	intraperitoneal	LD_{50}	229
500	rabbit	oral, 20 doses in 28 days	28-d EC	313
820	rat	oral in fuel oil	LD_{50}	652
1000	cows	dietary level; 3 week; milk residues	0.24 mg/kg	464
1000	cows	dietary level; 3 week; cream residues	0.19 mg/kg	464
1000	rat	oral,18 doses in 24 days	24-d EC	313
1000	rat	oral, daily,	17-wk EC	478
2260	rat	subcutaneous in fuel oil	LD_{50}	652
2520	male rat	oral	LD40	313
2800	rat	oral	LD_{50}	476

2960	rat	oral	LD_{50}	313
2960	male rat	oral	LD_{50}	313
3000	rat	oral	LD_{50}	464
3160	male rat	oral	LD40	313
3980	male rat	oral	LD100	313
4000	rat	oral	LD_{50}	475
		2,4,6-TCP		
276	male rat	intraperitoneal	LD_{50}	229
		3,4,5-TCP		
372	male rat	intraperitoneal	LD_{50}	229
372	male rat	intraperitoneal	LD_{50}	418

Table 7.5.2 (continued)

mg/kg	Species	Conditions	Effect	Ref.
		TCPs		
250	rats		LD_{50}	315
250	rats		LD_{50}	232
		2,3,4,5-TTCP		
97	female mice	intraperitoneal in 40% ethanol	LD_{50}	450
133	female mice	intraperitoneal in prop. glycol	LD_{50}	450
400	female mice	oral in 40% ethanol	LD_{50}	450
499	rats	intraperitoneal	EC	520
533	female gerbils	oral in propylene glycol	LD_{50}	450
572	male mice	oral in 40% ethanol	LD_{50}	450
677	female	oral in propylene	LD_{50}	450

	mice	glycol		
1507	rats	intraperitoneal	EC	520
2000	rats, both sexes	dermal in ethanol to 8 cm2 area	<14-d LD_{50}	106
		2,3,4,6-TTCP		
10	male rats	oral in olive oil	55-d NOEL	324
50	male rats	oral in olive oil	55-d NOEL	324
82	female mice	intraperitoneal in prop. glycol	LD_{50}	450
100	male rats	oral in olive oil	55-d EC	324
121	female mice	intraperitoneal in 40% ethanol	LD_{50}	450
130	male rats	intraperitoneal in olive oil	LD_{50}	229
131	female mice	oral in 40% ethanol	LD_{50}	450
163	male mice	oral in 40% ethanol	LD_{50}	450
300	male rats	oral in olive oil	24-h NOEL	324
360	male rats	oral in olive oil	24-h NOEL	324
410	male rats	oral in olive oil	24-h NOEL	324
432	male rats	oral in olive oil	24-h EC	324
518	male rats	oral in olive oil	24-h EC	324
632	male rats	oral in olive oil	24-h EC	324
698	female gerbils	oral in propylene glycol	LD_{50}	450
735	female	oral in propylene	LD_{50}	450

	mice	glycol		
		2,3,5,6-TTCP		
48	female mice	intraperitoneal in 40% ethanol	LD_{50}	450
89	male mice	oral in 40% ethanol	LD_{50}	450
109	female mice	oral in 40% ethanol	LD_{50}	450
109	female mice	intraperitoneal in prop. glycol	LD_{50}	450
543	female mice	oral in propylene glycol	LD_{50}	450
979	female gerbils	oral in propylene glycol	LD_{50}	450

Table 7.7 Chlorophenol limits for Humans as Recommended in the Literature.

chlorophenol	food µg/kg	man µg/kg/day	conditions	ref.
TCPs		0.5	adult limit	227
2,4,5-TCP		7.0	adult limit	226
2,4,6-TCP		0.89	adult limit	227
2,3,4,5,6-PCP		3.0	adult limit	224
2,3,4,5,6-PCP		30.0	adult limit	205
2,3,4,5,6-PCP	3.0		human consumption, USA	224

2,3,4,5,6-PCP	10.0-30.0		food-plant residues, Germany	224

Table 8.1.2 The Effects of Chlorophenols on Marine Algae.

mg/L	chlorophenol	species/conditions	effect	ref.
		Champia parvula		
0.28	2,3,4,5,6-PCP	23C, culture, reduced reproduction	2-d EC_{50}	2
0.465	2,3,4,5,6-PCP	23C, culture, reduced reproduction	2-d EC_{95}	2
		Macrocystis pyrifera kelp		
1.00	2,3,4,5,6-PCP	culture, photosynthesis inhibition	2-d EC_{100}	174, 218
2.66	2,3,4,5,6-PCP	culture, photosynthesis inhibition	4-d EC_{100}	174, 218
0.3	NaPCP	lab., flow through, photosynthesis inhibition	4-d EC_{50}	331
		Skeletonema costatum diatom		
0.011	2,3,4,5,6-PCP	culture density, pH 8.1, 19-22 C	5-d NOEL	755
0.020	2,3,4,5,6-PCP	culture density, pH 8.1, 19-22 C	5-d LOEL	755
2.5	3-MCP	growth inhibition	20%	369
5.0	3-MCP	growth inhibition	82%	369
3.3	4-MCP	growth inhibition	96-h EC_{50}	369, 372

3.6	4-MCP	cell number decrease	96-h EC_{50}	369, 372
1.0	2,3,4,5,6-PCP	C14 uptake rate	reduced	562
		Dunsbella tertiolecta green		
10-15	3-MCP	noticeable effects on growth	small	369
		Porphridium sp. red		
10-15	3-MCP	noticeable effects on growth	small	369
		marine plankton assemblages		
0.3	4-MCP	growth inhibition, species composition shifts	EC	354
1.0	4-MCP	growth inhibition, species composition shifts	EC	354
0.5	2,3,4,5,6-PCP	C14 uptake rates in estuarine species	reduced	562
0.2	2,3,4,5,6-PCP	C14 uptake in ***Isochrysis galbana***	reduced	562
0.5	2,3,4,5,6-PCP	C14 uptake in ***Thalassiosira pseudonana***	reduced	562
1.0	2,3,4,5,6-PCP	C14 uptake in ***Glenodinium hallii***	reduced	562

Table 8.1.3.1 The Effects of Chlorophenols on Marine Molluscs.

mg/L	chlorophenol	species and conditions	effect	ref.
		Mya arenaria clam		
37.0	4-MCP	static bioassay at pH 8.0	96-h LC_{100}	386
9.8	3,5-DCP	lab. study	35-h LC_{50}	386
2.4	2,4,5-TCP	lab. static bioassay at 10C	96-h LC_{100}	386
3.9	2,4,6-TCP	lab. static bioassay at 10C	96-h LC_{100}	386
11.8	2,3,4,6-TTCP	lab. static bioassay at 10C, 20 g clams	96-h LC_{100}	386
		Mytilus edulis bay mussel		
0.1	NaPCP	lab. static bioassay with 'Santobrite'	NOEL	164
0.2	NaPCP	lab., salinity 28 ppt, abnormal embryology	12%	680
0.2	NaPCP	lab., salinity 24 ppt, abnormal embryology	21%	680
0.3	NaPCP	lab., salinity 28 ppt, abnormal embryology	17.6%	680
0.3	NaPCP	lab., salinity 24 ppt, abnormal embryology	33.6%	680
0.4	NaPCP	lab., salinity 28 ppt, abnormal embryology	22.1%	680
0.4	NaPCP	lab., salinity 24 ppt, abnormal embryology	69.1%	680
1.0	NaPCP	lab. static bioassay with 'Santobrite'	3-d LC_{100}	164
1.0	NaPCP	attachment and growth	1-d EC_{100}	164

1.0	NaPCP	survival	4-d LC_{100}	164
		Crassostrea gigas pacific oyster		
0.007	2,3,4,5,6-PCP	larval development	EC	257
0.027	NaPCP	culture, abnormal embryo growth rate	48-h $EC_{4.3}$	120
0.048	NaPCP	abnormal growth and development	48-h EC_{50}	120
0.069	NaPCP	culture, abnormal embryo growth rate	48-h EC_{72}	120
0.11	NaPCP	culture, abnormal embryo growth rate	48-h EC_{100}	120
		Crassostrea virginica eastern oyster		
0.0018	2,3,4,5,6-PCP	estuarine benthos population size	NOEL	117
0.0158	2,3,4,5,6-PCP	estuarine benthos population size	reduced	117
0.04	2,3,4,5,6-PCP	static bioassay	96-h LC_{50}	205
0.077	2,3,4,5,6-PCP	flow through bioassay	96-h LC_{50}	205
0.04	NaPCP	abnormal growth	48-h EC_{50}	118
0.04	NaPCP	embryo survival and growth	96-h LC_{50}	78
0.04	NaPCP	static lab. embryo growth bioassay at 25C	48-h EC_{50}	555
0.077	NaPCP	abnormal growth	192-h EC_{50}	119

		Bursatella leachi		
0.007	2,3,4,5,6-PCP	larval devel. at 26C and 17 ppt salinity	9-w LC_{100}	202
		planktonic larvae		
0.076	2,3,4,5,6-PCP	devel. /matur., 26C and 17 ppt salinity	9-w EC	202

Table 8.1.3.2 The Effects of Chlorophenols on Marine Worms.

mg/L	chlorophenol	species/conditions	effect	ref.
		polychaetes		
		Neanthes succinea		
0.076	2,3,4,5,6-PCP	planktonic larval development and survival at 26C and 17 ppt salinity	reduced	282
		Neanthes arenaceodentata		
0.435	NaPCP	static bioassay	3-d LC_{100}	205
		Ophryotrocha diadema		
0.6	2,3,4,5,6-PCP	2 to 3 day old larvae, 21C, pH 8.1	96-h LC_{50}	93
0.9	2,3,4,5,6-PCP	2 to 3 day old larvae, 21C, pH 8.1	72-h LC_{50}	93
1.1	2,3,4,5,6-PCP	2 to 3 day old larvae, 21C, pH 8.1	48-h LC_{50}	93
1.2	2,3,4,5,6-PCP	adults, 21C, pH 8.1	72-h LC_{50}	93
1.2	2,3,4,5,6-	adults, 21C, pH 8.1	96-h	93

	PCP		LC_{50}	
1.5	2,3,4,5,6-PCP	adults, 21C, pH 8.1	48-h LC_{50}	93
1.5	2,3,4,5,6-PCP	2 to 3 day old larvae, 21C, pH 8.1	24-h LC_{50}	93
2.4	2,3,4,5,6-PCP	adults, 21C, pH 8.1	24 -h LC_{50}	93
		miscellaneous nematodes		
0.0018	2,3,4,5,6-PCP	biomass and density	NOEL	95
0.007	2,3,4,5,6-PCP	biomass and density	NOEL	95
0.0158	2,3,4,5,6-PCP	biomass and density	NOEL	95
0.161	2,3,4,5,6-PCP	biomass and density	reduced	95
0.622	2,3,4,5,6-PCP	biomass and density	reduced	95

Table 8.1.3.3 The Effects of Chlorophenols on Marine Crustaceans.

mg/L	chlorophenol	species and conditions	effect	ref.
		Mesidotea entomon isopod		
37.5	4-MCP	brackish water, pH 7.7	7-d LC_{50}	373
27.5	4-MCP	brackish water, pH 7.7	96-h LC_{50}	373
40.3	4-MCP	brackish water, pH 7.7	96-h LC_{50}	373

23.0	4-MCP	brackish water, pH 7.7	7-d LC_{50}	373
		Nitocra spinipes copepod		
21.0	4-MCP	static bioassay in brackish water at pH 7.8	96-h LC_{50}	385
0.27	2,3,4,5,6-PCP	static bioassay, 21C, pH 7.8, salinity 7 ppt	96-h LC_{50}	385
		Crangon crangon shrimp		
0.112	NaPCP	larvae, 1st. instar, 15C, pH 7.5-8.0	96-h LC_{50}	73
1.79	NaPCP	adults, 15C, pH 7.5-8.0	96-h LC_{50}	73
		Crangon septemspinosa shrimp		
5.3	2-MCP	laboratory static bioassay at 10C	96-h LC_{50}	386
4.6	4-MCP	laboratory static bioassay at pH 8.0	96-h LC_{50}	386
19.1	2,6-DCP	laboratory static bioassay at 10C	52-h LC_{50}	386
19.1	2,6-DCP	laboratory static bioassay at 10C	52-h LC_{50}	419
1.5	3,5-DCP	laboratory static bioassay	96-h LC_{50}	386
2.0	2,3,4-TCP	laboratory static bioassay, 10C, 2.4-4.5 g shrimp	96-h LC_{50}	386
2.7	2,3,6-TCP	laboratory static bioassay at 10C	96-h LC_{50}	386
11.8	2,3,4,6-TTCP	lab. replacement static bioassay, 10C, 2.4-4.5 g shrimp	96-h LC_{50}	386
3.3	2,3,4,5,6-PCP	laboratory static bioassay at 10C	96-h LC_{50}	386

0.11	NaPCP	laboratory static bioassay at 15C, larvae	96-h LC_{50}	73
1.79	NaPCP	laboratory static bioassay at 15C, adults	96-h LC_{50}	73

Table 8.1.3.3 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
0.1	4-MCP	***Pontoporeia affinis***, amphipod, brackish water, swimming ability	10-d EC	374
4.1	4-MCP	***Chaetogammarus marinus***	96-h LC_{50}	371
10.0	4-MCP	developmental effects in copepods	EC	354
29.7	4-MCP	***Mysidopsis bahia***, static bioassay	96-h LC_{50}	372
19.1	2,6-DCP	sand shrimp	52-h LC_{50}	419
3.83	2,4,5-TCP	mysid shrimp, static bioassay	LC_{50}	372
0.076	2,3,4,5,6-PCP	***Callinectes sapidus*** crab, planktonic larval development at 26C and 17 ppt salinity	reduced	202
0.076	2,3,4,5,6-PCP	larval development and maturation at 26C and 17 ppt salinity over 9 weeks	EC	202
2.3	2,3,4,5,6-PCP	***Leander japonicus*** shrimp	48-h TLm	72
5.6	2,3,4,5,6-PCP	***Penaeus duorum***, static bioassay	96-h LC_{50}	205
0.195	NaPCP	***Penaeus aztecus*** brown shrimp laboratory continuous-flow bioassay	96-h LC_{50}	119

		Paleamonetes varians shrimp		
0.363	NaPCP	lab. static bioassay, 15C, pH 7.5-8.0, larvae	96h LC_{50}	73
5.09	NaPCP	lab. static bioassay, 15C, pH 7.5-8.0, adults	96h LC_{50}	73
		Paleamonetes elegans shrimp		
0.084	NaPCP	lab. static bioassay, 15C, pH 7.5-8.0, larvae	96h LC_{50}	73
10.39	NaPCP	lab. static bioassay, 15C, pH 7.5-8.0, adults	96h LC_{50}	73

Table 8.1.3.3 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Paleamonetes pugio grass shrimp		
0.3	2,3,4,5-TTCP	limb regrowth, replacement static bioassay	11-d EC_{59}	521
0.37	2,3,4,5-TTCP	stage D-3 premolt, 20C, pH 7.6	96-h LC_{50}	521
0.86	2,3,4,5-TTCP	stage C intermolt, 20C, pH 7.6	96-h LC_{50}	521
0.1	NaPCP	25 mm long	NOEL	68
0.436	NaPCP	lab., replacement static bioassay, molting	96-h LC_{50}	68
0.436	NaPCP	lab., replacement static bioassay, molting	96-h LC_{50}	205
0.436	NaPCP	late ecdysis	72-h LC_{50}	76
0.436	NaPCP	25 mm long	96-h LC_{50}	76

0.436	NaPCP	25 mm long	96-h LC_{50}	68
0.444	NaPCP	late ecdysis	48-h LC_{50}	76
0.473	NaPCP	limb regeneration, unfed	EC_{50}	74
0.499	NaPCP	late ecdysis	24-h LC_{50}	76
0.5	NaPCP	survival of young to 2nd. ecdysis	LC	68
>0.515	NaPCP	laboratory continuous flow bioassay	96-h LC_{50}	119
0.565	NaPCP	limb regeneration, fed	EC_{50}	74
0.649	NaPCP	larvae	96-h LC_{50}	118
0.649	NaPCP	static bioassay	92-h LC_{50}	205
0.649	NaPCP	lab. static bioassay at 25C, 24 h old larvae	96-h LC_{50}	555
1.0	NaPCP	non-gravid, 25 mm long, to 1st. ecdysis	LC	67
1.0	NaPCP	non-gravid, 25 mm long, to 1st. ecdysis	LC	68
1.5	NaPCP	oxygen uptake rate	NOEL	65
1.5-5.0	NaPCP	early ecdysis	3-h LC_{100}	65
2.6-2.7	NaPCP	intermolt	96-h LC_{50}	76
2.632	NaPCP	lab. replacement static bioassay, intermolt	96-h LC_{50}	68
2.632	NaPCP	lab. replacement static bioassay, intermolt	96-h LC_{50}	205
2.743	NaPCP	lab, replacement static	96-h	68

		bioassay, premolt	LC_{50}	
2.743	NaPCP	lab, replacement static bioassay, premolt	96-h LC_{50}	205
3.1-3.3	NaPCP	intermolt	72-h LC_{50}	76
3.5-3.6	NaPCP	intermolt	48-h LC_{50}	76
4.2-5.9	NaPCP	intermolt	24-h LC_{50}	76
5.0	NaPCP	oxygen uptake rate	NOEL	65
5.1	NaPCP	adults,15C, pH 7.5-8.0	96-h LC_{50}	73
10-20	NaPCP	oxygen uptake rates	96-h EC	65

Table 8.1.3.4 The Effects of PCP on the Marine Polychaete Worm, *Ophryotrocha diadema*.

recorded	concentration of 2,3,4,5,6-PCP in µg/L				
effect	3	10	32	100	320
48-day experiment beginning with larval worms					
% worms dead	20	20	18	40	78
% eggs dead	21	40	45	70	-
% reproduction	90	68	46	19	0
larvae/egg	9.6	7.8	6.8	3.0	-
37-day experiment beginning with adult worms					

% worms dead	3	3	3	5	58
% eggs dead	24	19	36	32	36
% reproduction	86	112	78	44	1.7
larvae/egg	7.6	9.2	6.2	7.4	7.0

From Reference 93.

Table 8.1.3.5 The Effects of Chlorophenols on Miscellaneous Marine Invertebrates.

mg/L	chlorophenol	species/conditions	effect	ref.
		***Arbacia punctata*, sea urchin**		
0.3	2,3,4,5,6-PCP	culture of early embryos	4-h EC_{50}	182
0.9	2,3,4,5,6-PCP	spermicidal at 20C	1-h EC_{50}	182
		Anemones		
0.1	NaPCP	laboratory, flow-through bioassay	NOEL	164
1.0	NaPCP	attachment and growth, lab., flow-through	1-d EC_{100}	164
1.0	NaPCP	survival, lab., flow-through bioassay	3-d LC_{100}	164
		Barnacles		
0.1	NaPCP	laboratory, flow-through	NOEL	164

		bioassay		
1.0	NaPCP	attachment and growth, lab., flow-through bioassay	1-d EC_{100}	164
1.0	NaPCP	survival, lab., flow-through bioassay	3-d LC_{100}	164
1.0	NaPCP	***Molgula*** sp. tunicate, survival	1-d LC_{100}	164
1.0	NaPCP	***Bugula*** **sp.** bryozoan, survival	1-d LC_{100}	164

Table 8.1.3.6 Acute Toxicity of Chlorophenols to the Grass Shrimp *Palaemonetes pugio*.

chlorophenol congeners	96-h LC_{50} (95% limits) in mg/L	
	intermolt shrimp	molting shrimp
2,4-DCP	2.55 (2.28-2.86)	2.16 (1.49-2.77)
2,4,5-TCP	1.12 (0.92-1.43)	0.64 (0.36-0.80)
2,4,6-TCP	3.95 (3.28-4.95)	1.21 (1.11-1.31)
2,3,4,5-TTCP	0.86 (0.73-0.98)	0.37 (0.35-0.39)
2,3,4,6-TTCP	3.70 (2.98-5.25)	0.81 (0.64-0.89)
2,3,5,6-TTCP	4.10 (3.30-5.31)	1.17 (1.08-1.27)
2,3,4,5,6-	2.50 (1.91-	0.44 (0.18-

PCP	3.29)	0.67)

Comparison of Intermolt and Molt sensitivity level
From Reference 130.

Table 8.1.4 The Effects of Chlorophenols on Marine Fish.

mg/L	chlorophenol	species and conditions	effect	ref.
		Lagodon rhomboides pinfish		
0.038	2,3,4,5,6-PCP		96-h LC_{50}	119
0.038	2,3,4,5,6-PCP	lab., static bioassay at 20C, 48-h prolarvae	96-h LC_{50}	205
0.038	2,3,4,5,6-PCP	lab., static bioassay at 20C, 48-h prolarvae	96-h LC_{50}	555
0.053	2,3,4,5,6-PCP	laboratory, flow-through bioassay	96-h LC_{50}	119
0.053	2,3,4,5,6-PCP	laboratory, flow-through bioassay	96-h LC_{50}	205
0.0532	NaPCP		96-h LC_{50}	119
0.066	Dowicide-G	* 79% PCP, lab. static bioassay on larvae	96-h LC_{50}	118
0.066	Dowicide-G	* 79% PCP, lab. static bioassay on larvae	96-h LC_{50}	555
		Oncorhynchus nerka sockeye salmon		
1.7	2,4-DCP	laboratory static bioassay at 10C	24-h LC_{50}	90
0.9	2,4,5-TCP	laboratory static bioassay at pH 8.0	24-h LC_{50}	90

1.1	2,4,6-TCP	laboratory static bioassay at 10C	24-h LC_{50}	90
0.5	2,3,4,6-TTCP	laboratory static bioassay at 10C	24-h LC_{50}	90
0.002	2,3,4,5,6-PCP	laboratory static bioassay at 15C, larvae	EC	94
0.3	2,3,4,5,6-PCP	laboratory static bioassay at 15C, adults	24-h LC_{50}	90
		Mugil cephalis mullet		
0.112	2,3,4,5,6-PCP	laboratory, continuous-flow bioassay	96-h LC_{50}	205
0.112	NaPCP	laboratory, continuous-flow bioassay	96-h LC_{50}	119

Table 8.1.4 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Cyprinodon variegatus sheepshead minnow		
5.35	4-MCP	static bioassay	96-h LC_{50}	372
5.35	4-MCP	static bioassay	96-h LC_{50}	393
1.66	2,4,5-TCP	static bioassay	96-h LC_{50}	372
1.9	2,3,5,6-TTCP	static bioassay, 31C,10-31 ppt salinity, fry	96-h LC_{50}	393
2.0	2,3,5,6-TTCP	static bioassay, 31C,10-31 ppt salinity, fry	72-h LC_{50}	393
2.0	2,3,5,6-TTCP	static bioassay, 31C,10-31 ppt salinity, fry	48-h LC_{50}	393
2.0	2,3,5,6-	static bioassay, 31C,10-31 ppt	24-h	393

	TTCP	salinity, fry	LC_{50}	
0.088	2,3,4,5,6-PCP	flow through bioassay, survival reduction	60-d LC	556
0.195	2,3,4,5,6-PCP	flow through bioassay, survival and hatching reduction	60-d LC	556
0.223	2,3,4,5,6-PCP	flow through bioassay, 30C, 42-day old fry	96-h LC_{50}	118
0.223	2,3,4,5,6-PCP	flow through bioassay, 30C, 42-day old fry	96-h LC_{50}	555
0.240	2,3,4,5,6-PCP	flow through bioassay, 30C, 28-day old fry	96-h LC_{50}	118
0.240	2,3,4,5,6-PCP	flow through bioassay, 30C, 28-day old fry	96-h LC_{50}	555
0.329	2,3,4,5,6-PCP	flow through bioassay, 30C, 1-day old fry	96-h LC_{50}	118
0.329	2,3,4,5,6-PCP	flow through bioassay, 30C, 1-day old fry	96-h LC_{50}	555
0.389	2,3,4,5,6-PCP	flow through bioassay, survival	60-d LC_{100}	556
0.392	2,3,4,5,6-PCP	flow through bioassay, 30C, 14-day old fry	96-h LC_{50}	118
0.392	2,3,4,5,6-PCP	flow through bioassay, 30C, 14-day old fry	96-h LC_{50}	555
0.44	2,3,4,5,6-PCP	laboratory flow through bioassay	96-h LC_{50}	556
0.44	2,3,4,5,6-PCP	laboratory low through bioassay	96-h LC_{50}	678
0.442	2,3,4,5,6-PCP	flow through bioassay, 30C, fry, 1-2 cm long	96-h LC_{50}	556
		Anguilla anguilla eel		
0.1	2,3,4,5,6-PCP	seawater, pH 8.1, technical grade, hypermetabolic	8-d EC	667

