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ENVIRONMENTAL CHEMISTRY OF PENTACHLOROPHENOL

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APPLIED CHEMISTRY DIVISION

Commission on Pesticide Chemistry

ENVIRONMENTAL CHEMISTRY OF PENTACHLOROPHENOL

A Special Report on Pentachlorophenol In The Environment

Prepared for publication by D.G. Crosby

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ABSTRACT: Pentachlorophenol (PCP)[a] is a major industrial chemical and a general biocide used throughout the world. PCP, itself, is corrosive and toxic, but it also is readily degraded by sunlight, animals, plants, and soil microorganisms. Several analytical methods -- especially gas chromatography -- have become established which permit routine detection and measurement at environmental levels below $1:10^9$, and PCP residues have been detected widely in food, water, and human urine in approximately the $1:10^8$ to $1:10^{10}$ range (and higher under occupational conditions). However, technical PCP contains many neutral impurities, including hexa-, hepta-, and octachlorodibenzodioxins, chlorinated dibenzofurans, phenoxyphenols, and hexachlorobenzene, which are more slowly metabolized or environmentally degraded and some of which are extremely toxic. Sophisticated analytical methods to detect the presence of the dibenzodioxins and dibenzofurans have not yet received wide application, and evidence of the environmental occurrence of these substances is fragmentary. Although commercial PCP can be partially purified, the toxicity and mobility of both the parent compound and its impurities suggest that indiscriminate use of PCP products and the exposure of humans and domestic animals should be minimized. In addition, PCP occurs as a metabolite of several common pesticides which may contribute substantially to both observed environmental residues and human exposure.

a: The following abbreviations are used throughout the text: HCB (hexachlorobenzene), HCDD (hexachlorodibenzo-p-dioxin), HCDF (hexachlorodibenzofuran), HpCDD (heptachlorodibenzo-p-dioxin), HpCDF (heptachlorodibenzofuran), KPCP (potassium pentachlorophenate), LPCP (pentachlorophenyl laurate), NaPCP (sodium pentachlorophenate), OCDD (octachlorodibenzo-p-dioxin), OCDF (octachlorodibenzofuran), PCDD (pentachlorodibenzo-p-dioxin), PCDF (pentachlorodibenzofuran), PCP (pentachlorophenol), TCDD (tetrachlorodibenzo-p-dioxin), TCDF (tetrachlorodibenzofuran), TCP (tetrachlorophenol), TLV (threshold limit value).

CONTENTS

1. INTRODUCTION

1.1 Pentachlorophenol

Pentachlorophenol (PCP) was introduced in the 1930's as a preservative for timber and lumber. It still is used extensively for that purpose as well as in a wide variety of agricultural and industrial applications as fungicide, bactericide, herbicide, molluscicide, algicide, and insecticide (especially against wood-boring insects). Although the use level has declined somewhat in recent years, present production worldwide is estimated to be about 50,000 metric tons (5×10^7 kg) per year (Section 2).

PCP residues occur widely in man and his environment (Section 6). For example, PCP was detected in over 80% of urine specimens analyzed during a US Environmental Protection Agency survey of both occupationally exposed workers and the general public. Although PCP, itself is among the more toxic chemicals available to the public (Section 1.2), the

technical product contains neutral impurities which could prove to be more hazardous. Certain isomers of hexa- and heptachlorodibenzo-p-dioxin and chlorodibenzofurans have high acute and chronic toxicity (Tables 1.2, 1.3) and their physical and chemical properties differ markedly from those of the phenols (Section 3).

A wide variety of analytical methods have become available for analytical detection and measurement of PCP and its related compounds (Section 5). They reveal that while the chlorophenols are readily susceptible to breakdown through both biological and abiotic processes, the chlorinated impurities generally are more stable in the environment (Sections 3 and 4).

For these reasons, PCP is closely regulated in several nations, and the US Environmental Protection Agency took action in 1978 to limit registered uses in the United States. However, the chemical remains one of the most effective, available, and inexpensive agents against the microbial and insect-caused destruction of property, especially in tropical climates, and the perception of its persistence, human exposure, or environmental hazard by one society can differ markedly from that in others.

The scale and nature of its uses and the increasing controversy over possible effects on human health and the environment justify a review of the environmental chemistry of PCP by the Pesticide Chemistry Commission of IUPAC. The scientific literature on PCP has expanded rapidly -- some 200 Chemical Abstracts citations appeared in 1978 alone -- and several valuable reviews already exist (1-6). The present work does not attempt to repeat this extensive coverage; rather, its purpose is to present a documented overview and interpretation of major aspects of PCP chemistry as they apply to the properties, degradation, environmental fate, detection, and toxic exposure potential of the substance and its principal impurities. Literature citations end with September, 1980.

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1.2 Toxicity and Hazard

PCP is used because it is toxic -- to microorganisms (bactericide and fungicide), lower and higher plants (algicide and herbicide), invertebrate and vertebrate animals (insecticide, molluscicide), but incidentally, also to man. Typical values for its acute toxicity are shown in Table 1.1, but published accounts vary widely, in part because of improving methods and in part because of variability in the composition of the starting material. PCP is absorbed by and corrosive to skin and causes burns and blisters; it is highly irritating to the nose and throat (TLV 0.5 mg/m^3, 0.046 ppm), but the purified material does not cause chloracne. In mammals, acute exposure leads to elevated body temperature, increased respiratory rate, elevated blood pressure, hyperglycemia, and cardiovascular distress (4).

TABLE 1.1. Selected acute toxicity values for PCP[a]

Species	Route	Measure	Toxic Dose (mg/kg)
Rat (M,F)	Oral (peanut oil)	LD_{50}	146
(M)	Dermal (peanut oil)	LD_{50}	320
(M)	Inhalation	LD_{50}	11.7
(M,F)	Intraperitoneal	LD_{50}	420
Mouse	Oral	LD_{50}	130
	Dermal	LD_{50}	261
Rabbit	Oral	LD_{50}	550
Sheep	Oral (Sawdust)	LD_{50}	120
Calf	Oral (Sawdust)	LD_{50}	140
Goldfish (Carassius aureus)	Water[b]	LC_{50}	0.22 mg/L
Coho salmon (Onchorynchus kisutch)	Water[c]	LC_{50}	0.15 mg/L

[a]Ref. 4; [b]NaPCP; [c]KPCP

Long-term dietary exposure of rats to purified PCP produced, at the highest dose (30 mg/kg), only mild biochemical and physical effects which did not alter life span or tumor incidence (7). PCP was not teratogenic or mutagenic in the standard tests but was highly embryotoxic (3,4).

In contrast, several studies comparing technical-grade with purified PCP showed the former to produce chloracne, significant liver damage, and altered biochemistry consistent with the presence of dibenzodioxins and dibenzofurans (Table 1.2) (8,9). Some (but not all) of these "neutral impurities" are extremely toxic, the most potent effects presently being associated

TABLE 1.2. Toxic effects of PCP products[a]

Effect	Technical PCP[b]	Commercial-pure PCP[c]	Chemical-pure PCP[d]
Edema (chick)	+	-	-
Chloracne (rabbit)	+	-	-
Hematologic depression (rat)	+	-	-
Clinical chemistry change (cat)	+	-	-
Liver damage (rat)	+	-	-
Porphyria (rat)	+	-	-
Weight loss (rat)	+	+	+
Embryotoxicity (rat)	+	+	+

[a]Refs. 8,9; [b]HCDD 9-27 ppm, HCDF 90 ppm; [c]HCDD 1 ppm, HCDF 3 ppm; [d]HCDD <0.1 ppm, HCDF <0.1 ppm

TABLE 1.3. Acute oral toxicity of PCP impurities[a]

Impurity	Oral LD_{50} (mg/kg) (30 Days)	
	Guinea Pig	Mouse
1,2,4,7,8-PCDD	1.13	>5
1,2,3,4,7,8-HCDD	0.07	0.83
1,2,3,6,7,8-HCDD	0.07-0.10	1.25
1,2,3,7,8,9-HCDD	0.06-0.10	>1.44
1,2,3,4,6,7,8-HpCDD	>0.6	----
OCDD	----	>4000
Mixed PCDF[b]	----	200 (400)
2,3,7,8-TCDF	0.005-0.010	>6
2,3,4,7,8-PCDF	0.003-0.010	----
HCB	----	4000[c]

[a]Refs. 4,12,13; [b]42% TCDF, 54% PCDF, 4% HCDF; [c]Ref. 14

with chlorination at the 2, 3, 7, and 8 positions (Table 1.3). Different from PCP, the toxic symptoms are delayed; degeneration occurs in liver, thymus, and often in skin, and a variety of clinical chemistry changes are observed. The Table also shows that the guinea pig is unusually sensitive to such chemicals.

Despite its obvious toxicity and the occasional reports of accidental acute intoxication (47 US episodes between 1966 and 1976) (4), there has been remarkably little evidence of human damage due to long-term exposure. The US wood-treatment industry claims a good health and safety record (1), and a six-year epidemiological study concluded that no long-term effects could be observed even in occupationally-exposed workers (4). However, the acute and chronic effects in laboratory animals, the evidence of extensive PCP exposure in the general public (10,11), and increasing implication of PCP or its impurities in human and animal poisonings (4) make imperative the continued investigation and evaluation of PCP in the environment.

2. USES OF PCP

PCP has become one of the most versatile and widely-used biocides. For illustration, major registered uses of PCP in the USA are listed in Table 2.1 and some non-agricultural uses in the UK in Table 2.2. The latter has only a few items not included in the USA list but indicates additional uses of lauryl pentachlorophenate (LPCP). Other sources indicate that barium salts, acetate ester, copper salts, and amine salts are used in other countries (15, 16).

TABLE 2.1. Major registered uses of PCP in the USA

Use	Form
Herbicide and desiccant for forage seed crops.	PCP
Insecticides for beehives, seed plots, greenhouse use.	PCP
Microbiostat for commercial and industrial water cooling.	NaPCP
Postharvest wash for fruit	NaPCP
Microbiocide for burlap, canvas, cotton, rope and twine.	PCP
Microbiocide for leather.	KPCP, PCP
Microbiocide and insecticide for wood treatment.	PCP, NaPCP
Preservative for oil and water-based paint.	PCP
Slime control for pulp and paper.	PCP
Microbiocide for petroleum drilling mud and flood water.	PCP
Fumigant for shipping-van interiors.	PCP
Preservative for hardboard and particle-board.	PCP
Herbicide for non-food vegetation control.	PCP

TABLE 2.2. Non-agricultural uses of PCP in Great Britain (17)

Use	Form
Anti-mildew agent in the wool textile industry	LPCP, NaPCP
Mothproofing carried out by dyers and cleaners	LPCP
Wood preservation	PCP, LPCP, NaPCP
Paint additives	PCP
Antimicrobial (slimicide) agents in paper and board	PCP
Antifungal agent in textiles other than wool (cotton, flax and jute fabrics, ropes, cordage and tentage)	LPCP
Cable impregnation	LPCP
Antimildew agent in leather	?
Fungicide in adhesives	NaPCP
Bactericide in drilling fluids	?