0.1	2,3,4,5,6-PCP	fresh water, pH 7.1, technical grade, static bioassay	4-d EC	667
		Fundulus similis killifish		
>0.306	NaPCP	laboratory, flow through bioassay	96-h LC_{50}	119

Table 8.2.2 The Effects of Chlorophenols on Freshwater Algae.

mg/L	chlorophenol	species and conditions	effect	ref.
		Chlorella pyrenoidosa		
200	Phenol	growth	NOEL	3
500	Phenol	growth	EC_{100}	3
10.0	2-MCP	static bioassay, 25C, toxicity test	NOEL	284
96	2-MCP	static bioassay, pH 7.0	72-h EC_{50}	284
100	2-MCP	25C, pH 7.3, static, photosyn. reduct.	3-d 88%	284
500	2-MCP	25C, pH 7.3, static, photosyn. reduct.	3-d 74%	284
10	3-MCP	25C, 5% CO2, constant light, 1 g/L of cells, photosyn. reduct./O2 evolution.	3-d NOEL	284
40	3-MCP	static bioassay, pH 7.0	72-h EC_{50}	284
100	3-MCP	25C, 5% CO2, constant light, 1 g/L cells, photosyn. reduct./O2 evolution	3-d 82%	284
500	3-MCP	25C, 5% CO2, constant	EC_{100}	284

		light, 1 g/L cells, photosyn. reduct./O2 evolution		
40	4-MCP	static bioassay, pH 7.0	72-h EC_{50}	284
100	4-MCP	25C, 5% CO2, constant light, 1 g/L cells, photosyn. reduct./O2 evolution	3-d 84%	284
500	4-MCP	25C, 5% CO2, constant light, 1 g/L cells, photosyn. reduct./O2 evolution	3-d 27.4%	284
8.0	2,4-DCP	14-day static bioassay at 18C, growth	NOEL	3
10.0	2,4-DCP	lab. 14-day static bioassay at 18C, growth response	14-d EC_{100}	3
21.0	2,4-DCP	static bioassay, pH 7.0, chloro. reduct.	72-h EC_{50}	284
50	2,4-DCP	oxygen production reduced to	56.4%	284
50	2,4-DCP	photosynthetic production inhibition	2-h EC_{40}	501
100	2,4-DCP	oxygen production reduced to	42%	284
100	2,4-DCP	chlorophyll destruction	EC_{100}	284
163	2,4-DCP	nitrite and nitrate uptake and assimilation	EC_{100}	500
1.0	2,4,5-TCP	chlorophyll destruction	72-h NOEL	284
10.0	2,4,5-TCP	chlorophyll destruction	72-h EC_{100}	284
0.1	2,4,6-TCP	14-day static bioassay at 18C, growth	NOEL	3
1.0	2,4,6-TCP	lab. 14-day static bioassay	14-d	3

		at 18C, growth response	EC_{100}	
1.0	2,4,6-TCP	photosyn. suppression, 25C, 5% CO2	NOEL	284
10.0	2,4,6-TCP	chlorophyll destruction	72-h EC_{50}	284
100	2,4,6-TCP	photosyn. suppression, 25C, 5% CO2	72-h EC_{50}	284
0.0075	2,3,4,5,6-PCP	photosynthesis effects, 25C, pH 7.0	72-h EC_{100}	284
7.0	2,3,4,5,6-PCP	growth response, hard water, pH 8.0	96-h EC_{50}	85

Table 8.2.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Chlorella variegatum		
2.0	NaPCP	lab., static bioassay, 75% PCP, toxicity	72-h EC0	330
		Chlorella vulgaris		
170	2-MCP		96-h EC_{50}	399
29.0	4-MCP		96-h EC_{50}	399
3.6	2,4,5-TCP	toxicity	7-d EC_{50}	237
10.0	2,4,6-TCP		96-h EC_{50}	399
		Scenedesmus obliquus		
1.8	NaPCP	laboratory static bioassay for toxicity	7-d EC	330
2.0	NaPCP	laboratory static bioassay for toxicity	3-d EC	330
		Freshwater Periphyton		

		Communities		
0.048	2,3,4,5,6-PCP	instream growth and species ratios	EC	116
15	NaPCP	laboratory, growth effects over time	EC	163
15	NaPCP	pond, growth effects	7-d EC	163
20	NaPCP	laboratory, growth effects immediately	EC	163
		Ankistrodesmus braunii		
6-7	NaPCP	laboratory culture	48-h LC_{65}	8
		Green Algae		
500	2-MCP		EC	220
50	2,4-MCP		EC	220
10	2,3,5-TCP		EC	220
2.66	2,3,5,6-TTCP		EC	220
0.0075	2,3,4,5,6-PCP		EC	220
		Gomphonema parvulum diatom		
1.8	2,3,4,5,6-PCP		7-d EC	330
2.0	NaPCP	laboratory static bioassay, 75% PCP, toxicity test	3-d NOEL	330
		Nitzschia palea diatom		
1.8	2,3,4,5,6-PCP		3-d EC	330
2.0	NaPCP	laboratory static bioassay, 75% PCP, toxicity test	3-d EC	330
		Cyleniospermum		

		licheniforme green		
1.8	2,3,4,5,6-PCP		3-d EC	330
2.0	NaPCP	laboratory static bioassay, 75% PCP, toxicity test	3-d EC	330

Table 8.2.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Phaeodactylum tricornutum		
0.32	4-MCP	static bioassay pH 7.8, growth effects	NOEL	371
9.6	4-MCP	static bioassay pH 7.8, growth effects	96-h LC_{50}	371
		Scenedesmus pannonicus		
3.2	4-MCP	static bioassay pH 7.8, growth effects	NOEL	371
10	4-MCP	static bioassay pH 7.8, growth effects	96-h LC_{50}	371
		Scenedesmus quadricauda		
3.6	2,4-DCP	static bioassay, pH 7.0, cell division	7-d EC	42
0.080	2,3,4,5,6-PCP	pH 8.0, hard water, growth effects	96-h EC_{50}	85
		Scenedesmus capricornutum		
0.29	2,3,4,5,6-PCP	growth effects	96-h EC_{50}	85
0.41	2,3,4,5,6-	growth effects	48-h	85

	PCP		EC_{50}	
		Chlamydomonas reinhardii		
2.66	2,3,4,5,6-PCP	inhibition of oxidative phosphorylation, 3 min.	50%	219
		Microcystis aeruginosa green		
2.0	2,4-DCP	static bioassay, pH 7.0, cell division	8-d EC	383
1.8	2,3,4,5,6-PCP		3-d EC	330
2.0	NaPCP	lab. static bioassay, 75% PCP, toxicity	3-d EC	330
		Anabaena flos-aquae		
0.040	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 24-25 C	5-d NOEL	758
0.077	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 24-25 C	5-d LOEL	758
		Navicula pelliculosa		
0.0078	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 25 C	5-d NOEL	756
0.018	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 25 C	5-d LOEL	756
		Ankistrodesmus falcatus		
0.001-0.0013	2,3,4,5,6-PCP	photosynthesis and growth rate, 20 C, pH not reported	11-d LOEL	764
		Melosira		
0.001-0.0013	2,3,4,5,6-PCP	photosynthesis and growth rate, 20 C, pH not reported	8-d LOEL	764
		Microcystis		
0.001-	2,3,4,5,6-	photosynthesis and growth	10-d	764

0.0013	PCP	rate, 20 C, pH not reported	LOEL	

Table 8.2.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Selanastrum capricornutum		
70.0	2-MCP		96-h EC_{50}	399
29.0	3-MCP		96-h EC_{50}	399
4.79	4-MCP	cell production and growth	96-h EC_{50}	372
38.0	4-MCP		96-h EC_{50}	399
0.7	3,5-DCP	phosphate uptake rate	72-h EC_{10}	699
1.9	3,5-DCP	growth rate	72-h EC_{10}	699
2.9	3,5-DCP	phosphate uptake rate	72-h EC_{50}	699
6.3	3,5-DCP	growth rate	72-h EC_{50}	699
2.0	2,3,4-TCP		96-h EC_{50}	399
1.22	2,4,5-TCP	chlorophyll A production	96-h EC_{50}	372
3.5	2,4,6-TCP		96-h EC_{50}	399
2.66	2,3,5,6-TTCP	cell production rate	96-h EC_{50}	451
2.72	2,3,5,6-	chlorophyll A effects	96-h	451

	TTCP		EC_{50}	
0.012	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 24-25 C	5-d NOEL	757
0.017	2,3,4,5,6-PCP	cell growth, density, pH 7.5, 24-25 C	5-d LOEL	757

Table 8.2.5.1 The Effects of Chlorophenols on Aquatic Plants.

mg/L	chlorophenol	species and conditions	effect	ref.
		Elodea canadensis canadian waterweed		
0.23	2,3,4,5,6-PCP	growth reduction	21-d NOEL	113
0.38	2,3,4,5,6-PCP	growth reduction	21-d EC	113
0.81	2,3,4,5,6-PCP	growth reduction	14-d EC	113
1.44	2,3,4,5,6-PCP	growth reduction	7-d EC	113
		Eichhornia crassipes water hyacinth		
4.6	2,3,4,5,6-PCP	minimum dose to cause any effect	EC	332
73.9	2,3,4,5,6-PCP	lethal dose	LC_{100}	332
5.0	NaPCP	field static bioassay with 'Santobrite'	EC	332
80.0	NaPCP	field static bioassay with 'Santobrite'	LC_{100}	332

		Lemna perpusilla duckweed		
5.0	2,4-DCP	static culture, 27C, abnormal growth	14-d EC	3
5-15	2,4-DCP	static culture, 27C, abnormal growth	14-d EC	3
20	2,4-DCP	static culture, 27C, growth effects	14-d EC_{100}	3
0.1	2,4,6-TTCP	static culture, 27C, growth effects	14-d NOEL	3
5.0	2,4,6-TTCP	static culture, 27C, growth effects	14-d EC_{100}	3
15	2,3,4,5,6-PCP	serious effects on aquatic plants	none	162
		Lemna sp. duckweeds		
4.79	4-MCP		EC	220
5.92	2,3,6-TCP		EC	220
0.603	2,3,4,6-TTCP		EC	220
		Lemna gibba duckweed		
0.032	2,3,4,5,6-PCP	frond density, biomass, pH 5.0, 25C	14-d NOEL	759
0.072	2,3,4,5,6-PCP	frond density, biomass, pH 5.0, 25C	14-d LOEL	759
		Lemna minor duckweed		
283	4-MCP	static bioassay, pH 5.1, 25C, chlorosis	48-h EC_{50}	372
283	4-MCP	static bioassay, pH 5.1, 25C, chlorosis	48-h EC_{50}	381
58.3	2,4-DCP	static bioassay, pH 5.1, 25C, chlorosis	48-h EC_{50}	381
283	2,4-DCP	static bioassay, pH 5.1, 25C,	EC_{50}	381

		chlorosis		
1.66	2,4,5-TCP	static bioassay, pH 5.1, 25C, chlorosis	72-h EC_{50}	372
1.66	2,4,5-TCP	static bioassay, pH 5.1, 25C, chlorosis	72-h EC_{50}	381
5.92	2,4,6-TCP	static bioassay, pH 5.1, 25C, chlorosis	72-h EC_{50}	372
5.92	2,4,6-TCP	static bioassay, pH 5.1, 25C, chlorosis	72-h EC_{50}	381
0.603	2,3,4,6-TTCP	static bioassay, pH 5.1, 25C, chlorosis	72-h EC_{50}	381
1.4	2,3,4,6-TTCP	static bioassay, pH 5.1, 25C, chlorosis	48-h EC_{50}	381
0.189	2,3,4,5,6-PCP	static bioassay, pH 5.1, 25C, chlorosis	48-h EC_{50}	381
1.44	2,3,4,5,6-PCP	growth reduction	21-d EC	113

Table 8.2.5.2 PCP Concentration vs. Effects on *Lemna minor*, duckweed.

	PCP concentration in mg/L						
measured variable	0	1	2	3	4	5	6
oxygen evolution	100	75	65	55	25	5	2
chlorophyll content	100	85	75	55	30	20	15
glutamic dehydrogenase activity	100	85	75	50	30	20	10
alanine aminotransferase activity	100	-	-	-	-	-	10

Table entries are the % of the level or activity found in the control plants after a
60-hour exposure in culture. From reference 4.

Table 8.2.6.1 The Effects of Chlorophenols on Aquatic Insects.

mg/L	chlorophenol	species and conditions	effect	ref.
		Tanytarsus dissimilis chironomid		
>13.5	2,4,6-TCP		48-h LC_{50}	245
19.0	2,3,4,5,6-PCP	18.9C, pH 8.6, 3 rd to 4 th instar	96-h LC_{50}	114
31.3	2,3,4,5,6-PCP	18.5C, pH 8.5, 3 rd to 4 th instar	96-h LC_{50}	114
46	2,3,4,5,6-PCP	flow through bioassay	48-h EC_{50}	550
		Chironomus riparius midge		
0.384	2,3,4,5,6-PCP	25C, pH 4.0, range 295-435	24-h EC_{50}	6
0.465	2,3,4,5,6-PCP	25C, pH 6.0, range 446-496	24-h EC_{50}	6
1.984	2,3,4,5,6-PCP	25C, pH 9.0, range 1385-2691	24-h EC_{50}	6
631	2,3,4,5,6-PCP	35C, range 581-674	LC_{50}	5
1176	2,3,4,5,6-PCP	15C, range 816-1349	LC_{50}	5
1556	2,3,4,5,6-PCP	25C, range 1500-1631	LC_{50}	5
		Aedes aegypti mosquito		

1.8	2,3,4,5,6-PCP	26C, 3rd instar	NOEL	112
7.2	2,3,4,5,6-PCP	26C, 3rd instar	48-h LC_{50}	112
0.11	2,3,4,5,6-PCP	20C, ***Chironomus thummi*** midge	48-h LC_{50}	197
		Culex pipiens mosquito		
24	2,3,4,5,6-PCP	26C, 3rd instar	NOEL	112
34	2,3,4,5,6-PCP	26C, 3rd instar	48-h LC_{50}	112
		Callibacetes skokianus mayfly		
1.3	2,3,4,5,6-PCP	25.3C, pH 7.8	96-h LC_{50}	113
1.70	2,3,4,5,6-PCP	7.3-17.4C, pH 7.5-8.2, river water	96-h LC_{50}	113
1.78	2,3,4,5,6-PCP	13C, pH 7.6	96-h LC_{50}	206
		Philarctus quaeris caddis fly		
1.20	2,3,4,5,6-PCP	7.3-17.4C, pH 7.5-8.2, river water	96-h LC_{50}	206
1.26	2,3,4,5,6-PCP	13C, pH 7.6	96-h LC_{50}	113
5.9	2,3,4,5,6-PCP	***Claeon dipterum*** mayfly, 20C	48-h LC_{50}	197
16.0	2,3,4,5,6-PCP	***Claeon dipterum*** mayfly	48-h LC_{50}	223
0.38	2,3,4,5,6-PCP	***Nemoura cinerea*** stonefly, 20C	48-h LC_{50}	197
5.0	NaPCP	***Ischnura*** sp., nymph, 16C, pH 7.6	1-h LC_0	69

42.0	2,3,4,5,6-PCP	***Ischnura elegans*** damselfly, 20C	48-h LC_{50}	197
11.0	2,3,4,5,6-PCP	***Corixa punctata*** water boatman	48-h LC_{50}	197
15.0	NaPCP	aquatic insects but not all arthropods	LC	162
4.6-5.0	NaPCP	Chironomidae pH 7.6, 16C	1-h LC100	69
5.0	NaPCP	***Epicordulia*** sp., dragonfly nymphs, pH 7.6, 16C	1-h LC_0	69

Table 8.2.6.2 The Effects of Chlorophenols on Freshwater Molluscs.

mg/L	chlorophenol	species and conditions	effect	ref.
		Lymnaea acuminata snail		
0.16	2,3,4,5,6-PCP	18C, pH 7.9, hard. 210 mg/L as CaCO3	96-h LC_{50}	97
0.18	2,3,4,5,6-PCP	18C, pH 7.9, hard. 210 mg/L as CaCO3	96-h LC_{50}	97
		Limnaeid snails		
10	2,4-DCP		48-h LC_{100}	388
10	2,4,5-TCP		24-h LC_{100}	372
10	2,4,5-TCP		24-h LC_{100}	388
5	2,4,6-TCP		24-h LC_{100}	372
5	2,4,6-TCP		24-h	388

			LC_{100}	
1.51	2,3,4,6-TTCP		24-h LC_{100}	388
1.67	2,3,4,6-TTCP	static lab. bioassay, Dowicide F-88% PCP	24-h LC_{100}	172
2.5	NaPCP	static lab. bioassay, Dowicide F-88% PCP	24-h LC_{100}	172
		Lymnaea stagnalis snail		
0.05	2,3,4,5,6-PCP	viable eggs per young, pH 8.0, hard water.	16-d NOEL	85
0.09	2,3,4,5,6-PCP	chronic effects, pH 8.0, hard water	16-d EC	85
0.13	2,3,4,5,6-PCP	viable eggs per young, pH 8.0, hard water.	16-d EC_{50}	85
0.18	2,3,4,5,6-PCP	eggs per young, pH 8.0, hard water	16-d LC_{50}	85
0.2	2,3,4,5,6-PCP	20C	NOEL	112
0.24	2,3,4,5,6-PCP	eggs per young, pH 8.0, hard water	96-h LC_{50}	85
0.30	2,3,4,5,6-PCP	eggs per young, pH 8.0, hard water	48-h LC_{50}	85
0.56	2,3,4,5,6-PCP	20C	48-h LC_{50}	112
0.56	2,3,4,5,6-PCP	20C,	48-h LC_{50}	196
		Australorbis glabratus		
0.05	2,3,4,5,6-PCP	egg production and viability	7-d EC	215
0.1	2,3,4,5,6-PCP	egg production and viability	7-d EC	215
0.7-	2,3,4,5,6-	adults	24-h	215

1.06	PCP		LC_{50}	
1.0	2,3,4,5,6-PCP		48-h LC_{94}	627
2.5	2,3,4,5,6-PCP		48-h LC_{100}	627
2.0	NaPCP	static laboratory bioassay	6-h LC_{50}	626
9.5	NaPCP	field study	6-h LC_{95}	173
12.0	NaPCP	static laboratory bioassay	6-h LC_{95}	626

Table 8.2.6.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Pseudosuccinea columella Fossaria cubensis		
10	2-MCP	100% strength	24-h LC_{100}	388
10	4-MCP	Na salt, 25% strength	24-h LC_{100}	388
10	2,4-DCP	Technical grade	24-h LC_{100}	388
10	2,4-DCP	Na salt, 25% strength	24-h LC_{100}	388
2.5	2,4,5-TCP	Na salt, 85% strength	24-h LC_{100}	388
10	2,4,5-TCP	Technical grade	24-h LC_{100}	388
5.0	2,4,6-TCP	Technical grade	24-h LC_{100}	388
0.83	2,3,4,5,6-PCP	8% strength	24-h LC_{100}	388
1.0	2,3,4,5,6-PCP	Technical grade	24-h LC_{100}	388

1.25	2,3,4,5,6-PCP	20% strength	24-h LC_{100}	388
2.3	2,3,4,5,6-PCP		24-h LC_{100}	172
		Gillia altilis snail		
0.3	2,3,4,5,6-PCP	21C, pH 6.7, static bioassay	96-h LC_{50}	98
0.8	2,3,4,5,6-PCP	21C, pH 6.7, continuous-flow bioassay	96-h LC_{50}	98
		Viviparus bengalensis		
1.561	2,3,4,5,6-PCP	sublethal effects on mantle cells	EC	96
0.99	NaPCP	sublethal effects on mantle cells	EC	96
		Physa gyrina snail		
0.0257	2,3,4,5,6-PCP	reduced egg production	EC	113
0.026	2,3,4,5,6-PCP	reduced egg production	EC	104
0.026	2,3,4,5,6-PCP	reduced egg production	EC	206
0.104	2,3,4,5,6-PCP	growth reduction	EC	104
0.104	2,3,4,5,6-PCP	growth reduction	EC	206
0.111	2,3,4,5,6-PCP	egg number and survival, 29.6C, pH 7.2-8.9, hardness 126-168	NOEL	102
0.365	2,3,4,5,6-PCP	egg number and survival, 29.6C, pH 7.2-8.9, hardness 126-168	EC	102
0.475	2,3,4,5,6-	range 0.22-0.73	96-h	104

	PCP		LC_{50}	
0.475	2,3,4,5,6-PCP	range 0.22-0.73	96-h LC_{50}	206
0.73	2,3,4,5,6-PCP	river water, 7.3-17.4 C, pH 7.5-8.2	96-h LC_{50}	206
4.1	2,3,4,5,6-PCP		EC	113

Table 8.2.6.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
5.5	2,4,6-TCP	***Aplexia hypnorum*** snail	96-h LC_{50}	245
0.13	2,3,4,5,6-PCP	snail	EC	60
0.24	2,3,4,5,6-PCP	***Tapes philippinarum*** clam	96-h LC_{50}	223
0.32	2,3,4,5,6-PCP	***Physa*** sp., snail	48-h LC_{50}	223
0.62	2,3,4,5,6-PCP	***Indoplanorbis exustus*** red snail	48-h LC_{50}	223
0.77	2,3,4,5,6-PCP	***Semisulcospira libertina*** marsh snail	48-h LC_{50}	223
30	2,3,4,5,6-PCP	snail	LC	75

Table 8.2.6.3 The Effects of Chlorophenols on Protozoans and Coelenterates.

mg/L	chlorophenol	species and conditions	effect	ref.