The total world production of pentachlorophenol is not known with certainty but has been estimated (F. Korte, private communication) at 50,000 metric tons (50 million kg) per annum. The use of PCP in the USA amounts to about 23 million kg per annum (1), some 11% of which is in the form of NaPCP and KPCP; other PCP derivatives are not registered for use. In the UK, the use of PCP has been quoted (17) at over 1.25 million kg per annum, and all uses were nonagricultural; Canadian use is on the order of 2.5 million kg (18). In Japan, PCP has been an important herbicide in both paddy and upland rice (although its use has been regulated since 1971 due to toxicity to fish), and it is also used as a fungicide and in industrial applications; total production was 13.3 million kg in 1970 but fell to less than 3 million kg by 1971 (19).

Among its many and varied applications, the compound has been used extensively in water systems as a molluscicide for bilharzia control (particularly in the Far East), as a fungicide for wicker products such as baskets (Hong Kong), and as a disinfectant for cleaning floors (USA). However, by far the major use worldwide is for wood preservation. For example, about 80% of the USA use is in commercial wood treatment, while 6% is for reduction of slime in pulp and paper production and 3% goes for nonindustrial purposes such as fence-post treatment, paint preservation, weed control, etc. (3). The remaining 11%, converted to NaPCP, finds use in antimicrobial treatment of pressboard, insulation, and industrial cooling-water. PCP generally is formulated as a 5% solution in petroleum solvents, although it most often is shipped in solid form; NaPCP is applied as aqueous solutions.

PCP and its derivatives offer the advantages of oil-solubility, water solubility, biocidal effectiveness, and limited environmental persistence. Even in temperate climates, the usual 5-year functional life of wood products can be increased 8-fold by PCP-treatment; under tropical heat and humidity, some practical form of wood preservation has become imperative, and reapplication of preservative chemicals must be frequent due to their rapid dissipation. Wood may be pressure-treated commercially, or the PCP may be brushed on. The advantages and details of wood preservation have been reviewed by Nicholas (20).

In the USA, the principal commercial wood-treatment applications are to utility poles (4 x $10^7 ft^3$), lumber (2 x $10^7 ft^3$), and fence-posts ($10^7 ft^3$), although railroad ties and wharf pilings also represent major uses (6,21)(Table 2.3); about 0.23 kg of PCP is required for

TABLE 2.3. Volumes of wood materials treated with PCP in the USA, 1972-76 (thousand ft^3)(6)

Year	Cross Ties	Switch Ties	Piling	Poles	Cross Arms	Lumber and Timber	Fence Posts	Other	Total
1972	79	50	239	45,230	2,093	16,394	9,924	1,786	75,795
1973	53	7	288	47,193	2,234	19,663	9,055	1,528	80,022
1974	321	?	135	42,031	1,947	19,302	9,580	2,450	75,445
1975	334	24	384	32,155	1,301	15,837	9,953	783	60,771
1976	19	?	368	36,525	4,541	13,873	9,096	1,208	65,611

each ft^3 of treated wood. Although many other chemicals have been tested as wood preservatives, few have gained commercial prominence. The recognized PCP replacements -- creosote, arsenicals, and organotin compounds -- each have toxicological and environmental disadvantages. Chemically-treated wood has certain demonstrable advantages of cost, energy conservation, environmental compatability, and renewable resource utilization compared to its present alternatives such as metal or concrete (23).

Some 89% of the PCP used in US commercial wood treatment is applied in closed, pressurized systems, while the remaining 11% is applied by thermal, dip, and ground-line treatment, often by trained workers. In addition to the volumes indicated in Table 2.3, 8.5 x $10^7 ft^3$ of lumber is given dip treatment for control of sapstain (6). However, it is estimated that, in the USA alone, approximately 2 million pounds (9 x 10^5 kg) of PCP also is applied (mostly as 5% solution) by 3 million to 6 million private individuals each year -- a use which has been largely uncontrolled. As the Table of registered uses indicates, the small proportion of PCP not used for industrial purposes must be distributed among many common consumer items which touch on most aspects of everyday life. As only a single example, a 1973 list (22) showed 10 categories of approved food additive uses for PCP and its salts in the USA (Table 2.4). Other reported minor uses which involve direct human contact include bactericidal soaps, laundry products, and skin medication.

TABLE 2.4. US food additive uses of PCP and its salts (22)

Approved Use[a]	Specific Compound
Slime control on paper and paperboard	KPCP, NaPCP
Preservative in can-end cement	NaPCP
Defoaming agents	NaPCP
Paper contacting aqueous and fatty food	NaPCP
Animal glue for containers	NaPCP
Sealing gaskets for containers	KPCP, NaPCP
Preservative for wood products	PCP, NaPCP
Preservatives in coatings	NaPCP
Rubber antioxidant	NaPCP
Preservative for ammonium alginate	NaPCP

[a]Title 21, Code of Federal Regulations, USA.

3. CHEMICAL AND PHYSICAL PROPERTIES

Pentachlorophenol is used in the phenol form (PCP), as salts (commonly NaPCP), or as esters (laurate or acetate). The chemical and physical properties of the derivatives cannot be assumed to be identical to those of PCP itself in quantitative or even qualitiative terms, but comparatively little is known about them. Most of the following discussion refers to technical PCP.

3.1 Methods of Manufacture

PCP is manufactured by the chlorination of phenol and by the hydrolysis of hexachlorobenzene. In the classical process, chlorophenols are produced from the respective higher chlorinated benzenes by hydrolysis in 10-15% solutions of sodium hydroxide or sodium carbonate at temperatures up to 360°C and pressures of 280-300 bar. The more modern Raschig-Hooker

process uses catalytic hydrolysis with catalysts such as calcium phosphate or silicates and temperatures up to 450°C (24). Both processes have been used in chemical industry throughout much of the world.

Hexachlorobenzene hydrolysis has not been used commercially in the USA. Phenol is chlorinated directly, in the presence of catalytic amounts of aluminum chloride and organic chlorination promoters and stabilizers. Chlorine is fed into the primary reactor at 65-130°C (optimally 105°C) and atmospheric pressure; as chlorination proceeds, the temperature is allowed to increase at such a rate as to keep the reaction mixture fluid. Hydrogen chloride and unreacted chlorine are treated with phenol in a scrubber, and the resulting chlorophenol mixture can be collected or returned as feedstock to the primary reactor while HCl is recycled to the chlorine plant (25). The product (84-90% pure) is flaked or pelleted and bagged or is cast into blocks. In recent years, part of the technical PCP has been commercially purified by distillation at the factory, a process which reduces the levels of neutral impurities considerably (Table 3.1), but the extent to which the purified product is used is proprietary information.

TABLE 3.1. Typical composition of commercial PCP (3,7,8,9)

Component	Level (% or ppm)			
	Tech. PCP	Tech. PCP	Tech. PCP	Purified PCP
	%	%	%	%
PCP	85-90	84.6	88.4	90.4
TCP	4-8	3	4.4	10.4
Trichlorophenols	<0.1	----	<0.1	<0.1
Higher chlorophenols	2-6	----	6.2	----
Neutral fraction	ppm	ppm	ppm	ppm
TCDD	0	<0.1	---	<0.05
PCDD	---	<0.1	---	----
HCDD	9-27	8	4	1.0
HpCDD	---	520	125	6.5
OCDD	575-2510	1380	2500	15.0
TCDF	---	<4	----	----
PCDF	---	40	----	----
HCDF	present	90	30	3.4
HpCDF	present	400	80	1.8
OCDF	present	260	80	<1
HCB	---	----	---	400

With the variety of routes and conditions of manufacture, and the large number of manufacturers, the quality of PCP could be expected to vary significantly with source and date of manufacture. The impurities formed during manufacture presently provide the most controversial aspect of PCP use.

3.2 Composition of Technical PCP

Technical PCP is contaminated with a number of neutral and phenolic polychlorination reaction products (Table 3.1). The phenolic components include tri- and tetrachlorophenols and the predioxins and isopredioxins (16,26) formed by the condensation of two molecules of PCP (Figure 3.1) as well as those formed from the tri- and tetrachlorophenol impurities. The neutral contaminants include (i) polychlorodibenzodioxins such as IV formed by ring-closure of predioxins (II), (ii) polychlorodibenzofurans such as VII formed by reaction of PCP with hexachlorobenzene by loss of HCl, (iii) polychlorodiphenyl ethers such as V formed by free-radical condensations of PCP, (iv) chlorinated cyclohexenones and cyclohexadienones, notably 2,3,4,4,5,6-hexachlorocyclohexa-2,5-dien-1-one ("hexachlorophenol", VIII) formed by radical chlorination of chlorophenols, and (v) hexachlorobenzene (HCB, VI).

Considerable effort, much of it unpublished, has gone into analysis of commercial PCP. The values presented by Johnson et al. (8) (Table 3.1) are typical of the older work which did not reflect present understanding that predioxins, cyclohexadienones, and perhaps other major impurities could be converted to dioxins and dibenzofurans during analysis. More recent work (27,28), indicates that earlier values for the neutral impurities tended to be high (Table 3.2) and even more refined analysis employing capillary gas chromatography and mass fragmentography can distinguish the more toxic from less toxic isomers (27,29) (Table 3.3).

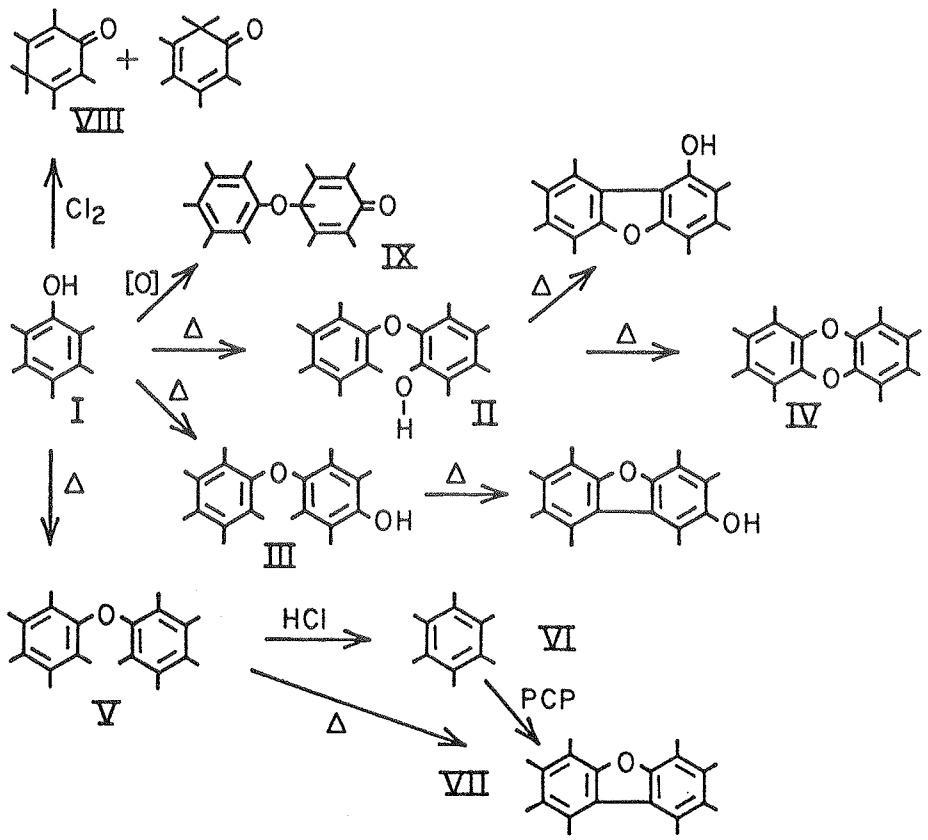

Fig. 3.1 Formation of PCP and its impurities. For clarity, Cl substituents are indicated only by lines.