		PROTOZOANS		
0.5	2,4-DCP	***Entosiphon sulcatum***, pH 6.9, cell division	72-h EC	42
1.5	2,4-DCP	***Uronema parduczi***, cell division effects, static bioassay	EC	382
5.8	2,4-DCP	***Chilomonas paramecium***, cell division	EC	382
1.4	2,3,5,6-TTCP	growth inhibition after 60 hours, # of cells	60-h EC_{50}	454
1.4	2,3,5,6-TTCP	growth inhibition after 60 hours, # of cells	60-h EC_{50}	455
		Tetrahymena pyriformis, ciliate		
67.97	2-MCP		2-d EC_{50}	244
15.0	2,4-DCP		2-d EC_{50}	244
0.68	2,4,5-TCP		24-h EC_{50}	237
119	2,3,4,5-TTCP	growth at 27C	EC_{50}	260
1.01	2,3,5,6-TTCP		2-d EC_{50}	244
0.15	2,3,4,5,6-PCP		24-h EC_{50}	237
0.72	2,3,4,5,6-PCP		48-h EC_{50}	244
		COELENTERATES		
		Hydra oligactis		
0.43	2,3,4,5,6-PCP	17C	LC0	112
0.73	2,3,4,5,6-PCP	17C	48-h LC_{50}	112

0.73	2,3,4,5,6-PCP	20C	48-h LC_{50}	197
0.032	NaPCP		21-d NOEL	333

Table 8.2.6.4 The Effects of Chlorophenols on Aquatic Worms.

mg/L	chlorophenol	species and conditions	effect	ref.
		OLIGOCHAETES		
0.259	2,3,4,5,6-PCP	***Branchiura sowerbyi - ****	96-h LC_{50}	618
0.305	2,3,4,5,6-PCP	***Limnodrilus hoffmeisteri*** - *	96-h LC_{50}	618
0.397	2,3,4,5,6-PCP	***Spirosperma ferox*** - *	96-h LC_{50}	618
0.527	2,3,4,5,6-PCP	***Quistadrilus multisetosus*** - *	96-h LC_{50}	618
0.582	2,3,4,5,6-PCP	***Stylodrilus heringianus*** - *	96-h LC_{50}	618
0.693	2,3,4,5,6-PCP	***Rhyacodrilus montana*** - *	96-h LC_{50}	618
0.906	2,3,4,5,6-PCP	***Spirosperma nikolskyi*** - *	96-h LC_{50}	618
0.970	2,3,4,5,6-PCP	***Varichaeta pacifica*** - *	96-h LC_{50}	618
0.11	NaPCP	***Nais communis*** - **	96-h LC_{50}	92
0.31	NaPCP	***Ilyodrilus frantzi*** - **	96-h LC_{50}	92
		Tubifex tubifex		

0.286	2,3,4,5,6-PCP	20C, pH 7.5	24-h LC_{50}	334
0.351	2,3,4,5,6-PCP	10C, pH 7.0, hardness 5.3	96-h LC_{50}	618
0.619	2,3,4,5,6-PCP	20C, pH 8.5	24-h LC_{50}	334
1.294	2,3,4,5,6-PCP	20C, pH 9.5	24-h LC_{50}	334
		Tubificid worms		
1.0	2,3,4,5,6-PCP	20C	48-h LC_{50}	197
0.31	NaPCP	static lab. bioassay, 20C, pH 7.5	24-h LC_{50}	334
0.67	NaPCP	static lab. bioassay, 20C, pH 8.5	24-h LC_{50}	334
1.4	NaPCP	static lab. bioassay, 20C, pH 9.5	24-h LC_{50}	334
		Liver flukes		
10.0	2,4-DCP	egg hatching at 20C	24-h $EC_{8.2}$	388
5.0	2,4,5-TCP	egg hatching at 20C	24-h EC_{100}	388
10.0	2,3,4,6-TTCP	egg hatching at 20C, Na salt	24-h $EC_{3.4}$	388
2.5	NaPCP	egg hatching at 20C	24-h EC_{100}	388
		Planaria		
13.0	2,4,5-TCP	***Dugesia japonica***	7-d EC_{50}	237
0.13	2,3,4,5,6-PCP	***Dugesia lugubris***, 20C	48-h LC_{50}	197
0.25	2,3,4,5,6-	***Erpobdella octoculata***,	48-h	197

	PCP	leech, 20C	LC_{50}	

* pH 7, 10C, hardness 5.3.
** pH 7, 10C, in the dark.

Table 8.2.6.5 The Effects of Chlorophenols on Aquatic Crustaceans.

mg/L	chlorophenol	species and conditions	effect	ref.
		CRAYFISH		
28	2,3,4,5,6-PCP	***Procambarus clarki***	48-h LC_{50}	223
		Cambarus robustus		
0.1	2,4-DCP	blood glucose level increase	48-h EC	387
1.0	2,4-DCP	blood glucose level increase	48-h EC	387
5	2,4-DCP	static bioassay	7-d LC_{100}	387
10	2,4-DCP		48-h LC_{100}	387
		Cambarus viridis (Orconectes)		
>5	2,3,4,5,6-PCP		72-h EC	69
5	NaPCP	laboratory static bioassay	LC_0	69
		Orconectes propinquus		
0.1	2,4-DCP	blood glucose level increase	48-h EC	387
1.0	2,4-DCP	blood glucose level increase	48-h EC	387
1.0	2,4-DCP	19C	10-d LC_{14}	387
5	2,4-DCP	static bioassay	7-d LC_{100}	387

10	2,4-DCP		48-h LC_{100}	387
		Orconectes immunis		
0.1	2,4-DCP	blood glucose level increase	48-h EC	387
1.0	2,4-DCP	blood glucose level increase	48-h EC	387
1.0	2,4-DCP	19C	10-d LC_{14}	387
5	2,4-DCP	static bioassay	7-d LC_{100}	387
10	2,4-DCP		48-h LC_{100}	387
>183	2,3,4,5,6-PCP	12.1C, pH 8.0	96-h LC_{50}	114
		Astacus fluviatilis		
5.4	2,3,6-TCP	lab., replace. static bioassay, 13C, pH 6.5	8-d LC_{50}	71
19	2,3,6-TCP	lab., replace. static bioassay, 13C, pH 7.5	8-d LC_{50}	71
9.0	2,3,4,5,6-PCP	lab., replace. static bioassay, 13C, pH 6.5	8-d LC_{50}	71
9.5	2,3,4,5,6-PCP	field study	NOEL	166
53	2,3,4,5,6-PCP	lab., replace. static bioassay, 13C, pH 7.5	8-d LC_{50}	71

Table 8.2.6.5 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		AMPHIPODS, COPEPODS, ISOPODS		
0.068	2,3,4,5,6-PCP	***Pseudodiaptomus coronatus***	96-h LC_{50}	78

0.27	2,3,4,5,6-PCP	***Nitocra spinipes***, 21C	96-h LC_{50}	181
0.7	2,3,4,5,6-PCP	***Gammarus pulex***, 20C	48-h LC_{50}	197
2.9	2,3,4,5,6-PCP	***Asellus aquaticus***, 20C	48-h LC_{50}	197
		Hyalella azteca		
0.1	2,3,4,5,6-PCP	survival of 1 week old animals	6-wk LC	416
0.386	2,3,4,5,6-PCP	survival of 1 week old animals	6-wk LC_{100}	416
		Hyalella knickerbockeri		
>5	2,3,4,5,6-PCP		72-h EC	69
5	NaPCP	laboratory static bioassay	1-h LC_0	69
		Asellus communis		
>5	2,3,4,5,6-PCP		72-h EC	69
5	NaPCP	laboratory static bioassay	1-h LC_0	69
		Asellus racovitzai		
2.30	2,3,4,5,6-PCP	pH 7.5-8.2, 7.3-17.4C, river water	96-h LC_{50}	206
2.37	2,3,4,5,6-PCP	pH 7.9, 8.6C	96-h LC_{50}	113
3.40	2,3,4,5,6-PCP	pH 7.8, 25.3C	96-h LC_{50}	113
4.32	2,3,4,5,6-PCP	pH 7.8, 4.2C	96-h LC_{50}	113
>7.77	2,3,4,5,6-PCP	pH 7.8, 3.2C	96-h LC_{50}	113
		Gammarus fasciatus		

0.023	2,3,4,5,6-PCP	survival of 1 week old animals	6-wk EC	416
0.1	2,3,4,5,6-PCP	blood glucose level increase	6-wk EC_{100}	416
0.386	2,3,4,5,6-PCP		6-wk LC_{100}	416
		Gammarus pseudolimnaeus		
0.092	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 6.5, 22C, range 0.065-0.132 mg/L	96-h LC_{50}	79
0.121	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 7.5, 22C, range 0.077-0.192 mg/L	96-h LC_{50}	79
0.28	2,3,4,5,6-PCP	flow-through bioassay	96-h LC_{50}	550
0.484	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 8.0, 22C, range 0.359-0.652 mg/L	96-h LC_{50}	79
0.790	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 8.5, 22C, range 0.585-1.067 mg/L	96-h LC_{50}	79
1.15	2,3,4,5,6-PCP	inhibition of biochemical reactions	96-h EC_{50}	252
1.37	2,3,4,5,6-PCP	inhibition of biochemical reactions	96-h EC_{50}	252
1.68	2,3,4,5,6-PCP	inhibition of biochemical reactions	48-h EC	252

Table 8.2.6.5 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Crangonyx pseudogracilis		

0.139	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 6.5, 22C, range 0.098-0.197 mg/L	96-h LC_{50}	79
0.22	2,3,4,5,6-PCP	pH 8.0, 24C	96-h LC_{50}	113
0.32	2,3,4,5,6-PCP	pH 8.0, 21.8C	96-h LC_{50}	113
0.465	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 7.5, 22C, range 0.323-0.668 mg/L	96-h LC_{50}	79
0.50	2,3,4,5,6-PCP	pH 8.3, 16.5C	96-h LC_{50}	113
0.929	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 8.0, 22C, range 0.723-1.194 mg/L	96-h LC_{50}	79
1.344	2,3,4,5,6-PCP	Dowicide EC7, 88% PCP, pH 8.5, 22C, range 0.692-2.608 mg/L	96-h LC_{50}	79
1.55	2,3,4,5,6-PCP	pH 7.8, 25.3C	96-h LC_{50}	113
1.89	2,3,4,5,6-PCP	pH 7.9, 8.6C	96-h LC_{50}	113
1.90	2,3,4,5,6-PCP	pH 7.5-8.2, 7.3-17.4C, river water	96-h LC_{50}	206
2.0	2,3,4,5,6-PCP	pH 7.8, 8.9C	96-h LC_{50}	113
2.77	2,3,4,5,6-PCP	pH 7.8, 6.8C	96-h LC_{50}	113
3.12	2,3,4,5,6-PCP	pH 7.8, 3.2C	96-h LC_{50}	113

Table 8.2.6.6 The Effects of Chlorophenols on Cladocerans (except *Daphnia magna*).

mg/L	chlorophenol	species and conditions	effect	ref.
		Daphnia carinata		
0.55	2,4,5,6-TCP		24-h EC_{50}	237
0.40	2,3,4,5,6-PCP		24-h EC_{50}	237
		Daphnia cucullata		
1.5	2,3,4,5,6-PCP	98% pure PCP, 19C	48-h LC_{50}	112
1.5	2,3,4,5,6-PCP	11 day old animals	48-h EC_{50}	83
		Daphnia pulex		
0.246	2,3,4,5,6-PCP	hardness 45	48-h LC_{50}	81
0.35-0.7	2,3,4,5,6-PCP	hardness 170	48-h LC_{50}	82
0.9-1.4	2,3,4,5,6-PCP	23C, pH 8.0, hard. 240, alkal. 230	48-h EC_{50}	58
1.04	2,3,4,5,6-PCP	22C, pH 7.8, hardness 120, alkalinity 110, conductivity 560	48-h LC_{50}	80
1.08	2,3,4,5,6-PCP	22C, pH 7.7, hardness 200, alkalinity 140, conductivity 340	48-h LC_{50}	80
1.14	2,3,4,5,6-PCP	22C, pH 7.8, hardness 120, alkalinity 110, conductivity 560	48-h LC_{50}	80
1.2	2,3,4,5,6-	98% pure PCP, 19C	NOEL	112

	PCP			
2.0	2,3,4,5,6-PCP	1 day old animals, 20C	48-h LC_{50}	83
2.0	2,3,4,5,6-PCP	98% pure PCP, 19C	48-h LC_{50}	112
>5	2,3,4,5,6-PCP		72-h EC	69
5	NaPCP	laboratory static bioassay	1-h LC0	69
		Moina macrocarpa		
3.0	2,3,4,5,6-PCP		3-h LC_{50}	223
		Ceriodaphnia affinis/dubia		
0.161	2,3,4,5,6-PCP	pH 7.9, reduction in # of young	EC	113
0.307	2,3,4,5,6-PCP	pH 8.0, 24C	48-h LC_{50}	113
0.347	2,3,4,5,6-PCP	pH 7.9, 25C	48-h LC_{50}	113

Table 8.2.6.6 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Ceriodaphnia reticulata		
0.0041	2,3,4,5,6-PCP	pH 7.3, reduction in # of young	EC	113
0.022	2,3,4,5,6-PCP	pH 7.3, reduction in # of young	EC	113
0.150	2,3,4,5,6-PCP	pH 7.3, 24C	48-h LC_{50}	113
0.164	2,3,4,5,6-PCP	hardness 45	48-h LC_{50}	81
0.24	2,3,4,5,6-	pH 7.8, 18C	48-h	113

	PCP		LC_{50}	
0.28	2,3,4,5,6-PCP	22C, pH 7.7, hardness 200, alkalinity 140, conductivity 340	48-h LC_{50}	80
0.31	2,3,4,5,6-PCP	22C, pH 7.8, hardness 120, alkalinity 110, conductivity 560	48-h LC_{50}	80
0.44	2,3,4,5,6-PCP	22C, pH 7.8, hardness 120, alkalinity 110, conductivity 560	48-h LC_{50}	80
0.55	2,3,4,5,6-PCP	22C, pH 7.7, hardness 200, alkalinity 140, conductivity 340	48-h LC_{50}	80
0.70	2,3,4,5,6-PCP	pH 7.9, 25C	48-h LC_{50}	113
0.8-1.2	2,3,4,5,6-PCP	lab., replacement static bioassay, 13C, pH 7.5	48-h LC_{50}	58
		DAPHNIDS, water fleas		
0.57	2,3,5,6-TTCP		48-h LC_{50}	459
2.5	2,3,5,6-TTCP		24-h LC_{50}	459
0.1	2,3,4,5,6-PCP		EC_{50}	114
3.6	2,3,4,5,6-PCP		48-h LC_{50}	323
		Simocephalus vetulus		
0.16	2,3,4,5,6-PCP	pH 7.3, 25C	48-h LC_{50}	113
0.196	2,3,4,5,6-PCP	pH 8.2, 25C	48-h LC_{50}	113
0.204	2,3,4,5,6-	pH 8.0, 24C	48-h	113

	PCP		LC_{50}	
0.250	2,3,4,5,6-PCP	pH 7.7, 25C	48-h LC_{50}	113
0.255	2,3,4,5,6-PCP	pH 8.0, 25C	48-h LC_{50}	113
0.264	2,3,4,5,6-PCP	pH 8.3-7.9	48-h LC_{50}	113
0.364	2,3,4,5,6-PCP	pH 8.3, 25C	48-h LC_{50}	113
0.670	2,3,4,5,6-PCP	pH 7.8, 18C	48-h LC_{50}	113
0.670	2,3,4,5,6-PCP	pH 7.5-8.2, 7-17C, river water	48-h LC_{50}	206

Table 8.2.6.7 The Effects of Chlorophenols on *Daphnia magna*

mg/L	Conditions	Effect	Ref.
	2-MCP		
1.0	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
1.35		7-d EC_{50}	400
2.6		96-h LC_{50}	335
2.6	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
3.73		7-d LC_{50}	400
7.4	18C, static bioassay	48-h LC_{50}	189

10.0	pH 8.0, static bioassay for immobility	NOEL	384
>22	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
23	pH 8.0, static bioassay for immobility	24-h EC_{50}	384
	4-MCP		
0.64		7-d EC_{50}	400
1.0	mobility bioassay	14-d NOEL	371
1.1	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
4.06	static bioassay	48-h EC_{50}	372
4.06	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
2.31		7-d EC_{50}	400
4.82	18C, static bioassay	48-h EC_{50}	189
4.82	18C, static bioassay	48-h EC_{50}	372
8.8	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
8.9		48-h LC_{50}	371
10.0	mobility bioassay	LD50	371
	2,4-DCP		
0.46	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
2.3	static bioassay	21-d LC_{50}	354

2.6	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
2.61	18C, static bioassay	48-h LC_{50}	189
2.8	pH 8, static immobility bioassay	24-h EC_0	384
3.9	pH 8, static immobility bioassay	24-h EC_{50}	384
5.1	static bioassay	48-h LC_{50}	354
>10	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
	2,4,5-TCP		
0.78	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
0.89		7-d EC_{50}	400
2.66	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
3.6		7-d EC_{50}	400
3.8	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56

Table 8.2.6.7 (continued)

mg/L	Conditions	Effect	Ref.
	2,4,6-TCP		
<0.41	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
0.69		2-d EC_{50}	400
3.34		2-d EC_{50}	400
6.04	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56

15	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
	2,3,4,6-TTCP		
0.01	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
0.29	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
0.05	74% tech. grade, 17C, pH 7.5, hardness 44 mg/L as CaCO3	28-d NOEL	236
>1.0	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
	2,3,5,6-TTCP		
0.01	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
0.57	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
2.5	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
	PCP		
0.047	survival	3-wk LC_0	416
0.047	young per female, 3 weeks exposure	102	416
0.1		LC_{50}	182
0.100	reproduction, life cycle, pH 8.0, 20 C	NOEL	762
0.139	survival	3-wk LC_{20}	416
0.139	young per female	91	416
0.143	hardness 45 mg/L	48-h LC_{50}	111
0.145	pH 8.0, 22.5C	LC_{50}	114

0.16	98% PCP, 19C	NOEL	112
0.17	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.18	chronic assay	NOEL	84
0.18	chronic assay	NOEL	93
0.19	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.23		EC_{50}	253
0.24	96% PCP, 17C, pH 7.4, hardness 40 mg/L	48-h EC_{50}	111
0.24	chronic assay	EC	205
0.24	20C, hard water, 1 day old	48-h LC_{50}	83
0.24	static bioassay	48-h EC_{50}	205
0.25	pH 8.0, 20C, hard water, chronic assay	21-d EC_0	84
0.25	pH 8.0, 20C, hard water, chronic assay	21-d EC_0	205
0.26	20C,hard water,1 day old	48-h LC_{50}	83

Table 8.2.6.7 (continued)

mg/L	Conditions	Effect	Ref.
0.26	static bioassay	48-h EC_{50}	205
0.32	pH 7.4-9.4, 22C, hardness 173 mg/L *	NOEL	56
0.32	88% PCP, 17C, pH 7.4 hardness 40 mg/L, 1st. instar	48-h EC_{50}	111
0.336	survival	3-wk	416

		LC_{25}	
0.336	young per female, 3 weeks exposure	87	416
0.34	reproduction assay, 1 mm long young	21-d NOEL	217
0.37	chronic effects, pH 8.0, 20C, hard water	21-d EC_0	85
0.40	pH 8.0, hard water	7-d LC_{50}	85
0.40	pH 8.0, hard water	14-d LC_{50}	85
0.40	pH 8.0, 20 C, hard water	21-d LC_{50}	84
0.40	20C, hard water, 1 day old	48-h LC_{50}	83
0.40	static bioassay	48-h EC_{50}	205
0.4	1 mm young	14-d LC_{50}	85
0.4	1 mm young	21-d LC_{50}	85
0.41	86 % PCP, 17C, pH 7.4 hardness 40 mg/L	48-h EC_{50}	111
0.43	20C	21-d LC_{50}	84
0.43	20C	21-d LC_{50}	205
0.44	pH 8.0, 20C, hard water	14-d LC_{50}	84
0.46	pH 8.0, 20C, hard water	14-d LC_{50}	84
0.46	24C	21-d LC_{50}	84

0.46	24C	21-d LC_{50}	205
0.47	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.48	98 % PCP, 19C	48-h LC_{50}	112
0.48	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.49	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.49	15C	21-d LC_{50}	84
0.49	15C	21-d LC_{50}	205
0.49	pH 7.7, 22C, hardness 200 mg/L alkalinity 110 mg/L, conductivity 560	48-h LC_{50}	80
0.500	reproduction, life cycle, pH 8.0, 20 C	LOEL	762
0.51-0.84	hardness 170 mg/L	48-h LC_{50}	82
0.51	pH 8.0, 20C, hard water	21-d LC_{50}	84
0.58	3 mm adults	7-d LC_{50}	85
0.6	pH 8.0, hard water	96-h LC_{50}	85
0.6	1 mm young	7-d LC_{50}	85
0.6	20C, static bioassay, 1 day old, not fed	48-h LC_{50}	84
0.68	3 mm adults	96-h LC_{50}	85

Table 8.2.6.7 (continued)

mg/L	Conditions	Effect	Ref.
0.68	pH 7.4-9.4, 22C, hardness 173 mg/L *	48-h LC_{50}	56
0.79	static bioassay	48-h EC_{50}	205
0.79	20C, hard water, 1 day old	48-h LC_{50}	83
0.8	1 mm long young	48-h LC_{50}	85
0.8		21-d LC_{50}	253
0.8	20C, static bioassay, 7 days old, not fed	48-h EC_{50}	84
0.9	pH 7.8, 22C, hardness 120 mg/L alkalinity 110 mg/L, conductivity 560	48-h LC_{50}	80
1.0	young per female, 3 weeks exposure	0	416
1.0	survival	3-wk LC_{100}	416
1.0	20C, static bioassay, 1 day old, fed	48-h EC_{50}	84
1.0	pH 8.0, 23C, hardness 240 mg/L, alkalinity 230 mg/L, conductivity 360	48-h EC_{50}	58
<1.0		24-h EC	70
1.04	pH 7.8, 22C, hardness 120 mg/L, alkalinity 110 mg/L, conductivity 560	48-h LC_{50}	80

1.05	pH 8.0, hard water	48-h LC_{50}	85
1.05	1 mm long young	48-h LC_{50}	85
1.12	pH 7.7, 22C, hardness 200 mg/L, alkalinity 110 mg/L, conductivity 340	48-h LC_{50}	80
1.2	static bioassay, 20C, 1 day old, not fed	48-h EC_{50}	84
1.3	static bioassay, 20C, 7 days old, not fed	24-h EC_{50}	84
1.4	3 mm long adults	48-h LC_{50}	85
1.5	static bioassay, 20C, 7days old, fed	48-h EC_{50}	84
1.5	pH 7.4-9.4, 22C, hardness 173 mg/L *	24-h LC_{50}	56
1.7	1 mm long young	24-h LC_{50}	85
1.7	static bioassay, 20C, 1 days old, fed	24-h EC_{50}	84
2.8	static bioassay, 20C, 7 days old, fed	24-h EC_{50}	84
	NaPCP		
0.2	reproduction effects, hardness 90 mg/L	NOEL	417
0.4	adult survival, hardness 90 mg/L	NOEL	417
0.4	reproduction effects, hardness 90 mg/L	1- wk EC	417
0.6	adult survival, hardness 90 mg/L	1- wk EC	417

Reference 57 data in Table 2.4.
* dissolved oxygen is 6.5 to 9.1 mg/L.

Table 8.2.6.8 The Effects of Pentachlorophenol on the Snail *Lymnaea acuminata* .

µg/L PCP	Effect	µg/L NaPCP	Effect
249	12-h LC_{16}	330	12-h LC_{16}
213	24-h LC_{16}	250	24-h LC_{16}
170	48-h LC_{16}	145	48-h LC_{16}
148	72-h LC_{16}	120	72-h LC_{16}
104	96-h LC_{16}	110	96-h LC_{16}
293	12-h LC_{50}	470	12-h LC_{50}
263	24-h LC_{50}	360	24-h LC_{50}
228	48-h LC_{50}	255	48-h LC_{50}
146	72-h LC_{50}	215	72-h LC_{50}
160	96-h LC_{50}	190	96-h LC_{50}
345	12-h LC_{84}	630	12-h LC_{84}
311	24-h LC_{84}	475	24-h LC_{84}
284	48-h LC_{84}	370	48-h LC_{84}
245	72-h LC_{84}	315	72-h LC_{84}
214	96-h LC_{84}	270	96-h LC_{84}

18C, pH 7.9, hardness 210 mg/L, alkalinity 188 mg/L.
From reference 97.

Table 8.2.6.9 The Effects of pH and Temperature on the mg/L of PCP needed to demonstrate a 96-h LC_{50} response in the Snail *Physa gyrina.*

	temperature in C						
pH	3.2	4.2	8.6	11.7	16.5	24	25.3
7.2						0.27	
7.8	1.25	1.38		0.81			0.58
7.9			0.73				
8.0						0.26	
8.1						0.22	
8.3					0.62		

From Reference 113.

Table 8.2.6.10 Summary of the Cladoceran Data.

From Tables 2.4, 8.2.6.6, 8.2.6.7, 8.2.6.11 and 8.2.6.12.

chlorophenol congeners	lowest effect levels found-mg/L.		highest NOEL -mg/L-	references
	chronic	acute		
2-MCP	1.35	2.6	10	400, 335, 384
3-MCP	13.8			57
4-MCP	0.64	4.06	1.1	400, 56, 56
2,3-DCP	4.09			57
2,4-DCP	2.48	2.3	0.46	57, 354, 56

2,5-DCP				
2,6-DCP	8.69			57
3,4-DCP	2.55			57
3,5-DCP	1.85			57
2,3,4-TCP	2.00			57
2,3,5-TCP	2.06			57
2,3,6-TCP	6.25			57
2,4,5-TCP	0.55	2.66	0.78	237, 56, 56
2,4,6-TCP	0.69	6.04	<0.41	400, 56, 56
3,4,5-TCP	0.82			57
2,3,4,5-TTCP	1.52			57
2,3,4,6-TTCP		0.29	0.05	56, 236
2,3,5,6-TTCP	1.87	0.57	0.01	57, 56, 56
2,3,4,5,6-PCP	0.0041	0.1	1.2	113, 182, 112

Table 8.2.6.11. The Effect of temperature, duplicate testing and test duration on the LC_{50} in mg/L to Sodium Chlorophenate using *Daphnia magna*

duplicate	24-hour LC_{50} data		48-hour LC_{50} data	
test number	20C	26C	20C	26C
1	0.63	0.38	0.44	0.34

2	0.78	0.51	0.38	0.42
3	0.66	0.81	0.45	0.47
4	0.55	0.81	0.38	0.45
5	0.55		0.32	
6	0.64	0.49	0.47	0.38
7	0.85	0.84	0.67	0.59
8	0.77	0.75	0.45	0.69
9	0.70	0.85	0.34	0.61
10	0.80	0.57	0.34	0.41
Mean	0.69	0.67	0.42	0.48
SD	0.10	0.17	0.09	0.11

pH 8.0-8.5, hardness 160-180 mg/L, from Reference 697.

Table 8.2.6.12. The Effect of temperature, duplicate testing and test duration on the LC_{50} in mg/L to Sodium Chlorophenate using *Daphnia pulex*

duplicate test number	24-hour LC_{50} data		48-hour LC_{50} data	
	20C	26C	20C	26C
1	0.53		0.35	
2	0.75	0.69	0.63	0.35
3	0.70	0.60	0.55	0.53
4	0.57	0.38	0.41	0.25
5	0.53	0.49	0.40	0.31
6	0.53	0.55	0.49	0.51
7	0.68	0.53	0.49	
8	0.65	0.60	0.50	0.50

9		0.87		0.75
10	0.81	0.86	0.51	0.57
Mean	0.64	0.62	0.48	0.47
SD	0.10	0.15	0.08	0.15

pH 8.0-8.5, hardness 80-90 mg/L, from Reference 697.

Table 8.2.7 The Effects of Chlorophenols on Amphibians.

mg/L	chlorophenol	species and conditions	effect	ref.
		Amblystoma mexicana (axolotl)		
0.13	2,3,4,5,6-PCP	20C, survival	LC_0	112
0.13	2,3,4,5,6-PCP	20C, survival	LC_0	551
0.30	2,3,4,5,6-PCP	20C, survival, 3-4 weeks old, Dutch water	48-h LC_{50}	112
0.30	2,3,4,5,6-PCP	20C, survival, 3-4 weeks old, Dutch water	48-h LC_{50}	551
		Xenopus laevis (clawed toad)		
0.21	2,3,4,5,6-PCP	20C, survival	LC_0	112
0.21	2,3,4,5,6-PCP	20C, survival	LC_0	551
0.26	2,3,4,5,6-PCP	20C, survival, 3-4 weeks old, Dutch water	48-h LC_{50}	112
0.26	2,3,4,5,6-PCP	20C, survival, 3-4 weeks old, Dutch water	48-h LC_{50}	551
		Rana pipiens (frog)		

0.6	NaPCP	tadpoles	3-d LC_0	69
1.0	NaPCP	tadpoles	6.3-h LC_{100}	69
5.0	NaPCP	tadpoles	1.3-h LC_{100}	69
		Rana catesbiana (frog)		
0.207	2,3,4,5,6-PCP	pH 8.0, 17.7C	LC_{50}	114
		Tadpoles		
0.25	2,3,4,5,6-PCP		48-h LC_{50}	323

Table 8.2.8.1 The Effects of Chlorophenols on Fish...CARP.

mg/L	chlorophenol	species and conditions	effect	ref.
		Alburnus alburnus		
0.066	2,3,4,5,6-PCP		96-h LC_{50}	251
0.078	2,3,4,5,6-PCP		48-h LC_{50}	251
		Aplocheilus latipes (tooth carp)		
0.14	2,3,4,5,6-PCP	larval stages	24-h LC_{50}	546
0.17	2,3,4,5,6-PCP	caged in rice field	24-h LC_{65}	546
0.34	2,3,4,5,6-PCP	caged in rice field, 72-h test	24-h LC_{100}	546

0.85	2,3,4,5,6-PCP	caged in rice field, 72-h test	24-h LC_{100}	546
		Cyprinus carpio (common carp)		
0.01	3-MCP	eggs	NOEL	368
1.0	3-MCP	adults	NOEL	368
10	3-MCP	reduction in egg hatching	26%	368
10	3-MCP	rate of malformed embryos	42%	368
100	3-MCP	reduction in egg hatching	100%	368
0.1	2,3,4,5,6-PCP	per 100 g per day, biochemical effects	10-d EC	254
0.11	2,3,4,5,6-PCP	>17 day old fish	24-h LC_{50}	157
0.13	2,3,4,5,6-PCP	fish were 9 to 10 days old	24-h LC_{50}	157
0.14	2,3,4,5,6-PCP	fish were 1 to 3 day sold	24-h LC_{50}	157
0.15	2,3,4,5,6-PCP	fish were 5 to 6 days old	24-h LC_{50}	157
0.18	2,3,4,5,6-PCP	egg	24-h LC_{50}	157
0.1	NaPCP	2-3 cm long, 0.15 to 0.25 g, metabolic rate	increase	138
1.0	NaPCP	2-3 cm long, 0.15 to 0.25 g	130-m LC_{50}	138
1.5	NaPCP	2-3 cm long, 0.15 to 0.25 g	180-m LC_{50}	138
2.0	NaPCP	2-3 cm long, 0.15 to 0.25 g	45-m LC_{50}	138
3.0	NaPCP	2-3 cm long, 0.15 to 0.25 g	31-m LC_{50}	138

0.12	2,3,4,5,6-PCP	carp	48-h LC_{50}	323
0.4	2,3,4,5,6-PCP	range: 0.2 to 0.6, percids and cyprinids	96-h LC_{50}	126
		Rutilus rutilus (roach)		
5.0	2-MCP	21C, pH 7.3	48-h LC_0	158
0.038	2,3,4,5,6-PCP		96-h LC_{50}	951
0.15-6.0	2,3,4,5,6-PCP	3 to 12 mg fish, respiration rate effects	increased	552

Table 8.2.8.1 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Carassius auratus / C. carassius (goldfish)		
10.0	2-MCP	laboratory static bioassay at 27C	8-h LC_{20}	392
10.0	2-MCP	21C, pH 7.3	48-h LC_0	158
12.37	2-MCP	pH 7.5, 25C, hardness 20 mg/L.	96-h LC_{50}	389
12.4	2-MCP	laboratory static bioassay	96-h LC_{50}	364
16	2-MCP		24-h LC_{50}	273
31.1	2-MCP		8-h LC_{42}	392
82.8	2-MCP	laboratory static bioassay at 27C	8-h LC_{64}	392
104	2-MCP	laboratory static bioassay at 27C	8-h LC_{83}	392
142-	2-MCP	laboratory static bioassay at	8-h	392

311		27C	LC_{100}	
20.6	3-MCP	laboratory static bioassay at 27C	8-h LC_{62}	392
70.5	3-MCP	laboratory static bioassay at 27C	8-h LC_{100}	392
6.3	4-MCP		8-h LC_{54}	372
6.3	4-MCP		8-h LC_{54}	392
6.3	4-MCP	laboratory static bioassay at 27C	8-h LC_{50}	392
9.0	4-MCP		24-h LC_{50}	372
9.0	4-MCP		24-h LC_{62}	392
54.3	4-MCP	laboratory static bioassay at 27C	8-h LC_{100}	392
0.26	2,4-DCP	pH 7.8, 22C, hardness 200 mg/L, treated 4 days as embryos then 4 days as larvae	8-d LC_{50}	376
0.39	2,4-DCP	pH 7.8, 22C, hardness 50 mg/L, embryos	4-d LC_{50}	376
1.24	2,4-DCP	pH 7.8, 22C, hardness 200 mg/L, treated 4 days as embryos then 4 days as larvae	8-d LC_{50}	376
1.24	2,4-DCP	pH 7.8, 22C, hardness 200 mg/L, treated 4 days as embryos then 4 days as larvae	8-d LC_{50}	495
1.48	2,4-DCP		LC	220
1.76	2,4-DCP	pH 7.8, 22C, hardness 50 mg/L, embryos	4-d LC_{50}	376

1.76	2,4-DCP	pH 7.8, 22C, hardness 50 mg/L, embryos	4-d LC_{50}	495
7.8	2,4-DCP		24-h LC_{50}	273
7.8	2,4-DCP		24-h LC_{50}	495
1.7	2,4,5-TCP		24-h LC_{50}	273
9.0	2,4,5-TCP	static bioassay	24-h LC_{50}	372
10	2,4,6-TCP		24-h LC_{50}	273
10	2,4,6-TCP	static bioassay	24-h LC_{50}	372
0.75	2,3,4,6-TTCP	20C, replacement at 8-hour intervals, static bioassay with 2-g fish	24-h LC_{50}	273

Table 8.2.8.1 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
0.19	NaPCP	laboratory continuous-flow bioassay	14-d LC_{50}	622
0.37	NaPCP	laboratory continuous-flow bioassay	21-h LC_{50}	622
0.05	2,3,4,5,6-PCP	laboratory static bioassay at 24C in soft water	96-h LC_{50}	124
0.052	2,3,4,5,6-PCP	pH 5.5	24-h LC_{50}	547
0.06	2,3,4,5,6-PCP	lab. static bioassay at 24C in soft water	96-h LC_{50}	124
0.06	2,3,4,5,6-	lab. static bioassay at 24C in	96-h	124

	PCP	medium hardness water	LC_{50}	
0.06	2,3,4,5,6-PCP	lab. static bioassay at 24C in hard water	72-h LC_{50}	124
0.07	2,3,4,5,6-PCP	lab. static bioassay at 24C in medium hardness water	72-h LC_{50}	124
0.08	2,3,4,5,6-PCP	lab. static bioassay at 24C in soft water	72-h LC_{50}	124
0.08	2,3,4,5,6-PCP	lab. static bioassay at 24C in medium hardness water	48-h LC_{50}	124
0.087	2,3,4,5,6-PCP		48-h LC_{50}	251
0.11	2,3,4,5,6-PCP	lab. static bioassay at 24C in hard water	48-h LC_{50}	223
0.12	2,3,4,5,6-PCP		48-h LC_{50}	223
0.17	2,3,4,5,6-PCP	lab. static bioassay at 24C in soft water	48-h LC_{50}	124
0.17	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.17-0.3	2,3,4,5,6-PCP		96-h LC_{50}	205
0.175	2,3,4,5,6-PCP		14-d LD50	622
0.19	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.20	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.20	2,3,4,5,6-PCP	19.1C, pH 7.8	LC_{50}	114
0.21	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.21	2,3,4,5,6-	pH 7.6, 23C, hardness 210	11-d	122

	PCP		LC_{50}	
0.21	2,3,4,5,6-PCP	pH 7.6, 23C, hardness 210	11-d LC_{50}	282
0.22	2,3,4,5,6-PCP	pH 7.4-7.8, 25C, flow-through bioassay, technical grade PCP	96-h LC_{50}	122
0.22	2,3,4,5,6-PCP	pH 7.4-7.8, 25C, flow-through bioassay, technical grade PCP	96-h LC_{50}	282
0.22	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122

Table 8.2.8.1 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
0.23	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.24	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.25	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.27	2,3,4,5,6-PCP	laboratory flow-through bioassay	24-h LC_{50}	122
0.27	2,3,4,5,6-PCP	laboratory flow-through bioassay	24-h LC_{50}	282
0.29	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.30	2,3,4,5,6-PCP	juveniles, pH 7.2, 25C, hardness 220 mg/L as $CaCO_3$	96-h LC_{50}	122
0.328	2,3,4,5,6-PCP	17.7C, pH 7.9	LC_{50}	114
0.34	2,3,4,5,6-	24C	21-h	622

	PCP		LC_{50}	
16	2,3,4,5,6-PCP	pH 10	24-h LC_{50}	547

Table 8.2.8.2 The Effects of Chlorophenols on Fish...MISCELLANEOUS SPECIES

mg/L	chlorophenol	species and conditions	effect	ref.
		Rhodeus sericeus amarus (bitterling)		
3.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Abramus brama (bream)		
3.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Gobio gobio (gudgeon)		
20	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Phoxinus phoxinus (minnow)		
4.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Perca fluviatilis (perch)		
4.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Scanius erythrophthalmus		
7.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Gasterosteus aculeatus (stickleback)		
5.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Tinca tinca (tench)		
3.0	2-MCP	21C, pH 7.3, 48-h exposure	sublethal	158
		Catastomus commersoni (sucker)		

0.085	2,3,4,5,6-PCP	pH 7.9, 23C	96-h LC_{50}	113
		Puntius ticto		
0.0144	NaPCP	650 g fish	24-h LC_{50}	133
		Pomoxis annularis (white crappie)		
0.065	NaPCP	laboratory study, range 0.056 to 0.075	LC_{100}	166
		Misgurnus anguillicaudata (dojo, pond loach)		
0.12	2,3,4,5,6-PCP		48-h LC_{50}	223
		Esox lucius (northern pike)		
0.045	2,3,4,5,6-PCP		96-h LC_{50}	251
		Gambusia affinis (mosquitofish)		
0.278	2,3,4,5,6-PCP	0.37 g fish, pH 8.0, 18.6C	LC_{50}	114
0.378	2,3,4,5,6-PCP	0.76 g fish, pH 8.0, 17.6C	LC_{50}	114
		Tilapia mossambica (cichlid)		
0.1	NaPCP	6 to 8 cm long, 4 to 6 g fish, metabolic rate	increase	138
3.0	NaPCP	6 to 8 cm long, 4 to 6 g fish	3-h LC_{50}	138
		Gobiomaris durmitor		
2	NaPCP	static bioassay	lethal	668
15	NaPCP	continuous-flow bioassay	lethal	668

Table 8.2.8.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Awavus taiasiia		
2	NaPCP	static bioassay	lethal	668
15	NaPCP	continuous-flow bioassay	lethal	668
		Agnostomus monticola		
2	NaPCP	static bioassay	lethal	668
15	NaPCP	continuous-flow bioassay	lethal	668
		Lepomis humilis (green sunfish)		
5	2,3,4,5,6-PCP	laboratory static bioassay	EC	637
20	2,3,4,5,6-PCP	laboratory static bioassay, repellent	EC	637
		Cichlasoma bimaculatum (cichlid)		
0.2	KPCP	25C, food intake	increase	150
0.2	KPCP	25C, energy loss	increase	150
0.2	KPCP	25C, growth	decrease	150
		Rhinomugil corsula (mullet)		
0.1	NaPCP	5 to 6 cm, 0.5 to 1.5 g fish, metabolic rate	increase	138
1	NaPCP	5 to 6 cm, 0.5 to 1.5 g fish	3-h LC_{50}	138
2	NaPCP	5 to 6 cm, 0.5 to 1.5 g fish	68-m LC_{50}	138
		Coregonus peled		
0.022	2,3,4,5,6-PCP		48-h LC_{50}	251
0.043	2,3,4,5,6-PCP		96-h LC_{50}	251
0.065	2,3,4,5,6-		48-h	251

	PCP		LC_{50}	
		Brachydanio rerio (zebrafish)		
5.6	4-MCP	static bioassay	96-h LC_{50}	371
3.9	2,4-DCP	static bioassay	96-h LC_{50}	396
0.4	2,3,4,5,6-PCP	static bioassay	48-h LC_{50}	549
		Campostoma anomblum (stoneroller)		
0.2	NaPCP	laboratory static bioassay	3-d LC_0	69
1	NaPCP	laboratory static bioassay	58-m LC_0	69
5	NaPCP	laboratory static bioassay	13-m LC_0	69
		Lepomis humilis (orange spotted sunfish)		
0.1	NaPCP	laboratory static bioassay	3-d LC_0	69
1	NaPCP	laboratory static bioassay	165-m LC_0	69
5	NaPCP	laboratory static bioassay	25-m LC_0	69
		Fundulus notatus (blackstripe topminnow)		
0.6	NaPCP	laboratory static bioassay, 91% NaPCP	3-d- LC_0	69
1	NaPCP	laboratory static bioassay, 91% NaPCP	7.3-h LC_{100}	69
5	NaPCP	laboratory static bioassay, 91% NaPCP	1.5-h LC_{100}	69

Table 8.2.8.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Semotilus atromaculatus (creek chub)		
0.4	NaPCP	laboratory static bioassay, 91% NaPCP	3-d LC_0	69
1	NaPCP	laboratory static bioassay, 91% NaPCP	1.8-h LC_{100}	69
5	NaPCP	laboratory static bioassay, 91% NaPCP	30-m LC_{100}	69
		Ericymba buccata (silver jaw minnow)		
0.2	NaPCP	laboratory static bioassay, 91% NaPCP	3-d LC_0	69
1	NaPCP	laboratory static bioassay, 91% NaPCP	1.8-h LC_{100}	69
5	NaPCP	laboratory static bioassay, 91% NaPCP	23-m LC_{100}	69
		Zacco platypus (minnow)		
0.17	2,3,4,5,6-PCP	caged in rice field	24-h LC_{65}	546
0.17	2,3,4,5,6-PCP	caged in rice field	72-h LC_{65}	546
0.23	2,3,4,5,6-PCP	larval stages	24-h LC_{50}	546
0.34	2,3,4,5,6-PCP	caged in rice field, 72-h test	24-h EC_{100}	546
0.85	2,3,4,5,6-PCP	caged in rice field, 72-h test	24-h LC_{100}	546
		(*Leuciscus*) *Idus idus melanotus* (golden orfe)		
8.3	2-MCP	pH 7 to 8	48-h	366

			LC_{50}	
3	3- & 4-MCP	pH 7 to 8	48-h LC_{50}	366
5	2,4-DCP		48-h LC_{50}	419
4	2,6-DCP	static bioassay	48-h LC_{50}	395
		Oryzias latipes (mendaka)		
13	2,4,5-TCP		96-h LC_{50}	237
0.082	2,3,4,5,6-PCP		48-h LC_{50}	223
0.93	2,3,4,5,6-PCP		LC_0	112
1.1	2,3,4,5,6-PCP		48-h EC_{50}	112
0.4	2,3,4,5,6-PCP		96-h LC_{50}	237
		Channa gachua		
0.39	NaPCP		96-h LC_{50}	123
0.43	NaPCP		72-h LC_{50}	123
0.56	NaPCP		48-h LC_{50}	123
0.79	NaPCP		24-h LC_{50}	123
		Salvelinus fontinalis (brook trout)		
0.109	2,3,4,5,6-PCP		9-d LC_{50}	622
0.128	2,3,4,5,6-	flow-through bioassay	96-h	205

	PCP		LC_{50}	
0.128	2,3,4,5,6-PCP	flow-through bioassay	96-h LC_{50}	622
0.3	2,3,4,5,6-PCP	static bioassay	24-h EC_{50}	622
0.118	NaPCP	adults in a lab. flow-through bioassay	9-d LC_{50}	622
0.315	NaPCP	adults in a lab. flow-through bioassay	24-h LC_{50}	622

Table 8.2.8.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Micropterus salmoides (largemouth bass)		
0.0075	2,3,4,5,6-PCP	11 mm long, <30 d old, growth reduction	EC	115
0.0252	2,3,4,5,6-PCP	growth retardation	EC	250
0.041	2,3,4,5,6-PCP	11 mm long, <30 d old, feeding behavior	EC	115
0.05	2,3,4,5,6-PCP	reduction in food conversion efficiency	30%	151
0.162	2,3,4,5,6-PCP	55 mm long, <84 days old, range 0.136 to 0.189	LC_{50}	115
0.281	2,3,4,5,6-PCP	11 mm long, <30 days old, range 0.275 to 0.287	LC_{50}	115
		Pimephales notatus (bluntnose minnow)		
0.36	2,3,4,5,6-PCP	laboratory static bioassay	4-h LC_{50}	632
0.92	2,3,4,5,6-	laboratory static bioassay	80-m	632

	PCP		EC_{50}	
4.6	2,3,4,5,6-PCP	laboratory static bioassay	21-m LC_{50}	632
0.2	NaPCP	laboratory static bioassay	3-d LC_0	69
1	NaPCP	laboratory static bioassay	2.5-h LC_{100}	69
5	NaPCP	laboratory static bioassay	42-m LC_{100}	69
		Ptychocheilus oreganensis (northern squawfish)		
10	2,4-DCP	lab. static bioassay, 10.6C, balance loss	EC	498
10	2,4-DCP	lab. static bioassay, 10.6C, balance loss	EC	615
10	2,4-DCP	lab. static bioassay, 10.6C, balance loss	3-h EC_{100}	615
10	2,4,5-TCP	lab. static bioassay, 10.6C, balance loss	1-h LC_{100}	615
1	TCP	lab. static bioassay, 11.1C, balance loss	1-h LC_{100}	615
1	TCP	lab. static bioassay, 11.1C, balance loss	4-h LC_{100}	615
5	TCP	lab. static bioassay, 13.9C, balance loss	0.5-h EC_{100}	615
5	TCP	lab. static bioassay, 13.9C, balance loss	1-h LC_{100}	615
10	TCP	lab. static bioassay, 10C, balance loss	1-h LC_{100}	615
10	TCP	lab. static bioassay, 10C, balance loss	0.5-h LC_{100}	615
10	TCP	lab. static bioassay, 20C, balance loss	1-h LC_{100}	615

		Jordanella floridae (american flagfish)		
55	2,3,4,5,6-PCP	larval & fry survival, pH 6.95, 25 C, <28-d	NOEL	763
102	2,3,4,5,6-PCP	larval & fry survival, pH 6.95, 25 C, <28-d	LOEL	763

Table 8.2.8.2 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Petromyzon marinus (lamprey)		
5	2,4-DCP	static bioassay, 12.8C, Lake Huron water	1-h EC_{100}	498
5	2,4-DCP	static bioassay, 12.8C, Lake Huron water	12-h LC_{100}	498
5	2,4-DCP	static bioassay, 12.8C, Lake Huron water	12-h LC_{100}	614
5	2,4-DCP	static bioassay with larvae	1-h EC	614
5	2,4-DCP	static bioassay with larvae	12-h LC_{100}	614
5	2,6-DCP	static bioassay with larvae	12-h EC	614
5	3,4-DCP	static bioassay with larvae, 12.8C	11-h LC_{100}	614
1	2,4,5-TCP	static bioassay with larvae, 12.8C, Dowicide 2	24-h NOEL	614
5	2,4,5-TCP	static bioassay with larvae, 12.8C, Dowicide 2	3-h LC_{100}	614
0.924	2,3,4,5,6-PCP	12.8C	4-h LC_{100}	614
		FISH		
58	2-MCP	minnows, static bioassay	24-h LC_{50}	461
58	2-MCP	23C	24-h TLm	184

18	3-MCP	23C	24-h TLm	184
14	4-MCP	23C	24-h TLm	184
3.2	2,4,6-TCP	23C	24-h TLm	184
3.2	2,4,6-TCP	static bioassay with shiners	24-h LC_{50}	461
0.2	2,3,4,5,6-PCP	cichlids growth	decreased	150
0.2	2,3,4,5,6-PCP	cichlids food intake	increased	150
0.2	NaPCP	fish in a field study	EC	171
0.2-0.6	NaPCP	19 species of fish, lab. static bioassays	LC	69
0.4	NaPCP	9 to 24C, toxicity rises as pH drops from 6.6	LC	69
0.4	NaPCP	9 to 24C, toxicity rises as pH drops from 6.6	LC	169
6	NaPCP	centrachids	EC	162
6	NaPCP	catfish in a field study	EC	162
9.5	NaPCP	catfish in a laboratory study	LC	166
9.5	NaPCP	catfish, eels and guppies in a field study	LC	166
9.5	NaPCP	catfish, eels and guppies in a field study	LC	173