TABLE 3.2. Total dioxin and dibenzofuran content of technical PCP samples

Compound	Range, ppm	Typical Analysis ppm	Reference
TCDD[a]	<0.02 - 1.25	<0.02	27
PCDD	<0.03 - 0.08	<0.03	27
HCDD	<0.03 - 38	4.2	27
HpCDD	0 - 870	54	16, 26, 27, 32, 81, 72
OCDD	0 - 3300	210	16, 26, 27, 32, 70, 71, 72
TCDF	<0.02 - 0.9	<0.02	27, 28, 32
PCDF	<0.03 - 0.65	0.10	27, 28, 32
HCDF	<0.03 - 39	23	27, 28, 32
HpCDF	<0.1 - 320	160	27, 28, 32
OCDF	<0.1 - 300	140	27, 28, 32

[a]1,2,3,4-isomer.

TABLE 3.3. Typical chlorodioxin isomer levels in PCP and NaPCP[a]

Isomer	Level (ppm)	
	PCP	NaPCP
1,2,4,6,7,9-HCDD	0.4	0.04
1,2,3,6,8,9-HCDD	1.7	0.16
1,2,3,7,8,9-HCDD	2.1	0.20
1,2,3,4,6,7,9-HpCDD	8.1	0.06
1,2,3,4,6,7,8-HpCDD	45.9	0.36
1,2,3,4,6,7,8,9-OCDD	210.	11.

[a]Calculated from data of Buser and Bosshardt (27), Samples 7 and 17.

Another major problem has been the variability of analyses among different analysts and analytical methods. A collaborative survey (D. Firestone, unpublished, 1972) in which 9 laboratories analyzed a single PCP sample by four standard methods produced extremely variable results; for example, HCDD values from one method varied between 0 and 200 ppm (average 77 ppm) and between 0 and 233 ppm within one laboratory. Although prior examination had showed TCDD to be absent, 11 values were reported for it ranging between 0.7 and 108 ppm. Villanueve et al. (30) reported similar difficulty. Again, it seems likely that recent sophisticated methods may produce more consistent results, and older values must be used with caution.

3.2.1 Dioxins and dibenzofurans. Modern analytical methods show that most PCP samples contain the higher-chlorinated dibenzodioxins and dibenzofurans; a summary of their reported content is shown in Table 3.2. The dioxin usually present in the highest concentration is the comparatively nontoxic OCDD, although sometimes this has been overestimated because of its generation from other impurities during analysis (1,16,31). Most workers have not detected any tetrachlorodioxin isomer in PCP, and indeed none has been found in PCP made in the USA (1). However, the TCDD reported by Buser and Bosshardt (27) in several samples was shown conclusively to be the unusual (and unexplained) 1,2,3,4-isomer. The two HpCDDs and 3 of 10 HCDD isomers were found (Table 3.3), and the 1,2,4,6,8,9-HCDF and 1,2,3,4,6,8,9-HpCDF predominated (28), but their range among 19 samples was very wide.

3.2.2 Predioxins, isopredioxins, and other polychlorodiphenyl ethers. These acidic impurities first were detected in PCP samples by Jensen and Renberg (16), who noted that the predioxins spontaneously formed dioxins on gas chromatography and, without prior removal, would be reported as dioxin; isopredioxins such as III do not form dioxins (Fig. 3.1). According to these authors, the predioxin level in a typical PCP sample often was significant:

Predioxin (II) of OCDD	0.6 - 1600 ppm
iso-Predioxin (III)	trace - 1600 ppm
Predioxin of HpCDD	trace
iso-Predioxin of HpCDD	1200 ppm

Polychlorodiphenyl ethers were detected qualitatively but not quantitatively in PCP by Firestone et al. (32) and contained 4 to 10 chlorine atoms. Decachlorodiphenyl ether (V) has been implicated in the thermal generation of HCB and octachlorodibenzofuran from PCP (33). The preparation and properties of a number of the ethers have been reported by Lundstrom and Hutzinger (34).

3.2.3 Cyclohexadienones. The neutral PCP impurities often include relatively large proportions of chlorinated cyclohexenones and cyclohexadienones of which the best known is "hexachlorophenol" -- not a phenol, but the ketone 2,3,4,4,5,6-hexachlorocyclohexa-2,5-dien-1-one (VIII). While dilute base converts hexachlorophenol to PCP, heat converts it to OCDD, yet another source of analytical error (35). The less-chlorinated members of the series, likewise formed by radical-chlorination of tri- and tetrachlorophenols, similarly produce chlorodibenzodioxins on heating (36).

3.2.4 Chlorinated hydrocarbons. Hexachlorobenzene (HCB) is a common but often overlooked contaminant of PCP which may occur in amounts as high as several percent (for example, see Ref. 7). It may arise from unreacted starting material in the hydrolytic manufacturing process, and it is the principal product when PCP is heated above 300°C (33). It has been suggested (33) that HCB is formed by action of dry HCl on decachlorodiphenyl ether at high temperature; it also could arise by the combination of pentachlorophenyl radicals with chlorine atoms. This latter type of reaction also could give polychlorinated biphenyls (PCB's), which indeed have been found in PCP (37).