Table 8.2.8.3 The Effects of Chlorophenols on Fish...*Lepomis macrochirus* (bluegill sunfish)

mg/L	Conditions	Effect	Ref.
	2-MCP		
6.6	pH 6.5-7.9, 22C, hardness 40	96-h LC_{50}	390

	mg/L, static bioassay, adults		
6.6	pH 6.5-7.9, 22C, hardness 40 mg/L, static bioassay, adults	96-h LC_{50}	364
8.1	pH 7.6-8.4, static bioassay, fry	96-h LC_{50}	391
8.1	hardness 20 mg/L	48-h LC_{50}	315
8.1	hardness 20 mg/L, lab. static bioassay, 20C	48-h LC_{50}	391
8.2	hardness 20 mg/L, lab. static bioassay, 20C	24-h LC_{50}	391
8.2	hardness 20 mg/L, lab. static bioassay, 20C	24-h LC_{50}	315
8.4	pH 7.4, 22C, lab. static bioassay, fry	96-h LC_{50}	360
8.4	juveniles, laboratory static bioassay	96-h LC_{50}	364
10	25C, laboratory static bioassay in soft water	24-h LC_{50}	391
10	adults, static bioassay	96-h LC_{50}	364
10	pH 7.5, static bioassay, 25C, adults, hardness 20 mg/L	96-h EC_{50}	387
	4-MCP		
3.8		LC	220
3.83	pH 6.5-7.9, 22C, static bioassay, hard. 40 mg/L	96-h LC_{50}	372
3.83	pH 6.5-7.9, 22C, static bioassay, hard. 40 mg/L	96-h LC_{50}	390
	2,4-DCP		
2.0	pH 6.5-7.9, 22C, static bioassay, hard. 40 mg/L	96-h LC_{50}	390
2.02	static bioassay	96-h LC_{50}	495
5.0	12.8C, laboratory static bioassay	1-h EC_{100}	498
5.0	12.8C, laboratory static bioassay	12-h	498

		LC_{100}	
5.0	12.8C, laboratory static bioassay	12-h LC_{100}	614
	2,6-DCP		
5.0	17C, laboratory static bioassay	5-h LC_{100}	614
	3,4-DCP		
5.0	17C, laboratory static bioassay	3-h LC_{100}	614
	2,3,5-TCP		
0.45		LC	220
	2,3,6-TCP		
0.32		LC	220

Table 8.2.8.3 (continued)

mg/L	Conditions	Effect	Ref.
	2,4,5-TCP		
0.45	static bioassay	96-h LC_{50}	372
0.45	pH 7.2, 22C, hardness 40 mg/L	96-h LC_{50}	390
1.0	12.8C, laboratory static bioassay, Dowicide 2	24-h NOEL	614
5.0	12.8C, laboratory static bioassay, Dowicide 2	2-h LC_{100}	614
	2,4,6-TCP		
0.32	pH 7.2, 22C, hardness 40 mg/L	96-h LC_{50}	390
0.32	static bioassay	96-h LC_{50}	372
0.41	pH 7.4-9.4, 22C, hardness 173 mg/L, DO 6.5-9.1 mg/L	96-h LC_{50}	245
	2,3,4,6-TTCP		
0.10	pH 7.5, 22C, hardness 38 mg/L, 74% tech. grade	96-h LC_{50}	236

0.12	pH 7.5, 22C, hardness 38 mg/L, 74% tech. grade	24-h LC_{50}	236
0.14		LC	220
0.14	pH 6.5-7.9, 22C, hardness 32-99 mg/L, static bioassay, fry	96-h LC_{50}	390
0.19	pH 6.5-7.9, 22C, hardness 32-99 mg/L, static bioassay, fry	24-h LC_{50}	390
	2,3,5,6-TTCP		
0.17		LC	220
0.17	pH 6.7-7.8, 22C, hardness 32-48 and alkalinity 28-34 mg/L, young	96-h LC_{50}	390
0.17	pH 6.7-7.8, 22C, hardness 32-48 and alkalinity 28-34 mg/L, young	96-h LC_{50}	459
0.4	pH 6.7-7.8,22C, hardness 32-48 and alkalinity 28-34 mg/L, young	24-h LC_{50}	390
0.4	pH 6.7-7.8,22C, hardness 32-48 and alkalinity 28-34 mg/L, young	24-h LC_{50}	459
	PCP		
0.03	24C, soft water	48-h LC_{50}	124
0.03	24C, medium hardness water	48-h LC_{50}	124
0.032	15C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.032	15C, pH 7.2-7.5, hardness 40-50 mg/L, static bioassay, alkalinity 30-35 mg/L	96-h LC_{50}	111
0.040	pH 7.6-8.5, hardness 126-168, growth assay	decreased	102
0.040	medium hardness water, 24C	24-h LC_{50}	124
0.040	hard water, 24C	48-h LC_{50}	124
0.050	soft water, 24C	24-h LC_{50}	124
0.050	hard water, 24C	24-h LC_{50}	124

Table 8.2.8.3 (continued)

mg/L	Conditions	Effect	Ref.
0.060		96-h LC_{50}	619
0.060	ranges to 0.077, static bioassay	96-h LC_{50}	205
0.065	15C, pH 7.4, hardness 44 mg/L, static bioassay	24-h LC_{50}	111
0.077		96-h LC_{50}	619
0.124	pH 7.6-8.5, hardness 126-168 mg/L	EC	102
0.174		14-d LC_{50}	622
0.20	16.4C, pH 7.8	LC_{50}	113
0.20	pH 7.5-8.2, 7.3-17.4C, river water	96-h LC_{50}	206
0.202	pH 8.0, 17.7C,	LC_{50}	114
0.215	pH 7.4, 15C, hardness 272 mg/L, FT bioassay	96-h LC_{50}	111
0.26	ranges to 0.305, replacement static bioassay	96-h LC_{50}	205
0.26	pH 7.2-7.7, 19C	96-h LC_{50}	121
0.27	pH 7.8, 24.5C	LC_{50}	113
0.305	19C, pH 7.4	96-h LC_{50}	121
0.340	15C, pH 7.4, hardness 272 mg/L, FT bioassay	24-h LC_{50}	111
0.365	pH 7.6-8.5, hardness 126-168	8-d LC_{100}	102
0.920	17C	8-h LC_{100}	614
0.920		48-h LC_{50}	633
	NaPCP		
0.02	24C, hard water	96-h LC_{50}	124
0.03		48-h LC_{50}	124
0.044	15C, pH 7.4, hardness 44 mg/L	96-h LC_{50}	111

0.048	pH 7.2, 23C, growth rate, food conversion	22-d LOEL	760 761
0.07	15C, pH 7.4, hardness 44 mg/L	24-h LC_{50}	111
0.1	'Santobrite', 20C, laboratory static bioassay	EC, not lethal	633
0.1	'Dowicide G', 12.8C, laboratory static bioassay	1-h LC_{100}	614
0.22	juveniles	14-d LC_{50}	622
0.30	juveniles	30-h LC_{50}	622
0.33	pH 7.2-7.7, 19C	96-h LC_{50}	121
0.34	20C, pH 7.4, hardness 272 mg/L	96-h LC_{50}	111
0.35	'Santobrite', 20C, laboratory static bioassay	24-h LC_{50}	633
0.35	'Santobrite', 20C, laboratory static bioassay	48-h LC_{50}	633
0.546	20C, pH 7.4, hardness 272 mg/L	24-h LC_{50}	111
1.0	'Dowicide G', 12.8C, laboratory static bioassay	4-h LC_{100}	614
5.0	'Dowicide G', 12.8C, laboratory static bioassay	1-h LC_{100}	614

Table 8.2.8.4 The Effects of Pentachlorophenol on Fish...*Rasbora daniconius neilgeriensis*

mg/L	Conditions	Effects	Ref.
0.010	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	NOEL	107
0.067	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	96-h LC_{16}	107

0.113	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	72-h LC_{16}	107
0.148	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	96-h LC_{50}	107
0.155	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	48-h LC_{16}	107
0.229	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	24-h LC_{16}	107
0.233	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	72-h LC_{50}	107
0.297	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	48-h LC_{50}	107
0.330	pH 7.8,31C, hardness 232, alkalinity 215, DO 6.4	96-h LC_{84}	107
0.336	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	12-h LC_{16}	107
0.361	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	24-h LC_{50}	107
0.501	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	12-h LC_{50}	107
0.531	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	72-h LC_{84}	107
0.570	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	48-h LC_{84}	107
0.570	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	24-h LC_{84}	107
0.748	pH 7.8, 31C, hardness 232, alkalinity 215, DO 6.4	12-h LC_{84}	107

Table 8.2.8.5 The Effects of Chlorophenols on Fish...*Pimephales promelas* (fathead minnows)

mg/L	Conditions	Effect	Ref.
	2-MCP		
3.9		EC	220
3.9	larval embryo stages	NOEL	364
3.9	chronic tests	NOEL	364
6.3	pH 7.5	192-h LC_{50}	214
9.4		96-h LC_{50}	402
9.7	pH 7.5, laboratory static bioassay	48-h LC_{50}	214
11	pH 7.5, 23C, hardness 451 mg/L	96-h LC_{50}	214
11.6	static bioassay, nominal concentration	96-h LC_{50}	364
11.63	pH 7.5, hard. 20 mg/L, 25C, lab. static bioassay	96-h LC_{50}	389
12.4	continuous-flow bioassay, measured conc.	96-h LC_{50}	364
12.4	pH 7.5	96-h LC_{50}	214
13	pH 7.5, 23C, hardness 45 mg/L	96-h LC_{50}	214
13.8	30 days old	96-h LC_{50}	152
14.48	pH 8.2, 25C, hardness 360 mg/L	96-h LC_{50}	389
14.5	static bioassay, nominal concentration	96-h EC_{50}	364
	4-MCP		
3.8	pH 7.6-8.3, static bioassay, juvenile	96-h LC_{50}	394
4	pH 7.6-8.3, static bioassay, fry	96-h LC_{50}	394
5	pH 7.6-8.3, static bioassay, adult	96-h LC_{50}	394
	2,4-DCP		
0.29	lowest tested value rated as	NOEL	495
0.36	pH 7.2-7.9, embryo to 28-day old	28-d LC_0	377

	larvae, survival		
0.365		MATC	495
0.375	pH 7.5, 25C, hardness 46 mg/L, 32 days old	EC	377
0.38	range 0.29-0.46, 4-week old larvae, survival	LC_0	377
0.46	highest tested value rated as	NOEL	495
6.5	pH 7.5, 23C, hardness 45 mg/L	8-d LC_{50}	214
7.4	pH 7.6, 25C, hardness 46 mg/L	8-d LC_{72}	375
7.4	pH 9.1, 25C, hardness 46 mg/L	8-d LC_0	375
8.23	pH 7.5	96-h LC_{50}	214
8.23	continuous-flow bioassay, juveniles	96-h LC_{50}	495
8.3	pH 7.5, 23C, hardness 45 mg/L	96-h LC_{50}	214
9.37	30 days old	96-h LC_{50}	152
	2,3,6-TCP		
0.72		EC	220

Table 8.2.8.5 (continued)

mg/L	Conditions	Effect	Ref.
	2,4,6-TCP		
0.1-1.0		96-h LC_{50}	462
0.1-10	laboratory static bioassay	96-h TLm	188
0.53	lowest tested value rated as	NOEL	372
0.6		96-h LC_{50}	617
0.72	early life stage	EC	451
0.72		MATC	372
0.97	highest tested value rated as	NOEL	372
2.74		96-h LC_{50}	245

4.55		96-h LC_{50}	402
5.8	pH 7.5, 23C, hardness 45 mg/L	8-d LC_{50}	214
6.4	pH 7.5, 23C, hardness 45 mg/L	96-h LC_{50}	214
8.6	pH 7.5, 23C, hardness 45 mg/L, flowing bioassay	96-h LC_{50}	214
9.04	flow-through bioassay	96-h LC_{50}	372
9.14	30 days old	96-h LC_{50}	152
9.7	pH 7.5, 23C, hardness 45 mg/L, static bioassay	96-h LC_{50}	214
	2,3,4,5-TTCP		
0.41	30 days old	96-h LC_{50}	152
0.41	pH 7.5, 25C, 30 days old, flow-through bioassay, hardness 46 mg/L	96-h LC_{50}	260
0.441	pH 6.9-7.7, 24C, 30 d old, flow-through bioassay	72-h LC_{50}	261
0.441	pH 6.9-7.7, 24C, 30 d old, flow-through bioassay	96-h LC_{50}	261
0.450	pH 6.9-7.7, 24C, 30 d old, flow-through bioassay	48-h LC_{50}	261
0.496	pH 6.9-7.7, 24C, 30 d old, flow-through bioassay	24-h LC_{50}	261
0.75	pH 6.5-7.9, 22C, hardness 32-99 mg/L, static bioassay, fry	96-h EC_{50}	261
	2,3,4,6-TTCP		
0.17	74% tech. grade, 22C, pH 7.4, hardness 40 mg/L	96-h LC_{50}	236
0.27	74% tech. grade, 22C, pH 7.4, hardness 40 mg/L	24-h LC_{50}	236
	PCP		
>0.013	tech. grade, growth assay	reduced	127

>0.027	tech. grade, survival assay	reduced	127
0.035	pH 6.5, fry survival	reduced	79
0.04	pH 7.6-85, hardness 126-168 mg/L, growth rate	reduced	102
0.045	pH 7.55, 25C, hardness 46 mg/L, fry	32-d NOEL	377
0.057		EC	205
0.058	pH 7.5, fry weight	reduced	79
0.073	25C, pH 7.55, hardness 46 mg/L, larval weight	32-d LOEL	377
>0.085	purified, chronic growth effects	reduced	127

Table 8.2.8.5 (continued)

mg/L	Conditions	Effect	Ref.
0.095	pH 6.5	24-h LC_{50}	79
0.12	adult, 16.6C, pH 7.9	LC_{50}	113
0.12	adult, 12.2C, pH 7.6	LC_{50}	113
0.124	pH 7.5, eggs, number hatching	reduced	79
0.124	pH 7.5, fry, survival	reduced	79
0.124	pH 7.6-8.5, hardness 126-168 mg/L	LC_0	102
0.125	pH 8.5, fry, survival	reduced	79
0.128	25C, pH 7.2-7.9, hardness 46 mg/L, egg hatch and larvae survival	28-d LC_{21}	377
0.13	25C, weight after 90 days	reduced	147
0.135	range 0.08-0.19, hardness 220 mg/L	96-h LC_{50}	122
0.141		19-d LC_{50}	622
0.16	adult, 10.3C, pH 8.1	LC_{50}	113
0.161	pH 8.0, fry, survival	reduced	79

0.17	adult, 10.1C, pH 8.0	LC_{50}	113
0.17	survival	LC_{0}	112
0.18	ranges to 0.314, flow through bioassay	96-h LC_{50}	205
0.18	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.19	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.19	adult, 4C, pH 8.0	LC_{50}	113
0.194	pH 8.0, 15C	96-h LC_{50}	623
0.2	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.2	7.3-17.4C, pH 7.5-8.2, river water	96-h LC_{50}	206
0.2	15C, pH 7.4-7.5, hardness 400, alkalinity 278 mg/L, death rate	24-h 80%	168
0.2	pH 7.5, 25C, hardness 45 mg/L	8-d LC_{50}	214
0.205	20C, pH 7.4, hard. 272, flow-through bioassay	96-h LC_{50}	111
0.205	technical grade	96-h LC_{50}	127
0.205	20C, pH 7.2-7.5, hardness 40-50, alkalinity 30-35 mg/L, static bioassay	96-h LC_{50}	128
0.205	20C	96-h LC_{50}	128
0.208	adult, 18.7C, pH 7.6	LC_{50}	113
0.21	technical grade, 25C, pH 7.4-7.8, flow-through bioassay	96-h LC_{50}	122
0.21	juveniles, pH 7.2, 25C, hardness 220 mg/L, laboratory flow-through bioassay	96-h LC_{50}	122
0.21		48-h LC_{50}	112
0.21	juveniles, pH 7.2, 25C, hardness 220 mg/L, laboratory flow-through bioassay	96-h LC_{50}	282

Table 8.2.8.5 (continued)

mg/L	Conditions	Effect	Ref.
0.21	pH 7.6, 25C, hardness 210 mg/L	11-d LC_{50}	122
0.218	pH 7.5	96-h LC_{50}	79
0.22	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.22	laboratory flow-through bioassay	24-h LC_{50}	122
0.22	laboratory flow-through bioassay	24-h LC_{50}	282
0.22	juvenile, 25C, pH 7.4-8.3, hardness 43-49 mg/L, flow -through bioassay	8-d LC_{50}	214
0.221	juvenile, 25C, pH 7.4-8.3, hardness 43-49 mg/L, flow-through bioassay	96-h LC_{50}	214
0.223	eggs, 25C, pH 7.2-7.9, hardness 46 mg/L	LC100	377
0.23	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.23	pH 7.5, 23C, hardness 45 mg/L	96-h LC_{50}	214
0.23	20C, pH 7.4, hardness 272, flow-through bioassay	24-h LC_{50}	111
0.24	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.24	30-day old fish	96-h LC_{50}	152
0.26	15C, pH 7.9-8.2, embryos, flow-through bioassay	96-h LC	554
0.261	pH 8.0	96-h LC_{50}	79
0.263		96-h LC_{50}	623
0.266	18.7C, pH 8.0	LC_{50}	114
0.27	pH 7.2, 25C, hardness 220 mg/L	96-h LC_{50}	122
0.3	15C, pH 7.4-7.5, hardness 400, alkalinity 278 mg/L, death rate	24-h 100%	168
0.3		LC_{50}	182
0.3	adult, 3.4C, pH 8.0	LC_{50}	113

0.31		21-h LC_{50}	622
0.314	pH 8.0, 25C	96-h LC_{50}	623
0.314	fry, 23.6C, pH 8.0	LC_{50}	113
0.34	25C, pH 7.8-8.2, embryos, flow-through bioassay	96-h LC	554
0.34	15C, pH 7.4-7.5, hard. 400, alkalinity 278 mg/L,	96-h LC_0	168
0.36	15C, pH 7.4-7.5, hard. 400, alkalinity 278 mg/L,	96-h LC_{40}	168
0.365	pH 7.6-8.5, hardness 126-168 mg/L	8-d LC100	102
0.378	pH 8.5	96-h LC_{50}	79
0.396	young, 23.9C, pH 8.0	LC_{50}	113
0.4	15C, pH 7.4-7.5, hard. 400, alkalinity 278 mg/L,	96-h LC_{70}	168
0.465	eggs, 25.2C, pH 7.9	LC_{50}	113
0.47	25C, pH 7.4, hardness 272, alkalinity 237 mg/L	96-h LC_{50}	127
0.48	young, 24.9C, pH 8.0	LC_{50}	113

Table 8.2.8.5 (continued)

mg/L	Conditions	Effect	Ref.
0.51	young, 24.8C, pH 7.9	LC_{50}	113
0.6	4-8 weeks old, 18-22C, pH 5.9, lab. static bioassay	24-h LC_{50}	548
0.6	4-8 weeks old, 18-22C, pH 5.9, lab. static bioassay	48-h LC_{50}	548
0.6	4-8 weeks old, 18-22C, pH 5.9, lab. static bioassay	72-h LC_{50}	548
0.6	4-8 weeks old, 18-22C, pH 5.9, lab. static bioassay	96-h LC_{50}	548

0.6	static bioassay	96-h LC_{50}	205
0.92	10C, pH 7.5, hardness 400, alkalinity 278 mg/L	260-min. LC_{50}	168
0.92	18C, pH 7.5, hardness 400, alkalinity 278 mg/L	81-min. LC_{50}	168
0.92	26C, pH 7.5, hardness 400, alkalinity 278 mg/L	46-min. LC_{50}	168
1	18C, pH 5.9, hardness 400, alkalinity 278 mg/L	28-min. LC_{50}	168
1	18C, pH 7.5, hardness 400, alkalinity 278 mg/L	81-min. LC_{50}	168
1	18C, pH 8.9, hardness 400 & alkalinity 278 mg/L	26-h LC_{50}	168
7.9	1-month old fish, static bioassay	48-h LC_{50}	214
8	4-8 weeks old, 18-22C, pH 5.9, lab. static bioassay	1-h LC_{50}	548
	NaPCP		
0.02	22C, hardness 200 mg/L, pH 7.7	48-h LC_{50}	80
0.02	22C, hardness 200 mg/L, pH 7.7	96-h LC_{50}	80
0.15	laboratory flow-through bioassay	14-d LC_{50}	622
0.21	15C, laboratory flow-through bioassay	48-h LC_{50}	623
0.21	15C, laboratory flow-through bioassay	96-h LC_{50}	623
0.21	15C, laboratory flow-through bioassay	LTC	623
0.27	20C, pH 7.4, hardness 272 mg/L	96-h LC_{50}	111
0.32	pH 8.0, 15C, laboratory static bioassay	24-h LC_{50}	168
0.33	25C, laboratory flow-through bioassay	LTC	623

0.34	laboratory flow-through bioassay	21-h LC_{50}	622
0.34	25C, laboratory flow-through bioassay	96-h LC_{50}	623
0.37	20C, pH 7.4, hardness 272 mg/L	24-h LC_{50}	111
0.37	25C, laboratory flow-through bioassay	48-h LC_{50}	623
0.40	pH 8.0, 15C, laboratory static bioassay	LC100	168

Table 8.2.8.6 The Effects of Chlorophenols on Fish...*Notopterus notopterus*

mg/L	Conditions	Effect	Ref.
	PCP		
0.0042	enzymatic activities in most tissues	EC	139
0.0065	enzymatic activities in all tissues	EC	139
0.083		30-d LC_{50}	139

mg/L	Conditions	Effect	Ref.
	NaPCP		
0.00043	36C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	NOEL	142
0.0011	23C, pH 7.2, DO 6.5 mg/L, fish 4.5 cm long	NOEL	132
0.003	23C, pH 7.2, DO 6.5 mg/L, fish 22.6 cm long	NOEL	132
0.0035	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	NOEL	132
0.0037	16C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	NOEL	142

0.004	13-20 cm and 35-60 g fish, protein and cholesterol down; P, N, Na, K, Mg, Ca, Fe, Cl and glucose up	change in serum levels	154
0.0045	23C, pH 7.2, DO 6.5 mg/L, fish 14.5 cm long	NOEL	132
0.0098	36C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	96-h LC_{50}	142
0.012	36C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	72-h LC_{50}	142
0.017	36C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	48-h LC_{50}	142
0.02	36C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	24-h LC_{50}	142
0.032	23C, pH 7.2, DO 6.5 mg/L, fish 4.5 cm long	96-h LC_{50}	132
0.035	23C, pH 7.2, DO 6.5 mg/L, fish 4.5 cm long	72-h LC_{50}	132
0.039	23C, pH 7.2, DO 6.5 mg/L, fish 4.5 cm long	48-h LC_{50}	132
0.044	23C, pH 7.2, DO 6.5 mg/L, fish 4.5 cm long	24-h LC_{50}	132
0.083	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	96-h LC_{50}	132
0.083	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	96-h LC_{50}	142
0.09	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	96-h LC_{50}	142
0.09	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	72-h LC_{50}	132
0.093	23C, pH 7.2, DO 6.5 mg/L, fish 22.6 cm long	96-h LC_{50}	132
0.1	23C, pH 7.2, DO 6.5 mg/L, fish	72-h LC_{50}	132

	22.6 cm long		
0.107	16C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	96-h LC_{50}	142
0.107	23C, pH 7.2, DO 6.5 mg/L, fish 22.6 cm long	48-h LC_{50}	132
0.109	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	48-h LC_{50}	132
0.109	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	48-h LC_{50}	142
0.11	16C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	72-h LC_{50}	142
0.113	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	24-h LC_{50}	132
0.113	23C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	24-h LC_{50}	142
0.114	16C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	48-h LC_{50}	142
0.119	16C, pH 7.2, DO 6.5 mg/L, fish 9.0 cm long	24-h LC_{50}	142
0.12	23C, pH 7.2, DO 6.5 mg/L, fish 22.6 cm long	24-h LC_{50}	132
0.131	23C, pH 7.2, DO 6.5 mg/L, fish 14.5 cm long	96-h LC_{50}	132
0.134	23C, pH 7.2, DO 6.5 mg/L, fish 14.5 cm long	72-h LC_{50}	132
0.138	23C, pH 7.2, DO 6.5 mg/L, fish 14.5 cm long	48-h LC_{50}	132
0.143	23C,pH 7.2, DO 6.5 mg/L, fish 14.5 cm long	24-h LC_{50}	132

Table 8.2.8.7 The Effects of Chlorophenols on Fish...*Oncorhynchus* Sp. (trout and salmon).

mg/L	chlorophenol	species and conditions	effect	ref.
		Oncorhynchus gorbuscha (pink salmon)		
0.6	2,4-DCP	14C, pH 8.2, hardness 180 mg/L, 0.8 g and 4.2 cm fry	48-h LC_{50}	415
0.6	2,4-DCP	14C, pH 8.2, hardness 180 mg/L, 0.8 g and 4.2 cm fry	96-h LC_{50}	415
0.8	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 0.8 g and 4.2 cm fry	48-h LC_{50}	415
0.8	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 0.8 g and 4.2 cm fry	96-h LC_{50}	415
0.8	2,4-DCP	14C, pH 6.3, hardness 5.3 mg/L, 0.8 g and 4.2 cm fry	24-h LC_{50}	415
0.8	2,4-DCP	14C, pH 6.3, hardness 5.3 mg/L, 0.8 g and 4.2 cm fry	96-h LC_{50}	415
		Oncorhynchus tshawytacha (chinook salmon)		
0.6	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 4.2 cm, 0.8 g	24-h LC_{50}	415
0.6	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 4.2 cm, 0.8 g	96-h LC_{50}	415
10	2,4-DCP	10.6C, static bioassay	1-h LC_{100}	498
10	2,4-DCP	10.6C, static bioassay	1-h LC_{100}	615
10	2,4,5-TCP	10.6C, static bioassay	1-h LC_{100}	615
1	TCP	11.1C, laboratory static bioassay, equilibrium loss	1-h EC_{100}	615

1	TCP	11.1C, laboratory static bioassay	4-h LC_{100}	615
0.031	2,3,4,5,6-PCP	10C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.065	2,3,4,5,6-PCP	10C, pH 7.4, hardness 44 mg/L, static bioassay	24-h LC_{50}	111
0.067	2,3,4,5,6-PCP	10C, pH 7.4, hardness 44 mg/L, static bioassay	48-h LC_{50}	111
0.068	2,3,4,5,6-PCP	10C, pH 7.4, hardness 44 mg/L, static bioassay	24-h LC_{50}	111
0.068	2,3,4,5,6-PCP	10C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.068	2,3,4,5,6-PCP	10C, pH 7.2-7.5, hardness 40-50 mg/L and alkalinity 30-35 mg/L, static bioassay	96-h LC_{50}	128

Table 8.2.8.7 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
0.072	2,3,4,5,6-PCP	flow-through bioassay	96-h LC_{50}	205
0.072	2,3,4,5,6-PCP	12C, pH 7.0, hardness 54 mg/L	96-h LC_{50}	625
0.0039	NaPCP	blood chemistry effects	96-h EC	246
0.0345	NaPCP	10C, pH 7.4, hardness 44 mg/L, static bioassay, fry	96-h LC_{50}	111
0.0675	NaPCP	10C, pH 7.4, hardness 44 mg/L, static bioassay, fry	96-h LC_{50}	111
0.073	NaPCP	10C, pH 7.4, hardness 44 mg/L, static bioassay, fry	24-h LC_{50}	111
0.078	NaPCP	12C, pH 7.0, flow-through, fry density 13.8 g/L	96-h LC_{50}	625

>0.1	NaPCP	10C, pH 7.4, hardness 44 mg/L, static bioassay, fry	24-h LC_{50}	111
0.165	NaPCP	10C, pH 7.4, hardness 272 mg/L, flow-through, fry	96-h LC_{50}	111
0.170	NaPCP	10C, pH 7.4, hardness 272 mg/L, flow-through	96-h LC_{50}	111
0.224	NaPCP	10C, pH 7.4, hardness 272 mg/L, flow-through	24-h LC_{50}	111
>0.25	NaPCP	10C, pH 7.4, hardness 272 mg/L, flow -through bioassay using fry	24- to 96-h LC_{50}	111
		Oncorhynchus clarki (cutthroat trout)		
0.055	2,3,4,5,6-PCP	range 0.01-0.1, 17C, pH 7.4, hardness 44 mg/L, 1.3 g	96-h LC_{50}	111
0.055	2,3,4,5,6-PCP	range 0.01-0.1, 12C, pH 7.4, hardness 44 mg/L, 1.3 g	96-h LC_{50}	111
0.055	NaPCP	range 0.01-0.1, 12C, pH 7.4, hardness 44 mg/L, 1.3 g	96-h LC_{50}	111
		Oncorhynchus keta (chum salmon)		
1.1	2,4-DCP	14C, pH 7.8, hard. 40 mg/L, 4.2 cm and 0.8 g fry	24-h LC_{50}	415
1.1	2,4-DCP	14C, pH 7.8, hard. 40 mg/L, 4.2 cm and 0.8 g fry	96-h LC_{50}	415

Table 8.2.8.7 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Oncorhynchus kisutch (coho salmon)		
1.5	2,4-DCP	14C, pH 6.3, hardness 5.3	24-h LC_{50}	415

		mg/L, 4.2 cm, 0.8 g fish		
1.5	2,4-DCP	14C, pH 6.3, hardness 5.3 mg/L, 4.2 cm, 0.8 g fish	96-h LC_{50}	415
1.8	2,4-DCP	14C, pH 8.2, hardness 180 mg/L, 4.2 cm, 0.8 g fish	24-h LC_{50}	415
1.8	2,4-DCP	14C, pH 8.2, hardness 180 mg/L, 4.2 cm, 0.8 g fish	96-h LC_{50}	415
2.2	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 4.2 cm, 0.8 g fish	24-h LC_{50}	415
2.2	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 4.2 cm, 0.8 g fish	96-h LC_{50}	415
10	2,4-DCP	10.6C, laboratory static bioassay	1-h LC_{100}	498
10	2,4-DCP	10.6C, laboratory static bioassay	1-h LC_{100}	615
10	2,4,5-TCP	10.6C, laboratory static bioassay	1-h LC_{100}	615
1	TCP	11.1C, laboratory static bioassay, equilibrium loss	1-h EC_{100}	615
1	TCP	11.1C, laboratory static bioassay	2-h LC_{100}	615
5	TCP	13.9C, laboratory static bioassay	0.5-h LC_{100}	615
10	TCP	10C, laboratory static bioassay	1-h LC_{100}	615
0.0032	2,3,4,5,6-PCP		sub lethal	205
0.034	2,3,4,5,6-PCP	ranges up to 0.089, static bioassay	LC_{50}	205
0.034	2,3,4,5,6-PCP	10C, pH 7.0, hardness 5.5 mg/L	96-h LC_{50}	281
0.037	2,3,4,5,6-		96-h LC_{50}	281

	PCP			
0.055	2,3,4,5,6-PCP		LC	220
0.089	2,3,4,5,6-PCP	10C, pH 7.0, hardness 15 mg/L	96-h LC_{50}	281
2.8	2,3,4,5,6-PCP	10C, laboratory static bioassay	EC_{50}	635
0.032	NaPCP	11C, pH 7.0, laboratory static bioassay	96-h LC_{50}	281
0.05	NaPCP	ranges up to 0.13, 0.5 g of fish per L	96-h LC_{50}	281
0.092	NaPCP	10C, pH 7.0, laboratory static bioassay	96-h LC_{50}	281
0.15	KPCP	flow-through bioassay	24-h LC_{50}	671
0.15	KPCP	flow-through bioassay, catabolism	increased	671
0.15	KPCP	flow-through bioassay, loss of fatty acids	22%	671

Table 8.2.8.7 (continued)

mg/L	chlorophenol	species and conditions	effect	ref.
		Oncorhynchus nerka (sockeye salmon)		
0.7	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 0.8 g and 4.2 cm fry	24-h LC_{50}	415
0.7	2,4-DCP	14C, pH 7.8, hardness 40 mg/L, 0.8 g and 4.2 cm fry	96-h LC_{50}	415
0.0016	2,3,4,5,6-PCP		EC	220
*	2,3,4,5,6-PCP	rate of growth and food conversion efficiency * (1.74	reduced	258

		µg/L)		
0.046	2,3,4,5,6-PCP	ranges to 0.12, static bioassay	96-h LC_{50}	205
0.046	2,3,4,5,6-PCP	pH 7.2, 13C, hardness 85 mg/L	96-h LC_{50}	281
0.058	2,3,4,5,6-PCP	flow-through bioassay	96-h LC_{50}	205
0.058	2,3,4,5,6-PCP	pH 6.8, 15C	96-h LC_{50}	94
0.12	2,3,4,5,6-PCP	pH 7.7, 8C, hardness 47 mg/L	96-h LC_{50}	281
1.61	2,3,4,5,6-PCP	pH 6.8, 15C, growth inhibition	56-d EC_{50}	94
1.66	2,3,4,5,6-PCP	pH 6.8, 15C, food conversion efficiency	56-d EC_{50}	95
0.0017	NaPCP	growth rate (1.74 µg/L)	EC_{50}	94
0.0018	NaPCP	food conversion efficiency (1.80 µg/L)	EC_{50}	94
0.002	NaPCP	fry growth rate and food conversion efficiency	reduced	94
0.032	NaPCP	ranges up to 0.092, 0.5 g of fish per L	96-h LC_{50}	281
0.05	NaPCP	pH 7.2, 13C, laboratory static bioassay	96-h LC_{50}	281
0.063	NaPCP	fry, 15C, pH 6.8, 90% saturated DO	96-h LC_{50}	94
0.130	NaPCP	8C, pH 7.7, laboratory static bioassay	96-h LC_{50}	281

Table 8.2.8.7 (continued)

mg/L	species and conditions	effect	ref.

Oncorhynchus mykiss (rainbow trout)			
2-MCP			
2.1	pH 7.7, 12C, hardness 280 mg/L	96-h LC_{50}	611
2.1		LC_{100}	220
2.6		96-h LC_{50}	365
2.6		96-h LC_{50}	315
2.7		48-h LC_{50}	315
2.8		24-h LC_{50}	315
3-MCP			
2.9	pH 7.7, 12C, hardness 280 mg/L	96-h LC_{50}	611
2.9		LC_{100}	220
10.0	pH 7.5, 15C, soft water	48-h LC_{50}	190
4-MCP			
8.5	pH 7.0	96-h LC_{50}	144
9.1	pH 8.0	96-h LC_{50}	144
2,4-DCP			
0.07		EC_{100}	220
0.07	larvae, hardness 200 mg/L	24-d LC_{50}	376
0.07	14C, pH 7.8, hardness 200 mg/L, embryos	23-d LC_{50}	376
0.07	14C, pH 7.8, hard. 200 mg/L, embryos and larvae	27-d LC_{50}	376
0.08	14C, pH 7.8, hardness 50 mg/L, embryos	23-d LC_{50}	376
0.08	14C, pH 7.8, hard. 50 mg/L, embryos and larvae	27-d LC_{50}	376
0.9	14C, pH 6.3, hardness 5.3 mg/L, 4.2 cm, 0.8 g fish	24-96h LC_{50}	415
1.4	14C, pH 7.8, hardness 40 mg/L, 4.2 cm, 0.8 g fish	24-96h LC_{50}	415
1.8	14C, pH 8.2, hardness 180 mg/L, 4.2 cm, 0.8 g fish	24-96h LC_{50}	415
2.8	12C, pH 7.7, hardness 280 mg/L	96-h LC_{50}	611

5.0	12.8C, laboratory static bioassay	3-h LC_{100}	614
5.0	12.8C, laboratory static bioassay	6-h LC_{100}	614
5.0	18.8C, Lake Huron water	3.5-h LC_{100}	498
2,6-DCP			
5.0	12.8C, laboratory static bioassay	13-h LC_{100}	614
3,4-DCP			
5.0	12.8C, laboratory static bioassay	3-h LC_{100}	614
TCP			
10.0	10C, laboratory static bioassay	1-h LC_{100}	615

Table 8.2.8.7 (continued)

mg/L	species and conditions	effect	ref.
2,4,5-TCP			
0.1	12.8C, laboratory static bioassay, Dowicide-2	24-h NOEL	614
1.0	12.8C, laboratory static bioassay, Dowicide-2	4-h EC	614
1.0	static bioassay	48-h LC_{50}	372
1.0	15C, soft water	48-h LC_{50}	190
5.0	12.8C, laboratory static bioassay, Dowicide-2	24-h LC_{100}	614
2,4,6-TCP			
0.05	bile concentration	increases	137
0.20	enzyme effects	96-h EC_{50}	245
0.45	12C, pH 6.4, hardness 280 mg/L	96-h LC_{50}	611

0.73		96-h LC_{50}	245
2,3,4,5-TTCP			
0.205	pH 6.9-7.7, 10 g fish, flow-through bioassay	96-h LC_{50}	261
0.284	pH 6.9-7.7, 10 g fish, flow-through bioassay	72-h LC_{50}	261
0.284	pH 6.9-7.7, 10 g fish, flow-through bioassay	48-h LC_{50}	261
0.304	pH 6.9-7.7, 10 g fish, flow-through bioassay	24-h LC_{50}	261
2,3,4,6-TTCP			
0.085	pH 7.2, 12C, hardness 44 mg/L, static bioassay, 74% technical grade	96-h LC_{50}	236
0.1	pH 7.2, 12C, hardness 44 mg/L, static bioassay, 74% technical grade	24-h LC_{50}	236
PCP			
*	15C, fry * (0.035 µg/L)	NOEL	126
*	15C, fry, growth rate * (0.66 µg/L)	reduced	126
0.0074	28-day growth inhibition	27%	616
0.0074	92-day growth inhibition	9%	283
0.092	41-day growth inhibition	9%	629
0.01	eggs held at low temperatures	EC	156
0.01	biomass, eggs @ 10C, alevins @ 15C, fry @ 20C	reduced	156
0.011	pH 8.0, 10.3C	28-d EC	636
0.011	pH 8.1, 14.8C	28-d EC	636
0.011	pH 7.9, 20.1C	28-d EC	636
0.012	pH 7.5, 12.5 C, number of viable oocytes	NOEL	146
0.018	77d fry, pH 7.2, 10C, hardness 50 mg/L	96-h LC_{50}	148

0.019	growth effects	reduced	259
0.019	juvenile mortality	LC	259
0.02	biomass, eggs and alevins @ 5C, fry @ 12C	reduced	156
0.02	96-h enzymatic effects	EC	249
0.021	pH 7.8, 10C	30-d EC	140
0.022	pH 7.5, 12.5 C, number of viable oocytes	18 -d EC	146

Table 8.2.8.7 (continued)

mg/L	species and conditions	effect	ref.
0.026	tissue level after 24-h exposure	increases	134
0.026	tissue level after 24-h exposure	increases	141
0.028	5.4C, pH 7.8	28-d EC	636
0.028	11.7C, pH 7.9	28-d EC	636
0.028	20-d growth inhibition	11%	629
0.028	20-d growth inhibition	18%	629
0.028	21-d growth inhibition	19%	629
0.028	28-d growth inhibition	12%	629
0.028	38-d growth inhibition	18%	629
0.029	tissue	increases	137
0.029	bile	magnifies	137
0.032	42-d sac fry, pH 7.2, 10C, hardness 50 mg/L	96-h LC_{50}	148
0.034	1 g fish, 6C, pH 7.4, hard. 44 mg/L, static bioassay	96-h LC_{50}	111
0.034	alevins	LC99	259
0.044	pH 5.7, 10C, hardness 4.0 mg/L	96-h LC_{50}	281
0.044	ranges up to 0.092 mg/L, static bioassay	96-h LC_{50}	205

0.046		41-d LC_{100}	629
0.052	11C, pH 7.2-7.5, alkalinity 30-35 mg/L and hardness 40-50 mg/L, static bioassay	96-h LC_{50}	128
0.052	1 g fish, 11C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.052	1 g fish, 11C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.052	fry	96-h LC_{50}	212
0.055	1 g fish, 11C, pH 7.4, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.056		48-h LC_{50}	223
0.069	pH 7.0, 10C, hardness 10.0 mg/L	96-h LC_{50}	281
0.07	20C, pH 8.0, hardness 3.6 mg/L, flow-through bioassay, respiration rate effects	24-h EC75	549
0.075		96-h LC_{50}	619
0.080	pH 7.5, 15C, hardness 42 mg/L	96-h LC_{50}	622
0.085	pH 7.0, 12C, hardness 51.5 mg/L	96-h LC_{50}	281
0.088	physiological effects	32-h LT50	248
0.089	pH 7.0, 12C, hardness 47 mg/L	96-h LC_{50}	281
0.090	pH 7.5, 15C, hardness 42 mg/L	96-h LC_{50}	622
0.092	pH 7.0, 12C, hardness 5 mg/L	96-h LC_{50}	281

0.092		96-h LC_{50}	619
0.093		48-h LC_{50}	248
0.096	pH 7.0, 12C, hardness 47 mg/L	96-h LC_{50}	611

Table 8.2.8.7 (continued)

mg/L	species and conditions	effect	ref.
0.115	pH 7.9, 9.9C	LC_{50}	114
0.115	10C, pH 7.4, hardness 44 mg/L, static bioassay, 2.2 g fish	96-h LC_{50}	111
0.121	10C, pH 7.4, hardness 44 mg/L, static bioassay, yolk fry	96-h LC_{50}	111
0.13	pH 7.5-8.0, 14-15C, lab. static bioassay, 2.7 g fish	96-h LC_{50}	670
0.14	10C, pH 7.4, hardness 44 mg/L, static bioassay, 2.2 g fish	24-h LC_{50}	111
0.15	pH 8.0, 15C, hardness 145 mg/L	96-h LC_{50}	621
0.157		48-h LC_{50}	628
0.16	12C	48-h LC_{50}	629
0.17		not lethal	112
0.18	pH 8.0, 15C, hardness 145 mg/L	96-h LC_{50}	621
0.19	pH 8.0, 15C, hardness 145 mg/L	96-h LC_{50}	621
0.2	fry	96-h	211

		LC_{50}	
0.2		48-h LC_{50}	112
0.213	pH 8.2, 15C, hardness 365 mg/L	96-h LC_{50}	211
0.213	pH 8.2, 15C, hardness 365 mg/L	10-d LC_{50}	211
0.22	pH 8.0, 15C, hardness 145 mg/L	96-h LC_{50}	621
0.23	10C	60-h LC_{50}	629
0.48	pH 7.2, 10C, hard. 50 mg/L, 28-day eggs, late eye	96-h LC_{50}	148
0.924	12.8C	4-h LC_{100}	614
1.3	pH 7.2, 10C, hardness 50 mg/L, 24-hour eggs	96-h LC_{50}	148
3.0	pH 7.2, 10C, hard. 50 mg/L, 14-day eggs, early eye	96-h LC_{50}	148
3.0	pH 7.2, 10C, hardness 50 mg/L, 0-hour eggs	96-h LC_{50}	148
NaPCP			
0.01	3 mg/L O2, fertilization to yolk absorption	LC_{100}	140
0.02	fertilization to yolk absorption, steelhead	some death	140
0.02	5 mg/L O2, fertilization to yolk absorption	LC_{100}	140
0.04	fertilization to yolk absorption, steelhead	LC_{100}	140
0.047	pH 5.7, 10C, laboratory static bioassay,	96-h LC_{50}	281
0.047	fry density 0.5 g/L (ranges up to 0.1 mg/L)	96-h LC_{50}	281

0.05	fertilization to hatch, steelhead	24-h LC_{100}	140
0.05	pH 7.0, 11C, laboratory static bioassay,	96-h LC_{50}	281
0.055	12C, pH 7.4, hardness 44 mg/L, static assay, 1 g	24-h LC_{50}	111
0.055	12C, pH 7.4, hardness 44 mg/L, static assay, 1 g	96-h LC_{50}	111
0.058	6C, pH 7.4, hardness 44 mg/L, static bioassay, 1 g	96-h LC_{50}	111
0.062	6C, pH 7.4, hardness 44 mg/L, static bioassay, 1 g	24-h LC_{50}	111

Table 8.2.8.7 (continued)

mg/L	species and conditions	effect	ref.
0.096	pH 7.0, 12C, laboratory static bioassay,	96-h LC_{50}	281
0.098	pH 7.0, 12C, laboratory static bioassay,	96-h LC_{50}	281
0.1	12.8C, Dowicide G, laboratory static bioassay	24-h NOEL	614
0.106	pH 7.1, 12C, laboratory static bioassay,	96-h LC_{50}	281
0.128	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, yolk sac fry	96-h LC_{50}	111
0.16	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, 1.9 g fish	96-h LC_{50}	111
0.165	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, fry	96-h LC_{50}	111
0.17	18C, Santobrite, laboratory flow-through bioassay, fish 3 to 12 months old	48-h LC_{50}	629

0.173	10C, pH 7.4, hardness 272 mg/L, flow -through bioassay, 1.9 g fish	24-h LC_{50}	111
0.2	5 day old steelhead alevins	24-h LC_{100}	140
0.25	10C, Santobrite, laboratory static bioassay, yearlings	60-h LC_{50}	629
0.28	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, yolk sac fry	24-h LC_{50}	111
>0.3	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, eyed eggs	24-h LC_{50}	111
>0.3	10C, pH 7.4, hardness 272 mg/L, flow -through bioassay, eyed eggs	96-h LC_{50}	111
0.3	fertilized steelhead eggs	1-wk LC_{100}	140
0.31	10C, pH 7.4, hardness 272 mg/L, flow-through bioassay, fry	24-h LC_{50}	111
1.0	12.8C, Dowicide G, laboratory static bioassay	4-h LC_{100}	614
5.0	12.8C, Dowicide G, laboratory static bioassay	1-h LC_{100}	614

Table 8.2.8.8 The Effects of Chlorophenols on Fish...*Poecilia* (*Lebistes*) *reticulata* (guppies).

mg/L	Conditions	Effect	Ref.
2-MCP			
3	21C, pH 7.3	48-h LC_0	158
7.1	pH 6.1	14-d LC_{50}	363
11.2	pH 7.3	14-d LC_{50}	363

12	static bioassay	96-h LC_{50}	351
13.5	pH 7.8	14-d LC_{50}	363
13.8	pH 7.0, 26C	96-h LC_{50}	144
20.2	static bioassay	96-h LC_{50}	364
20.2	pH 7.5, lab. static bioassay, 25C, hardness20 mg/L	96-h LC_{50}	389
3-MCP			
6.4	pH 6.1	14-d LC_{50}	363
6.4	pH 7.3	14-d LC_{50}	363
7.9	pH 7.8	14-d LC_{50}	363
4-DCP			
6.3	26C, pH 5, hardness 90 mg/L	96-h LC_{50}	144
7.7	pH 6	96-h LC_{50}	144
7.84	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	144
8.49	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	144
9.0	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	144
2,4-DCP			
3.3	pH 3.3	14-d LC_{50}	363
3.3	pH 6.1	14-d LC_{50}	363
3.5	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	144
4.2	pH 7.3	14-d LC_{50}	363
5.5	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	144
5.9	pH 5.9	14-d LC_{50}	363
5.9	pH 7.8	14-d LC_{50}	363
7.6	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	144
2,6-DCP			
3.9	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	144
7.8	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	144
17.86	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	144

2,3,5-TCP			
0.882	pH 6.1	24-h LC_{50}	363
1.57	pH 7.3	24-h LC_{50}	363
4.74	pH 7.8	24-h LC_{50}	363

Table 8.2.8.8. (continued)

mg/L	Conditions	Effect	Ref.
2,4,5-TCP			
0.987	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	144
0.987	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	616
1.244	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	144
1.244	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	616
3.060	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	144
3.060	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	616
2,4,6-TCP			
0.61	26C, pH 5, hardness 90 mg/L	96-h LC_{50}	144
0.61	26C, pH 5, hardness 90 mg/L	96-h LC_{50}	616
0.889	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	144
0.889	26C, pH 6, hardness 90 mg/L	96-h LC_{50}	616
2.271	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	144
2.271	26C, pH 7, hardness 90 mg/L	96-h LC_{50}	616
7.859	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	144
7.859	26C, pH 8, hardness 90 mg/L	96-h LC_{50}	616
3,4,5-TCP			
1.14	minimum recorded	7-d LC_{50}	419
2.37	minimum recorded	7-d LC_{50}	419
2,3,4,5-TTCP			
0.442	pH 6.1	24-h LC_{50}	363