3.3 Chemical Properties of PCP

As with any phenol, the hydroxyl group of PCP takes part in nucleophilic reactions (e.g., it forms esters with organic and inorganic acids and ethers with alkylating agents such as methyl iodide or diazomethane). Electron withdrawal by the ring-chlorines causes PCP to be unusually acidic [pK_A 4.70 in water (38), roughly comparable to propionic acid, pK_A 4.9] and a relatively weak nucleophile, while stabilizing its salts (sodium pentachlorophenate is a stable item of commerce). Although the high degree of chlorination makes the aromatic ring sufficiently electropositive to form stable charge-transfer complexes with electron donors, the ring chlorines are as resistant to nucleophilic displacement (e.g., by hydroxide ions) under normal conditions as those of the chlorinated aromatic hydrocarbons (2).

Oxidation of PCP produces pentachlorophenoxyl radicals which combine reversibly to form "dimers". For example, in the presence of fuming nitric acid or nitronium fluoborate, PCP gives 2,3,4,5,6-pentachloro-4-pentachlorophenoxy-2,5-cyclohexadienone (IX) (39,40). The radicals or their dimers can be oxidized further to 2,3,5,6-tetrachlorobenzoquinone (chloranil).

Pyrolysis of alkali metal salts of PCP (>300°C) results in condensation of two molecules to form OCDD (41) (Figure 3.1). The reaction proceeds through the intermediate phenoxyphenol ("predioxin") readily detectable in technical PCP; nonvolatile, polymeric phenylene ethers are formed concurrently by reaction of the chlorophenol at positions other than ortho. Small amounts of initiators such as chlorine or cyclohexadienones allow OCDD formation at comparatively low temperatures (200°C) in high yield from PCP itself (33), and pyrolysis of the chlorinated cyclohexadienones provides OCDD smoothly (73% yield from VII at 270-280°C), probably through an intermediate such as IX. The pyrolytic conversion of a PCP salt into OCDD is accompanied by a large proportion of hexachlorobenzene (41), perhaps from decomposition of decachlorodiphenyl ether (V), which may explain the erratic occurrence of this substance in technical PCP. Pyrolysis of tri- or tetrachlorophenolates generates a mixture of dioxin isomers due to the Smiles rearrangement (42); potassium 2,3,4,5-tetrachlorophenate yielded only 20% of the expected 1,2,3,6,7,8-HCDD but 80% of its rearranged 1,2,3,7,8,9-HCDD isomer. In fact, a number of the PCP dioxin impurities may be rearrangement products (43).

The absorption of light energy allows PCP to undergo a number of reactions under very mild conditions; the long-wave absorption maxima lie near 300 nm in organic solvents or below pH 5 (Section 3.4). In either water or organic solvents, PCP undergoes photochemical reduction to isomeric tri- and tetrachlorophenols (44) (Fig. 3.2). Nucleophiles such as bromide ion can displace chloride from the excited PCP ring (45), and, in dilute aqueous solutions exposed to sunlight, PCP or its salts undergo the replacement of ring chlorines by hydroxyl groups. The resulting tetrachlorohydroquinone (X), tetrachlorocatechol (XI) and tetrachlororesorcinol (XII) are readily oxidized by air to quinones such as chloranil (XIII) which subsequently are dechlorinated. If the original PCP solution is sufficiently concentrated (as in the case of a rice-paddy), the tetrachlorodiols react with the quinones to give a variety of non-toxic minor products (46); under most circumstances, the quinone solution is rapidly degraded to dichloromaleic acid (XIV) which is converted into small fragments, CO_2, and HCl within a few days (47).

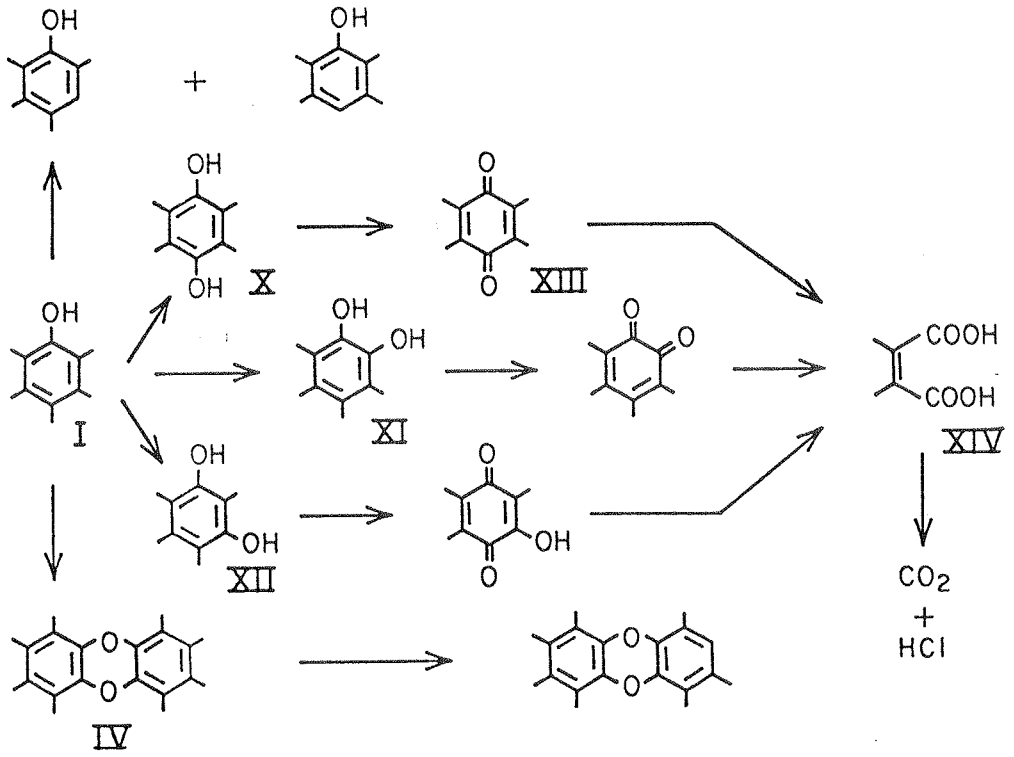

Fig. 3.2 Photolysis of PCP. For clarity, Cl substituents are indicated only by lines.