0.77	pH 7.3	24-h LC_{50}	363
2.32	pH 7.8	24-h LC_{50}	363
2,3,4,6-TTCP			
0.348	pH 6, 26C, hardness 90 mg/L	96-h LC_{50}	144
1.090	pH 7, 26C, hardness 90 mg/L	96-h LC_{50}	144
3.665	pH 8, 26C, hardness 90 mg/L	96-h LC_{50}	144
2,3,5,6-TTCP			
0.39	pH 6.1	24-h LC_{50}	363
1.37	pH 7.3	24-h LC_{50}	363
3.94	pH 7.8	24-h LC_{50}	363
PCP			
0.043	26C, pH 5.0, hardness 90 mg/L	96-h LC_{50}	616
0.107	26C, pH 6.0, hardness 90 mg/L	96-h LC_{50}	144
0.117	26C, pH 6.0, hardness 90 mg/L	96-h LC_{50}	616
0.217	flow-through bioassay	96-h LC_{50}	205
0.33	26C, hardness 80 mg/L	24-h LC40	168

Table 8.2.8.8. (continued)

mg/L	Conditions	Effect	Ref.
0.42		48-h LC40	223
0.442	26C, pH 7.0, hardness 90 mg/L	96-h LC_{50}	616
0.45	hard water, pH 8.0	96-h LC_{50}	85
0.453	26C, pH 7.0, hardness 90 mg/L	96-h LC_{50}	144
0.462	hardness 165 mg/L, pH 8.5	90-d LC_{45}	631
0.62		LC_0	112
0.72	pH 8.0, hard water	96-h LC_{50}	85

0.82	pH 8.0, hard water	48-h LC_{50}	85
0.85		48-h LC_{50}	112
0.88	pH 8.0, hard water	96-h LC_{50}	85
0.906	26C, pH 8.0, hardness 90 mg/L	96-h LC_{50}	144
0.911	26C, pH 8.0, hardness 90 mg/L	96-h LC_{50}	616
0.92		LC_{100}	166
0.924	26C, pH 5.9, hardness 80 mg/L	30-m LC_{50}	168
0.924	26C, pH 7.5, hardness 80 mg/L	80-m LC_{50}	168
0.924	26C, pH 8.9, hardness 80 mg/L	24-h LC_{50}	168
1.05	pH 8.0, hard water	48-h LC_{50}	85
1.80		24-h LC_{94}	630
3.7		3.3-h LC_{100}	630
7.4		1.5-h LC_{100}	630
13.8		40-m LC_{100}	630
23.1		25-m LC_{100}	630
NaPCP			
0.5	laboratory study, static bioassay	40-m LC_{45}	631
1.0	laboratory study	LC_{100}	166
2.0	laboratory study	60-h LC_{94}	630
2.0	24C, pH 7.6-7.8, hardness 358 mg/L, static bioassay, 11% chlorophenols and 79% commercial NaPCP	7-d LC_{50}	670
4.0	laboratory study	3.3-h LC_{100}	630
8.0	laboratory study	1.5-h LC_{100}	630

9.5	field study	LC_{100}	166
15.0	laboratory study	4-m LC_{100}	630
25.0	laboratory study	25-m LC_{45}	630

Table 8.2.8.9 The Effects of Chlorophenols on Fish...*Ictalurus punctatus* (channel catfish).

mg/L	chlorophenol	conditions	effect	ref.
1.07	2,4-DCP	pH 7.8, 22C, hardness 200 mg/L, 4-d old embryos	8-d LC_{20}	376
1.35	2,4-DCP	pH 7.8, 22C, hardness 50 mg/L, 4-day old embryos	8-d LC_{20}	376
1.70	2,4-DCP	pH 7.8, 22C, hardness 200 mg/L, 4-d old embryos	96-h LC_{20}	376
1.85	2,4-DCP	pH 7.8, 22C, hardness 50 mg/L, 4-day old embryos	96-h LC_{20}	376
0.14	2,3,4,6-TTCP	22C, pH 7.0, hardness 40 mg/L, static bioassay, 74% technical grade	24-h LC_{50}	236
0.14	2,3,4,6-TTCP	22C, pH 7.0, hardness 40 mg/L, static bioassay, 74% technical grade	96-h LC_{50}	236
0.066	2,3,4,5,6-PCP	pH 7.4, 20C, hardness 44 mg/L, static bioassay	96-h LC_{50}	111
0.068	2,3,4,5,6-PCP	pH 7.4, 20C, hardness 44 mg/L, static bioassay	24-h LC_{50}	111
0.068	2,3,4,5,6-PCP	20C, pH 7.2-7.5, hardness 40-50 mg/L, alkalinity 30-35 mg/L, static bioassay	96-h LC_{50}	128
0.072	2,3,4,5,6-	pH 7.4, 20C, hardness 44	24-h	111

	PCP	mg/L, static bioassay	LC_{50}	
0.12	2,3,4,5,6-PCP	reagent grade, fingerlings	24-h LD50	669
0.13	2,3,4,5,6-PCP	pH 7.7, 18.6C	LC_{50}	114
0.14	2,3,4,5,6-PCP	commercial grade, fingerlings	24-h LD50	669
0.077	NaPCP	pH 7.4, 20C, hardness 44 mg/L	24-h LC_{50}	111
0.2	NaPCP	pH 7.5, 15C, hard. 272 mg/L, yolk sac fry	24-h LC_{50}	111
0.299	NaPCP	pH 7.4, 15C, hard. 272 mg/L, yolk sac fry	96-h LC_{50}	111
0.46	NaPCP	25C, laboratory static bioassay, fingerlings	24-h LC_{50}	624
0.46	NaPCP	25C, laboratory static bioassay, fingerlings	96-h LC_{50}	624
1.5	NaPCP	25C, laboratory static bioassay, fingerlings	4-h LC_{50}	624
5.4	NaPCP	25C, laboratory static bioassay, fingerlings	1-h LC_{50}	624

Table 8.2.8.10 The Effects of Chlorophenols on Fish...*Salmo* sp.

mg/L	chlorophenol	species and conditions	effect	ref.
		Salmo salar (atlantic salmon)		
0.046	2,3,4,5,6-PCP	6-15C, temperature preference effects	24-h EC	634
0.8	2,3,4,5,6-PCP	6-15C	24-h LC_{50}	634

		Salmo trutta (brown trout)		
1.7	2,4-DCP	5C	24-h LC_{50}	90
5	2,4-DCP	5C	24-h LC_{50}	90
4	2,6-DCP		24-h LC_{50}	419
0.8	2,3,5-TCP	5C, 4 to 5 g fish	24-h LC_{50}	90
0.9	2,4,5-TCP	5C	12-h LC_{50}	90
1.1	2,4,6-TCP	5C	24-h LC_{50}	90
0.5	2,3,4,6-TTCP	5C, 4.5 g fish, static bioassay	24-h LC_{50}	90
0.045	2,3,4,5,6-PCP		96-h LC_{50}	251
0.157	2,3,4,5,6-PCP		48-h LC_{50}	628
0.160	2,3,4,5,6-PCP	6 to 8 cm long, 4 to 6 g fish, metabolic rate	48-h LC_{50}	629
0.17	NaPCP	18C, lab. flow-through bioassay using 'Santobrite'	48-h LC_{50}	629

Table 8.2.8.11 The Effects of Pentachlorophenol on Fish...*Notropis* sp.

mg/L	chlorophenol	species and conditions	effect	ref.
		Notropis cornutus (shiner)		
0.056	2,3,4,5,6-PCP	20C, feeding rate increase	57%	149

0.18	2,3,4,5,6-PCP	20C, feeding rate increase	65%	149
0.18	2,3,4,5,6-PCP	20C, conversion efficiency loss	75%	149
0.18	2,3,4,5,6-PCP	20C, growth reduction	25%	149
0.32	2,3,4,5,6-PCP	20C, survival	LC	149
		Notropis umbratilis (redfin shiner)		
0.2	NaPCP	laboratory static bioassay	3-d LC_0	69
1	NaPCP	laboratory static bioassay	100-m LC_{100}	69
5	NaPCP	laboratory static bioassay	16-m LC_{100}	69
		Notropis spilopterus (spotfin shiner)		
0.36	NaPCP	lab. static bioassay in 'Santobrite'	234-m LC_{50}	632
0.92	NaPCP	lab. static bioassay in 'Santobrite'	74-m LC_{50}	632
4.6	NaPCP	lab. static bioassay in 'Santobrite'	18-m LC_{50}	632
		Notropis whipplii (steelcolour shiner)		
0.2	NaPCP	laboratory static bioassay	3-d LC_0	69
1	NaPCP	laboratory static bioassay	65-m LC_0	69
5	NaPCP	laboratory static bioassay	15-m LC_0	69
		Notropis atherinoides (emerald		

		shiner)		
0.1	NaPCP	lab. static bioassay in 'Dowicide G'	102-h LC_{69}	681
0.2	NaPCP	lab. static bioassay in 'Dowicide G'	LC	681
0.36	NaPCP	lab. static bioassay in 'Santobrite', 19C	418-m LC_{50}	632
0.5	NaPCP	lab. static bioassay in 'Dowicide G'	72-h LC_{50}	681
0.5	NaPCP	lab. static bioassay in 'Dowicide G'	120-h LC_{0}	681
0.92	NaPCP	lab. static bioassay in 'Santobrite', 19C	87-m LC_{50}	632
4.6	NaPCP	lab. static bioassay in 'Santobrite', 19C	16-m LC_{50}	632

Table 8.2.8.12 Summary of the Fish Data from Tables 8.2.8.1 to 8.2.8.11

Chlorophenol congeners	lowest effect levels found in mg/L		references
	chronic	acute (LC_{50}) *	
2-MCP	3.0	2.1	158, 611
3-MCP	10.0	2.9	368, 611
4-MCP	-	3.0	366, 366
2,3-DCP	-	-	-
2,4-DCP	0.07	0.07	220, 376
2,5-DCP	-	-	-
2,6-DCP	5.0	3.9	614, 144
3,4-DCP	-	-	-

3,5-DCP	-	-	-
2,3,4-TCP	-	-	-
2,3,5-TCP	-	0.8	90
2,3,6-TCP	0.72	-	220
2,4,5-TCP	1.0	0.45	614, 390
2,4,6-TCP	0.20	0.32	245, 390
3,4,5-TCP	-	1.14	419
TCPs	1.0	1.0	615, 615
2,3,4,5-TTCP	0.75	0.205	261, 261
2,3,4,6-TTCP	-	0.085	236
2,3,5,6-TTCP	-	0.17	-
2,3,4,5,6-PCP	0.00066	0.018	126, 148, 84
K/NaPCP	0.00174	0.0098	94, 142

* There are two lower reported acute effects than those shown in the table. However this table reports only LC50 data and for 2,3,5- and 2,3,6-TCP reference 220 reports only 'lethal effects' to the bluegill sunfish, ***Lepomis macrochirus***, at 0.45 and 0.32 mg/L, respectively.

Table 8.3.1 Taste Thresholds for Chlorophenols in Fish Muscle.

Chlorophenols	Thresholds in µg/g wet weight (references) -fish species
2-MCP	15 (335, 360)-fish, carp; 60 (190)-trout; 2000 (360)-bluegill
3-MCP	25 (190)-trout; 60 (335, 360)-fish, carp

4-MCP	45 (335, 372, 190)-fish, rainbow, trout
2,3-DCP	84 (190)-trout
2,4-DCP	0.4 (190, 335)-bass, fish; 1 (190)-trout; 14 (190)-bluegill
2,5-DCP	23 (190, 372)-trout, rainbow;
2,6-DCP	35 (190, 372)-trout, rainbow;
2,4,6-TCP	52 (190, 372)-trout, rainbow;
2,3,4,5,6-PCP	20 (190)-fish

Table 8.3.2 Literature Water Levels and Guidelines to Prevent Taste Problems in Fish Flesh.

Chlorophenols	Guideline in µg/L (source-reference)
2-MCP	2 (US EPA, 1980, 1986-694, 695); 15 (Ontario, 1984-220); 60 (US EPA, 1979 -105, 672)
3-MCP	15 (Ontario, 1984-220)
4-MCP	15 (Ontario, 1984-220); 45 (US EPA, 1979-105, 672)
2,4-DCP	0.4 (Ontario, 1984-220; US EPA, 1979-105, 672)
2,3,6-DCP	52 (Ontario, 1984-220)
2,4,6-DCP	52 (US EPA, 1979-105, 672)
MCPs	7 (Ontario, 1984-220)
DCPs	0.2 (Ontario, 1984-220)
the sum of TCPs, TTCPs and PCP	0.1 (British Columbia objective, 1985-536)
Chlorophenols	Threshold Water Levels in µg/L

	(reference)
2-MCP	0.1 (675), 15(676), 60 (364), 2000 (360, 364)
3-MCP	50-60 (361, 370)
4-MCP	5 (676)

Table 8.4.1 Literature Guidelines or Objectives for the Protection of Aquatic Life from Chlorophenols.

µg/L	Source, Type of Guideline/Objective, Conditions	References
	Sum of all Monochlorophenols	
7.0	CCREM-1987, Ontario-1984, freshwater life, *	265, 220
	Sum of all Dichlorophenols	
0.2	CCREM-1987, Ontario-1984, freshwater life, *	265, 220
	Sum of all Trichlorophenols	
18	CCREM-1987, Ontario-1984, freshwater life, *	265, 220
	Sum of all Tetrachlorophenols	
1.0	CCREM-1987, Ontario-1984, freshwater life, *	265, 220
	Sum of Tri-,Tetra- and Penta-Chlorophenols	
0.2	B.C.-1985, freshwater life, site specific objective	536
	Sum of all Chlorophenols	
6.0	B.C.-1989, freshwater life, fish	404

	toxicity, effluent **	
	2-MCP	
180	US EPA-1979, freshwater invertebrates, acute *	105, 672
290	US EPA-1979, freshwater fish, chronic criterion	105, 672
1800	US EPA-1979, freshwater fish, acute criterion	105, 672
2000	USA-1983, freshwater, chronic effects criterion	91
4380	USA-1983, 1980, 1986, 1987, freshwater acute effects, lowest observed effect	91, 694, 695, 696
50000	US EPA-1979, freshwater plants, criterion	105, 672
	4-MCP	
180	US EPA-1979, freshwater invertebrates, acute *	105, 672
510	US EPA-1979, marine invertebrates, acute *	105, 672
540	US EPA-1979, freshwater fish, acute criterion	105, 672
790	US EPA-1979, marine fish, acute criterion	105, 672
3300	US EPA-1979, marine plants, acute criterion	105, 672
4800	US EPA-1979, freshwater plants, acute criterion	105, 672
29700	USA-1983, 1980, 1986, 1987, marine water, acute *	91, 694, 695, 696
	2,4-DCP	
110	US EPA-1979, freshwater	105, 672

	invertebrates, acute *	
365	USA-1983, 1980, 1986, 1987, freshwater, chronic *	91, 694, 695, 696
770	US EPA-1979, freshwater fish, acute criterion	105, 672
2020	USA-1983, 1980, 1986, 1987, freshwater, acute *	91, 694, 695, 696
5000	US EPA-1979, freshwater plants, acute criterion	105, 672

* guideline; ** objective.

Table 8.4.1 (continued)

µg/L	Source, Type of Guideline/ Objective, Conditions	References
	2,4,5-DCP	
63	US EPA-1979, freshwater fish, acute criterion	105, 672
66	US EPA-1979, marine invertebrates, acute *	105, 672
110	US EPA-1979, freshwater invertebrates, acute *	105, 672
250	US EPA-1979, marine fish, acute criterion	105, 672
890	US EPA-1979, marine plants, criterion	105, 672
1200	US EPA-1979, freshwater plants, criterion	105, 672
	2,4,6-TCP	
150	US EPA-1979, freshwater fish, acute criterion	105, 672

240	US EPA-1979, freshwater invertebrates, acute *	105, 672
5900	US EPA-1979, freshwater plants, criterion	105, 672
	2,3,4,6-TTCP	
12	US EPA-1979, freshwater invertebrates, acute *	105, 672
20	US EPA-1979, freshwater fish, acute criterion	105, 672
600	US EPA-1979, freshwater plants, criterion	105, 672
	2,3,5,6-TTCP	
24	US EPA-1979, freshwater fish, acute criterion	105, 672
32	US EPA-1979, freshwater invertebrates, acute *	105, 672
280	US EPA-1979, marine fish, acute criterion	105, 672
380	US EPA-1979, marine invertebrates, criterion	105, 672
440	US EPA-1979, marine plants, criterion	105, 672
2700	US EPA-1979, freshwater plants, criterion	105, 672
	2,3,4,5,6-PCP	
0.4	IJC-1980, freshwater life, guideline	258
0.5	Sask.-1988, Dalela *et al.* -1979, freshwater life; Ont.-1984, acute toxicity; CCREM-1987, chronic.	398, 108, 220, 265
3.2	USA-1983, 1980, 1986, 1987, freshwater chronic *	91, 694, 695, 696
6.2	US EPA-1979, freshwater 24 -h	105

	mean, criterion	
7.5	US EPA-1979, freshwater plants, criterion	105, 672
7.9	US EPA-1986, marine 4-d mean, once in 3 years *	266, 326, 695
8.5	US EPA-1979, marine invertebrates, acute *	105, 672
9.6	US EPA-1979, marine fish, chronic criterion	105, 672
10.1	Gupta-1983, freshwater life, guideline	107
13	US EPA-1986, marine 1-h mean, once in 3 years *	266, 326, 695
14	US EPA-1979, freshwater invertebrates, acute *	105, 672
14	US EPA-1979, freshwater life, maximum level *	105
25	US EPA-1979, freshwater and marine fish, acute *	105, 672
34	USA-1983, 1980, 1986, 1987, marine, chronic *	91, 694, 695, 696
53	USA-1983, 1980, 1986, 1987, marine water, acute *	91, 694, 695, 696
55	USA-1983, 1980, 1986, 1987, freshwater, acute *	91, 694, 695, 696
200	US EPA-1989, maximum contaminant level	359
290	US EPA-1979, marine plants, criterion	105, 672

Table 8.5.1 Raw Aquatic Life Toxicity Guidelines for Chlorophenols in µg/L at 10C.

chlorophenol	pH	pH	pH	pH	pH	pH	pH
congeners	5.6	5.8	6.0	6.2	6.4	6.6	6.8
2-MCP	6.5	6.5	6.5	6.5	6.5	6.5	6.5
3-MCP	4.5	4.5	4.6	4.6	4.6	4.6	4.7
4-MCP	6.1	6.1	6.1	6.2	6.2	6.2	6.2
2,3-DCP	1.1	1.1	1.1	1.2	1.2	1.3	1.3
2,4-DCP	1.4	1.4	1.5	1.5	1.5	1.6	1.7
2,5-DCP	1.1	1.1	1.1	1.2	1.2	1.3	1.4
2,6-DCP	0.2	0.2	0.2	0.2	0.2	0.2	0.2
3,4-DCP	1.1	1.1	1.1	1.2	1.2	1.3	1.3
3,5-DCP	0.6	0.6	0.6	0.7	0.7	0.8	0.8
2,3,4-TCP	0.5	0.5	0.5	0.6	0.6	0.7	0.8
2,3,5-TCP	0.4	0.4	0.5	0.5	0.5	0.6	0.7
2,3,6-TCP	0.3	0.4	0.4	0.5	0.6	0.7	0.8
2,4,5-TCP	0.4	0.5	0.5	0.5	0.5	0.6	0.7
2,4,6-TCP	0.6	0.6	0.6	0.7	0.8	0.1	0.1
3,4,5-TCP	0.3	0.3	0.3	0.3	0.3	0.4	0.4
2,3,4,5-TTCP	0.2	0.2	0.2	0.2	0.3	0.3	0.4
2,3,4,6-TTCP	0.2	0.2	0.3	0.3	0.4	0.5	0.6
2,3,5,6-TTCP	0.1	0.1	0.1	0.2	0.2	0.3	0.3
2,3,4,5,6-PCP	0.1	0.1	0.2	0.2	0.3	0.3	0.4

The table values are for 10C; conversion is a factor of 2 for every 10C.
Table values should be divided by 2 at 20C and multiplied by 2 at 0C.
The conversion formula is:

Guidelines @ Tnew = Guidelines @ T10 (1/2x) , where x=(Tnew -10)/10.

Table 8.5.1 (continued)

chlorophenol	pH	pH	pH	pH	pH	pH	pH	pH
congeners	7.0	7.2	7.4	7.6	7.8	8.0	8.2	8.4
2-MCP	7.0	7.1	7.5	7.8	8.4	9.0	9.8	10.8
3-MCP	4.7	4.8	4.9	5.0	5.3	5.5	5.8	6.2
4-MCP	6.4	6.4	6.5	6.7	6.8	7.0	7.3	7.6
2,3-DCP	1.5	1.6	1.8	2.2	2.4	2.8	3.3	4.1
2,4-DCP	1.8	1.9	2.1	2.3	2.7	3.1	3.6	4.3
2,5-DCP	1.5	1.6	1.8	2.1	2.5	3.0	3.6	4.4
2,6-DCP	2.8	3.3	3.8	4.7	5.8	7.2	9.0	11.6
3,4-DCP	1.5	1.6	1.8	2.0	2.4	2.8	3.3	4.1
3,5-DCP	0.9	1.1	1.3	1.5	1.8	2.3	2.8	3.6
2,3,4-TCP	0.9	1.0	1.2	1.5	1.8	2.3	2.9	3.7
2,3,5-TCP	0.8	0.9	1.1	1.3	1.6	2.0	2.5	3.1
2,3,6-TCP	1.0	1.3	1.6	2.1	2.7	3.5	4.5	6.0
2,4,5-TCP	0.7	0.8	1.0	1.2	1.4	1.8	2.2	2.8
2,4,6-TCP	1.5	1.8	2.3	2.9	3.7	4.8	6.1	7.9
3,4,5-TCP	0.4	0.4	0.5	0.5	0.6	0.8	0.8	1.0
2,3,4,5-TTCP	0.5	0.6	0.8	1.0	1.3	1.6	2.1	2.7
2,3,4,6-TTCP	0.8	1.0	1.3	1.7	2.2	2.9	3.8	5.0
2,3,5,6-TTCP	0.4	0.6	0.8	1.0	1.3	1.7	2.2	2.9
2,3,4,5,6-PCP	0.6	0.7	1.0	1.3	1.7	2.2	2.9	3.8

The table values are for 10C; conversion is a factor of 2 for every 10C.
Table values should be divided by 2 at 20C and multiplied by 2 at 0C.
The conversion formula is:

Guidelines @ Tnew = Guidelines @ T10 (1/2x) , where x=(Tnew -10)/10.

Table 8.5.1.1 Constants used in Calculating Raw Aquatic Life Toxicity Guidelines for Chlorophenols at different pH values.

chlorophenol	constants				
congeners	log P (Ko/w)	pKa	10.05-pKa	C	GMW
2-MCP	2.15	8.65	1.40	1.04	129
3-MCP	2.50	9.10	0.95	1.19	129
4-MCP	2.39	9.37	0.68	1.06	129
2,3-DCP	3.19	7.60	2.45	1.94	163
2,4-DCP	3.08	7.85	2.20	1.81	163
2,5-DCP	3.20	7.50	2.55	1.95	163
2,6-DCP	2.84	6.91	3.14	1.83	163
3,4-DCP	3.20	7.60	2.45	1.94	163
3,5-DCP	3.50	7.10	2.95	2.24	163
2,3,4-TCP	3.70	6.90	3.15	2.41	197
2,3,5-TCP	3.80	6.90	3.15	2.48	197
2,3,6-TCP	3.90	6.00	4.05	2.71	197
2,4,5-TCP	3.80	7.07	2.98	2.44	197
2,4,6-TCP	3.60	6.20	3.85	2.47	197
3,4,5-TCP	4.20	7.70	2.35	2.59	197
2,3,4,5-TTCP	4.40	6.20	3.85	3.01	232
2,3,4,6-	4.45	5.46	4.59	3.18	232

TTCP					
2,3,5,6-TTCP	4.90	5.30	4.75	3.51	232
2,3,4,5,6-PCP	5.15	4.71	5.34	3.80	266

The basic equation from reference 144 is:

$$\log(1/LC_{50}pH) = \log(1/LC_{50}HA)-\log(4pH-pKa+1) .$$

The term $\log(1/LC_{50}HA)$ is a constant, C, for each chlorophenol and does not change with pH. Rearranging to solve for the constant un-ionized term, which is a function of the chemical properties of the chlorophenol, gives:

$$\log(1/LC_{50}pH) +\log(4pH-pKa+1)=C.$$

Substituting experimental values into this equation and regressing against the properties of the chlorophenols leads to the expression:

$$C= 0.67 \log P+ 0.19 (10.05-pKa)-0.67.$$

Using this expression one can fill in values for C for each chlorophenol in the table above.
Table 8.5.1.1 (continued)

One can now calculate the LC_{50} for guppies at any pH within the useful range of this equation, using as a working equation:

$$\log A = C-B \text{ ; where } A = 1/LC_{50} \text{ at the new pH}$$

and

$$B = \log(4pH-pKa+1) \text{ at the new pH.}$$

Since the equation returns LC_{50} values in millimoles and not mg/L, the answer needs to be multiplied by the molecular weight of the chlorophenol.