The pentachlorophenoxide anion (λ_{max} 320 nm) also can displace chloride from PCP in sufficiently concentrated solution with eventual cyclization to OCDD in water at ambient temperature (45,48). In more concentrated solutions of NaPCP (about 2%), a number of colored condensation products and chloranil also were isolated (46), while UV irradiation of very dilute aqueous solutions of NaPCP (0.1-1 ppm) caused chemical alteration in which the lethal effects of irradiated solutions on snail eggs were lowered (49).

The photochemical fate of PCP both as a solid film and adsorbed on silica was investigated by Gäb et al. (50). UV absorption by aromatics often is changed following their adsorption on active surfaces; adsorption of PCP on silicic acid resulted in an absorption shift of 15 nm toward longer wavelengths. After 7 days of UV irradiation (>290 nm), only 12% of the adsorbed PCP could be recovered, although no breakdown products were detected. Unlike the very stable hexachlorobenzene and pentachlorobenzene, a PCP film also was mineralised when irradiated in an oxygen stream. After 7 days irradiation on Pyrex, about 15% of adsorbed PCP was converted to CO_2 and HCl. Consequently, the UV component of sunlight has the necessary energy to decompose otherwise stable and persistent substances such as PCP whether in solution, thin films, or adsorbed.

Chlorination of PCP (or overchlorination of phenol during PCP manufacture) gives rise to a series of interconvertible nonaromatic cyclic ketones including hexachloro-2,5-cyclohexadien-1-one (VIII), hexachloro-3-cyclohexen-1-one, heptachloro-3-cyclohexen-1-one, and octachloro-3-cyclohexen-1-one which have highly reactive chlorines. Their reduction under mild conditions, or treatment with base, provides chlorophenols; for example, PCP is formed from VIII by boiling with aqueous alkali or by reduction with aqueous sulfur dioxide or potassium iodide (51). Heat (as in a gas chromatograph) also regenerates phenols from the cyclohexadienones (36), as does UV light (52).

3.4 Physical Properties of PCP and NaPCP

Some common physical properties of PCP and NaPCP are summarized in Table 3.4 (3,4). The pure substances are white crystalline solids, while the commercial material generally is seen as tan or gray flakes. Pure anhydrous PCP melts near 190°C (its monohydrate melts at 174°C), but technical products may melt at 187-189° or less due to impurities; PCP salts are high-melting. Detailed physical properties are given by Carswell and Nason (53).

TABLE 3.4. Selected physical properties of PCP (38,53,54)

Property	PCP	NaPCP
Mp°C	190.2°	Dec
Bp°C	300.6°	Dec
Vp Torr (mm Hg)		
0°C	1.7×10^{-5}	
20°C	1.7×14^{-4}	
50°C	3.1×10^{-3}	
100°C	0.14	
200°C	25.6	
300°C	758.4	
Solubility in water (g/L)		
0°C	0.005	
20°C	0.014	22.4
30°C	0.020	33
50°C	0.035	
70°C	0.085	
Solubility in organic solvents (g/L, 25°)		
Methanol	180	22
Acetone	50	37
Benzene	15	insol.
pK_A (25°)	4.70	
Partition coefficient (Kp), 25°		
Octanol-water	1760 (log Kp 2.15)	
Hexane-water	1.03×10^5 (log Kp 5.01)	

PCP partially decomposes to HCB, OCDD, and tar at its boiling point (41); the vapor pressure of 760 torr is achieved at 300.6°C, but even at ambient temperatures PCP must be considered to be relatively volatile (53). NaPCP is nonvolatile -- its sharp PCP odor is due to slight hydrolysis -- and high temperatures convert it largely to OCDD (Section 3.3). At

100°C, 0.167 g of PCP steam-distills with 100 g of water at acidic pH (54).

PCP is soluble in most organic solvents but only slightly soluble in water. However, its solubility, volatility, and partitioning must be considered in relation to its ionization. At pH 2.7, PCP is only 1% ionized, while at 6.7 -- the pH of many natural waters -- it is 99% ionized. The 18 mg/L aqueous solubility (20°C) at the slightly acidic pH generated by its dissolution (pH 5) increases rapidly with increasing pH to over 2×10^5 mg/L as NaPCP at pH 10. In seawater (pH 8.1), less than 0.1% of the PCP exists in nonionic form.

The UV absorption spectra also depend upon pH. PCP shows an absorption maximum at 303 nm (ε = 2900); at alkaline pH (>10), the absorption maxima of pentachlorophenate ion lie at 248 nm (ε = 10,700) and 323 nm (ε = 5,300), while at the pH 7.3 (>99% ionic) characteristic of many natural waters, the long-wavelength value is 320 nm (ε = 4,150) (2,47,55).