The guppy data is at 26C and to convert it to 10C one needs to apply the temperature conversion equation:

Guideline @10 = guideline@26/ (1/2x) where x = (26-10)/10.

This works out to 0.33 so all the guppy guidelines are divided by 0.33. This results in a guppy LC_{50} value at 10C and pH 7.2 for PCP, of 4.18 mg/L, which can be compared to the rainbow trout value under the same conditions of 0.018 mg/L; guppy LC_{50} values in mg/L need to be multiplied by 4.31 to give rainbow LC_{50} values in µg/L. To convert these LC_{50} values to guidelines they need to be multiplied by 0.041which is the LC_{50}-to-chronic no effect threshold conversion. The net conversion factor for guppy LC_{50} values at 36C in mg/L, to aquatic life guidelines at 10C, in µg/L, is: 0.54 or (4.31 x 0.041) /0.33.

Table 8.5.2 Recommended Chlorophenol Guidelines to Prevent Tainting of Fish Muscle.

chlorophenols	Recommended Water Guidelines in µg/L.
MCPs	0.1
DCPs	0.2
TCPs, TTCPs and PCP	use aquatic life toxicity guidelines
chlorophenols	Fish Muscle Concentration in µg/g wet weight.
2-MCP	10
3-MCP	20
4-MCP	40
2,3-DCP	80
2,4-DCP	0.2
2,5-DCP	20
2,6-DCP	30

2,4,6-TCP	50
2,3,4,5,6-PCP	20

Table 8.6.1 Calculated or Experimental No Observed Effect and Non-Lethal Levels of Chlorophenols to Freshwater Life .

µg/L	Organisms and Conditions	Effect	Ref.
	2-MCP		
1000	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
3000	***Poecilia reticulata***	LC_0	158
3900	***Pimephales promelas***, larval embryonic stages, chronic	NOEL	364
5000	***Idus idus melanotis,*** pH 7-8	48-h LC_0	366
10000	***Daphnia magna***, pH 8.0, mobility	NOEL	384
10000	***Chlorella pyrenoidosa***, 25C, static bioassay,	NOEL	284
10000	***Carassius auratus***	48-h LC_0	273
	3-MCP		
10	***Cyprinus carpio***, egg	NOEL	368
1000	***Cyprinus carpio***, adults	NOEL	368
1000	***Idus idus melanotis***, pH 7-8	48-h LC_0	366
10000	***Chlorella pyrenoidosa***, 25C, 5% CO2, pH 7.0, oxygen evolution and photosynthesis	3-d NOEL	284
	4-MCP		
320	***Phaeodactylum tricornutum***, pH 7.8, growth	NOEL	371

1000	***Daphnia magna***, mobility assay	14-d NOEL	371
1100	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
2000	***Idus idus melanotis***, pH 7-8	48-h LC_0	366
3200	***Scenedesmus pannonicus***, pH 7.8, growth	NOEL	371
3200	***Cyprinodon variegatus***	96-h LC_0	393
3200	***Brachydanio rerio***	96-h LC_0	371
	2,4-DCP		
290-460	***Pimephales promelas***	NOEL	495
290-460	***Pimephales promelas***, 4 week old larvae	LC_0	377
360	***Pimephales promelas***, pH 7.2-7.9, embryo to 28 days	28-d LC_0	377
380	***Pimephales promelas,***		
460	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
1000	***Leuciscus idus / Idus idus melanotis***	48-h LC_0	395
2800	***Daphnia magna***, pH 8.0, mobility	24-h EC_0	384
3000	***Brachydanio rerio***	96-h LC_0	396
7400	***Pimephales promelas***, pH 9.1, 25C, hardness 46 mg/L	8-d LC_0	375
8000	***Chlorella pyrenoidosa***, 18C, growth	14-d NOEL	3

Table 8.6.1 (continued)

µg/L	Organisms and Conditions	Effect	Ref.
	2,4,5-TCP		

100	***Oncorhynchus mykiss***, 12.8C, (Dowicide-2)	24-h NOEL	614
780	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
1000	***Lepomis macrochirus*** (Dowicide-2)	24-h NOEL	614
1000	***Petromyzon marinus***	24-h NOEL	614
1000	***Chlorella pyrenoidosa***, chlorophyll	72-h NOEL	284
	2,4,6-TCP		
100	***Chlorella pyrenoidosa***, 18C, growth	NOEL	3
410	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
530	***Pimephales promelas***, lowest tested value rated as	NOEL	372
970	***Pimephales promelas***, highest tested value rated as	NOEL	372
1000	***Chlorella pyrenoidosa***, 25C, 5% CO2, photosynthesis	72-h EC_0	463
	2,3,4,6-TTCP		
10	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
50	***Daphnia magna***, pH 7.5, 17C, hardness 44 mg/L	28-d NOEL	236
	2,3,5,6-TTCP		
10	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173mg/L	NOEL	56
	2,3,4,5,6-PCP/NaPCP		
0.035	***Oncorhynchus mykiss***, 15C	NOEL	126
0.43	***Notopterus notopterus***, 36C	NOEL	142

1.1	***Notopterus notopterus***, 4.5 cm long, 23C, pH 7.2	NOEL	132
2	***Chlorella variegata***	NOEL	330
2	***Gomphonema parvulum***	3-d NOEL	330
3.0	***Notopterus notopterus***, 22.6 cm long, 23C, pH 7.2	NOEL	132
3.5	***Notopterus notopterus***, 9 cm long, 23C, pH 7.2	NOEL	132
3.7	***Notopterus notopterus***,16C	NOEL	142
4.5	***Notopterus notopterus***,14.5 cm long, 23C, pH 7.2	NOEL	132
10.0	***Rasbora daniconius neilgeriensis***, pH 7.8, 31C, hardness 215 mg/L, alkal. 215 mg/L, DO 6.4 mg/L	NOEL	107
32	***Hydra oligactis***	21-d NOEL	333
50.0	***Lymnaea stagnalis***, eggs and young, survival, 20C	NOEL	85
100	***Oncorhynchus mykiss***, 12.8C, (Dowicide G)	24-h NOEL	614
100	***Lepomis humilis***	3-d LC_0	69
100	***Lemna perpusilla***, 27C, growth	14-d NOEL	3
111	***Physa gyrina***, 29.6C, pH 7.2-8.9, hard. 126-168 mg/L	NOEL	102
124	***Lepomis macrochirus***	LC_0	102
124	***Pimephales promelas***	LC_0	102
130	***Amblystoma mexicanum***, 20C	LC_0	112
130	***Amblystoma mexicanum***, 20C	LC_0	551

Table 8.6.1 (continued)

µg/L	Organisms and Conditions	Effect	Ref.
160	***Daphnia magna***, 19C	EC_0	112
170	***Oncorhynchus mykiss***	LC_0	112
170	***Pimephales promelas***	LC_0	112
180	***Daphnia magna***, chronic test	NOEL	84
180	***Daphnia magna***, chronic test	NOEL	93
200	***Notropis umbratilis***	3-d LC_0	69
200	***Notropis whipplii***	3-d LC_0	69
200	***Pimephales notatus***	3-d LC_0	69
200	***Ericymba buccata***	3-d LC_0	69
200	***Campostoma anomblum***	3-d LC_0	69
200	***Daphnia magna***, hardness 90 mg/L, reproduction	NOEL	417
200	***Lymnaea stagnalis***, 20C	NOEL	112
210	***Xenopus laevis***, 20C	LC_0	551
210	***Xenopus laevis***, 20C	LC_0	112
230	***Elodea canadensis***, growth	21-d NOEL	113
320	***Daphnia magna***, pH 7.4-9.4, 22C, hardness 173 mg/L	NOEL	56
340	***Daphnia magna***,1 mm long, reproduction	NOEL	217
340	***Pimephales promelas***, 15C, pH 7.4-7.5, hard. 400 mg/L	24-h LC_0	168
400	***Semotilus atromaculatus***	3-d LC_0	69
400	***Daphnia magna***, hardness 90 mg/L, adult survival	NOEL	417
600	***Rana pipiens***, tadpoles	3-d LC_0	69
600	***Fundulus notatus***	3-d LC_0	69
620	***Poecilia reticulata***	LC_0	112

930	***Oryzias latipes***	LC_0	112
1000	***Notropis whipplii***	65-m LC_0	69
1000	***Campostoma anomblum***	58-m LC_0	69
1000	***Lepomis humilis***	165-m LC_0	69
1200	***Daphnia pulex***, 19C	NOEL	112
1800	***Aedes aegypti***, 26C, 3rd instar	NOEL	112
5000	***Notropis whipplii***	15-m LC_0	69
5000	***Campostoma anomblum***	13-m LC_0	69
5000	***Lepomis humilis***	25-m LC_0	69
5000	***Daphnia pulex***	1-h LC_0	69
9500	***Astacus fluviatilis***	NOEL	166
24000	***Culex pipiens***, 26C, 3rd instar	NOEL	112

Table 8.6.2 The Lowest Literature LC_{50} values for Freshwater Life.

chlorophenol	LC_{50}	Organisms and Conditions	Refs.
congeners	µg/L		
2-MCP	2100	***Oncorhynchus mykiss***, 96-h, pH 7.7, 12C	611
3-MCP	2900	***Oncorhynchus mykiss***, 96-h, pH 7.7, 12C	611
4-MCP	3000	***Idus idus melanotis***, 48-h, pH 7-8	366
2,3-DCP			
2,4-DCP	70	***Oncorhynchus mykiss***, larval, pH 7.8, 27-d	376
2,5-DCP			

2,6-DCP	3900	***Poecilia reticulata***, 26C, pH 6.0	144
3,4-DCP			
3,5-DCP			
2,3,4-TCP			
2,3,5-TCP	800	***Salmo trutta***, 5C, 24-h	90
2,3,6-TCP	5400	***Astacus fluviatilis***, crayfish, 13C, pH 6.5, 8-d	71
2,4,5-TCP	450	***Lepomis macrochirus***, 96-h	372
2,4,6-TCP	320	***Lepomis macrochirus***, 96-h	390
3,4,5-TCP	1140	***Poecilia reticulata***, 7-d	419
2,3,4,5-TTCP	205	***Oncorhynchus mykiss***, rainbow, 96-h, pH 6.9-7.7	261
2,3,4,6-TTCP	85	***Oncorhynchus mykiss***, rainbow, 96-h, pH 7.2, 12C	236
2,3,5,6-TTCP	170	***Lepomis macrochirus***, fry, 96-h, pH 6.7-7.8	390
2,3,4,5,6-PCP	18.0	***Oncorhynchus mykiss***, fry, 96-h, pH 7.2, 10C	148
K/NaPCP	9.8	***Notopterus notopterus***, 36C, pH 7.2, 96-h	142

Table 8.6.3 The Lowest Literature Effect Level for Freshwater Life.

chlorophenol	level	Organisms and Conditions	Refs.
congeners	µg/L		
2-MCP	1350	***Daphnia magna***, 7-d EC_{50}	400
3-MCP	2500	***Skeletonema costatum,*** growth	369
4-MCP	100	***Pontoporeia affinis***, swimming	374

2,3-DCP	4090	***Daphnia magna***, pH 7.8-8.2, 24-h IC_{50}	57
2,4-DCP	70	***Oncorhynchus mykiss***, larval, pH 7.8, 27-d	376
2,5-DCP			
2,6-DCP	3900	***Poecilia reticulata***, 26C, pH 6.0	144
3,4-DCP	2550	***Daphnia magna***, pH 7.8-8.2, 24-h IC_{50}	57
3,5-DCP	1850	***Daphnia magna***, pH 7.8-8.2, 24-h IC_{50}	57
2,3,4-TCP	1100	***Photobacterium phosphoreus***, microtox	240
2,3,5-TCP	450	***Lepomis macrochirus***, toxicity	220
2,3,6-TCP	320	***Lepomis macrochirus***, acute toxicity	220
2,4,5-TCP	550	***Daphnia carinata***, 24-h EC_{50}	237
2,4,6-TCP	200	***Oncorhynchus mykiss***, enzyme effects, 96-h	245
3,4,5-TCP	820	***Daphnia magna***, pH 7.8-8.2, 24-h IC_{50}	57
2,3,4,5-TTCP	176	***Photobacterium phosphoreus***, microtox	367
2,3,4,6-TTCP	71	***Oncorhynchus mykiss***, bile changes	137
2,3,5,6-TTCP	170	***Lepomis macrochirus***, acute toxicity	220
2,3,4,5,6-PCP	0.66	***Oncorhynchus mykiss***, fry, 15C, reduced growth	126

Table 8.6.4 The Lowest Literature LC_{50} values for Marine Life.

chlorophenol	LC_{50}	Organisms and Conditions	Refs.
congeners	µg/L		
2-MCP	5300	***Crangon septemspinosa***, shrimp, 10C, 96-h	386
3-MCP			
4-MCP	4100	***Chaetogammarus marinus***, 96-h	371
2,3-DCP			
2,4-DCP	1490	***Paleamonetes pugio***, molting, 96-h	130
2,5-DCP			
2,6-DCP	19100	***Crangon septemspinosa***, shrimp, 10C, 52-h	386
3,4-DCP			
3,5-DCP	1500	***Crangon septemspinosa***, shrimp, 10C, 96-h	386
2,3,4-TCP	2000	***Crangon septemspinosa***, shrimp, 10C, 96-h	386
2,3,5-TCP			
2,3,6-TCP	2700	***Crangon septemspinosa***, shrimp, 10C, 96-h	
2,4,5-TCP	360	***Paleamonetes pugio***, molting, 96-h	130
2,4,6-TCP	1110	***Paleamonetes pugio***, molting, 96-h	130
3,4,5-TCP			
2,3,4,5-TTCP	350	***Paleamonetes pugio***, molting, 96-h	130
2,3,4,6-	500	***Oncorhynchus***, salmon, 96-h	90

TTCP			
2,3,5,6-TTCP	1080	***Paleamonetes pugio***, molting, 96-h	130
2,3,4,5,6-PCP	38	***Lagodon rhomboides***, pinfish, 96-h	119
K/NaPCP	40	***Crassostrea virginica***, oyster, 96-h	78

Table 8.6.5 The Lowest Literature Effect Level for Marine Life .

chlorophenol	level	Organisms and Conditions	Refs.
congeners	µg/L		
2-MCP	5300	***Crangon septemspinosa***, shrimp,10C, 96-h LC_{50}	386
3-MCP	2500	***Skeletonema costatum***, diatom, growth reduction	369
4-MCP	100	***Pontoporeia affinis***, amphipod, swimming	374
2,3-DCP			
2,4-DCP	500	***Entosiphon sulcatum***, pH 6.9, toxic	42
2,5-DCP			
2,6-DCP	19100	***Crangon septemspinosa***, shrimp, 10C, 52-h LC_{50}	386
3,4-DCP			
3,5-DCP	1500	***Crangon septemspinosa***, shrimp,10C, 96-h LC_{50}	386
2,3,4-TCP	2000	***Crangon septemspinosa***, shrimp,10C, 96-h LC_{50}	386
2,3,5-TCP			

2,3,6-TCP	2700	***Crangon septemspinosa***, shrimp, 10C, 96-h LC_{50}	386
2,4,5-TCP	360	***Paleamonetes pugio***, molting, 96-h LC_{50}	130
2,4,6-TCP	1110	***Paleamonetes pugio***, molting, 96-h LC_{50}	130
3,4,5-TCP			
2,3,4,5-TTCP	350	***Paleamonetes pugio***, molting,96h LC_{50}	130
2,3,4,6-TTCP	500	***Oncorhynchus***, salmon, 96-h LC_{50}	90
2,3,5,6-TTCP	1080	***Paleamonetes pugio***, molting, 96-h LC_{50}	130
2,3,4,5,6-PCP	1.6	***Oncorhynchus nerka***, sockeye, larva, growth, 15C	94
K/NaPCP	27	***Crassostrea gigas***, oyster, embryo growth, 48-h $EC_{4.3}$	120

Table 8.6.6 Chlorophenol Guidelines Calculations in µg/L.

chlorophenol congeners	Col. 1	Col. 2	Col. 3	Col. 4	Col. 5	Col. 6
2-MCP	23.6	424.8	17.4	174.4	17.4	7.1
3-MCP	20.8	374.4	15.4	153.7	15.4	4.8
4-MCP	10.6	190.8	7.8	78.3	7.8	6.4
2,3-DCP	6.83	122.4	5.1	50.5	5.1	1.6
2,4-DCP	3.53	63.6	2.6	26.1	2.6	1.9
2,5-DCP	3.04 *	54.7	2.3	22.5	2.3	1.6
2,6-DCP	12.3	222.0	9.1	90.9	9.1	3.3

3,4-DCP	3.64	65.5	2.7	26.9	2.7	1.6
3,5-DCP	2.75	49.8	2.0	20.3	2.0	1.1
2,3,4-TCP	2.95	53.2	2.2	21.8	2.2	1.0
2,3,5-TCP	3.00	54.0	2.2	22.2	2.2	0.9
2,3,6-TCP	9.71	175.2	7.2	71.8	7.2	1.3
2,4,5-TCP	2.74	49.3	2.0	20.2	2.0	0.8
2,4,6-TCP	7.20	129.6	5.3	53.2	5.3	1.8
3,4,5-TCP	1.16	20.9	0.9	8.6	0.9	0.4
2,3,4,5-TTCP	2.32	41.8	1.7	17.1	1.7	0.6
2,3,4,6-TTCP	6.56 *	118.1	4.9	48.5	4.9	1.0
2,3,5,6-TTCP	2.99	53.9	2.2	22.1	2.2	0.6
2,3,4,5,6-PCP	1.00	18.0	0.7	7.4	0.7	0.7

* estimated using ratio data from Saarikoski *et al.* (144) as calculated.

Column 1-Experimental acute toxicity ratios for ***Daphnia magna*** from Devillers *et al.* (57); chlorophenol value divided by the PCP value.
Column 2-Calculated lowest LC_{50} value; the ratio from column 1, multiplied by the lowest rainbow trout PCP LC_{50} of 18 µg/L from Leeuwen *et al.* (148): 10C, pH 7.2, hardness 50 mg/L and 96 hours exposure.
Column 3-Calculated mean NOEL threshold, based on rainbow trout LC_{50} datum. It is the LC_{50} data of column 2, multiplied by 0.041, an acute-to-mean NOEL threshold factor.
Column 4-The LOEL for PCP from Webb and Brett (94) corrected first to pH 7.2, then to 10°C, and multiplied by the ratio in Column 1.
Column 5-The guidelines developed from Column 4 by multiplying by a factor of 0.1 to get an estimate of the NOEL from the chronic LOEL.
Column 6-The equivalent guidelines derived using the equations in Saarikoski *et al.* (144) for guppies, corrected to pH 7.2 and 10C.

(Columns 3 and 5 are identical; calculating the NOEL from acute or from chronic data gives the same result. No safety factor is applied to an NOEL. Column 6 is very similar).

The equations from Saarikoski *et al.* (144) underestimate and overestimate, respectively, the toxicity when the pH is greater than or less than the pKa; otherwise the agreement between the two acute data methods is as good as could be expected. Since the guidelines derived from Devillers (57) can not be extended to other pH values, the equations of Saarikoski *et al.* (144) need to be used if pH corrections are desired. A more practical solution is to use the EPA pH conversion equation for PCP. Inspection of Table 8.5.1 shows that corrections for pH are sometimes desirable since there is considerable variation with pH for some congeners. For rainbow trout, guidelines can be derived by multiplying the lowest LC_{50} of 18 µg/L by an acute-to-mean NOEL ratio of 0.041. One could also apply a safety factor of 0.1 to the lowest chronic response of 3.49 µg/L for salmonid fry, Webb and Brett (94). All these methods give the same result for PCP, 0.7 µg/L. The final guidelines in Column 5 were calculated from the LOEL data of Webb and Brett and corrected to pH 7.2 and a temperature of 10°C, since this paper documented the lowest chronic response found in a primary reference.

Table 8.6.7 The Lowest Values found in the Literature for the Specified Response: in µg/L.

(these are reported numbers and have not been corrected to 10C and pH 7.2)

Chlorophenol Congeners	Lethal Effects	Reproduction Growth and Development Effects	Mobility and Behavior Effects	Photosynthesis Respiration Microtox and Enzyme Effects	Proposed Toxicity Guideline @ 10C & pH 7.2
2-MCP	2100	88700	1350	6800	17
3-MCP	2900	2500	13800	14100	15
4-MCP	3000	300	100	178000	7.8

2,3-DCP	-	-	4090	4600	5.1
2,4-DCP	70	500	2480	100	2.6
2,5-DCP	-	25400	-	-	2.3
2,6-DCP	3900	-	8690	-	9.1
3,4-DCP	5000	-	2550	-	2.7
3,5-DCP	1500	-	1850	4150	2.0
2,3,4-TCP	2000	5900	2000	1100	2.2
2,3,5-TCP	450	2960	2060	1110	2.2
2,3,6-TCP	320	-	6250	-	7.2
2,4,5-TCP	360	3200	550	1000	2.0
2,4,6-TCP	100	100	690	5920	5.3
3,4,5-TCP	1140	-	820	-	0.9
2,3,4,5-TTCP	205	300	1520	176	1.7
2,3,4,6-TTCP	85	800	-	71	4.9
2,3,5,6-TTCP	170	1400	1870	2720	2.2
2,3,4,5,6-PCP	9.8	3.49	3.49	3.49	0.7

Table 8.6.8 Chlorophenol Guidelines: calculated in µg/L, at 10°C, from pH 5.7 to pH 9.2.

chlorophenol congeners	pH 5.7	pH 6.2	pH 6.7	pH 7.2	pH 7.7	pH 8.2	pH 8.7	pH 9.2
2-MCP	3.9	6.4	11	17	29	48	79	130
3-MCP	3.4	5.6	9.3	15	25	42	70	115
4-MCP	1.7	2.9	4.8	7.8	13	22	36	59
2,3-DCP	1.1	1.8	3.1	5.1	8.3	14	23	38

2,4-DCP	0.6	1.0	1.6	2.6	4,3	7.2	12	20
2,5-DCP	0.5	0.8	1.4	2.3	3.7	6.2	10	17
2,6-DCP	2.0	3.3	5.5	9.1	15	25	41	68
3,4-DCP	0.6	1.0	1.6	2.7	4.4	7.4	12	20
3,5-DCP	0.5	0.7	1.2	2.0	3.4	5.6	9.2	15
2,3,4-TCP	0.5	0.8	1.3	2.2	3.6	6.0	9.9	16
2,3,5-TCP	0.5	0.8	1.3	2.2	3.7	6.1	10	17
2,3,6-TCP	1.6	2.6	4.4	7.2	12	20	33	54
2,4,5-TCP	0.5	0.7	1.2	2.0	3.3	5.6	9.2	15
2,4,6-TCP	1.2	1.9	3.2	5.3	8.8	15	24	40
3,4,5-TCP	0.2	0.3	0.5	0.9	1.4	2.4	3.9	6.4
2,3,4,5-TTCP	0.4	0.6	1.0	1.7	2.8	4.7	7.8	13
2,3,4,6-TTCP	1.1	1.8	2.9	4.9	8.0	13	22	36
2,3,5,6-TTCP	0.5	0.8	1.3	2.2	3.6	6.1	10	17
2,3,4,5,6-PCP	0.2	0.3	0.5	0.7	1.2	2.0	3.4	5.5

Multiply the table values by 2 at 0°C and by 0.5 at 20°C.
The guidelines for PCP are final; those for the other congeners are interim.