3.5 Chemical and Physical Properties of Dioxins and Dibenzofurans

Dioxins and related cyclic ethers are rather stable chemically, perhaps due to their planar and electropositive rings (56). For example, OCDD distilled (sublimed) unchanged at 350°C and was recovered quantitatively from hot sulphuric acid (41). However, they exhibit UV absorption maxima between 305 and 320 nm (4) and share with PCP the facile photochemical reduction upon UV (and sunlight) irradiation. In an organic solvent as hydrogen donor, 2,3,7,8-tetrachlorodibenzo-p-dioxin was rapidly dechlorinated *via* tri- and dichlorodioxins (57), and other dioxins were similarly dechlorinated (43,58); the chlorodibenzofurans were dechlorinated more slowly (59,60) and OCDD slower still, although transient hepta-, hexa-, and other chlorodioxins were detectable (29,59,61). Direct oxidation has not been reported, but oxidation of the oxonium salt formed in strong acid produces a blue radical-cation useful in dioxin analysis (62).

While photochemical dechlorination is by far the most rapid known reaction of these compounds, three conditions are required: the dioxin or dibenzofuran must be accompanied by a hydrogen-donating solvent (such as a pesticide or formulating agent), UV light of 290-320 nm must be present, and the light must penetrate the solvent (63). The rate of photoreduction is inversely proportional to the degree of chlorination, and therefore dechlorinated products do not accumulate (57). Unlike PCP, pure OCDD and TCDD do not appear to be photolyzed in water at appreciable rates due, perhaps, to their very low solubility. While organic TCDD solutions were degraded by sunlight on a leaf or glass surface within a few hours, pure TCDD in the absence of solvent was stable (63). UV irradiation of TCDD adsorbed on silica allowed only 8% to be recovered after 7 days (F. Korte, unpublished, 1979).

The higher-chlorinated dioxins and dibenzofurans melt above 200°C, are slightly soluble in common organic solvents (~1 g/L), have µg/L solubilities in water, and exhibit vapor pressures on the order of 10^{-6} to 10^{-7} torr (4). The solubility and vapor pressures are roughly similar to those of DDT, which is known to volatilize readily from soil and water; while the neutral PCP impurities might logically be expected to behave in the same way, no volatilization rates have been measured. In the absence of organic hydrogen-donors, the dioxins and dibenzofuran vapor should be photochemically stable in the atmosphere, although TCDD has been reported to react readily with ozone (64).

As expected, partition coefficients between organic solvents and water are high (4). Unlike PCP and TCP, the toxic dioxins and dibenzofurans are insoluble in dilute alkali, although the more chlorinated members such as HpCDD and OCDD are degraded by a few minutes' boiling with aqueous-alcoholic potassium hydroxide (65). This property provides not only an important initial extraction step but also a possible means of PCP purification.

3.6 The Fate of PCP and Its Impurities in Treated Wood

The treatment of wood with PCP dissolved in light petroleum or low-pressure natural gas causes deep penetration into the cells, particularly those toward the interior. While some of the compound inevitably "bleeds" out and may vaporize or be washed away, with time the biological activity decreases and extractable PCP declines although physical analysis shows the PCP concentration to remain constant (66). Staining, selective extraction, scanning electron microscopy, and electron microprobe analysis all indicate that the PCP is bound to the cell walls, primarily in the middle lamella, perhaps by H-bonding to lignin, formation of charge-transfer complexes, or acetal or ester formation (67,68).

The concentrations of neutral PCP impurities forced into the wood may remain relatively constant with depth, but PCP migrating to the surface is converted to OCDD, HpCDD, and HCDD by sunlight (69). HCl also may be produced during photolysis, and PCP often is not satisfactory for treating fabrics as they may be damaged (20).

3.7 Conclusions

PCP is an important industrial chemical, produced in many countries and widely used in most. Its numerous applications -- on wood, leather, and paint, for example -- ensure human exposure. Except for the usual hydroxyl reactions, it is quite stable in the laboratory, but it absorbs and is rapidly degraded by UV light and would not be expected to persist in the open environment (although it remains unchanged for long periods in treated wood).

However, it also reacts thermally; its manufacture gives use to chlorinated dioxins, dibenzofurans, and other impurities whose reported levels depend upon the production route and conditions as well as the method of analysis. While the slightly toxic OCDD predominates and the highly toxic TCDD is absent, toxic HCDD and HpCDD isomers -- those with 2,3,7,8 chlorination -- often are present in substantial quantities. Their chlorination pattern shows them to arise from the tetrachlorophenol impurities and the forcing conditions of the major manufacturing processes.

This suggests that purer feedstock, milder reaction conditions, intentional overchlorination, and inhibition of free-radical intermediates could reduce the levels of toxic impurities, although probably not avoid them completely. The other alternative is their removal by distillation or extraction, but whether the cost of even partial purification and the subsequent safe disposal of the neutral fraction can be justified on the basis of reduced human exposure still is open to question.

The usually-stable dioxins and dibenzofurans also are degraded by sunlight; while desirable for the less-chlorinated members, this means that OCDD can be converted to HCDD and HpCDD -- on the surface of wood, for example. Sunlight also can generate OCDD from PCP under environmental conditions. Sophisticated analytical methods to detect and measure the neutral PCP impurities now exist and must be applied not only to the technical PCP itself but, especially, to determination of actual human exposure to this compound and the associated dioxins and dibenzofurans.

4. BIOLOGICAL UPTAKE AND TRANSFORMATION OF PCP AND ITS IMPURITIES

4.1 Uptake and Metabolism of PCP in Animals

Although PCP is acutely toxic to animals, it is readily metabolized and eliminated by most of the species examined to date. For example, rats eliminated a single oral dose of [^{14}C] PCP (either 10 or 100 mg/kg body weight) by conversion to tetrachlorohydroquinone, excretion as unchanged PCP and its glucuronide conjugate in the urine (~70% of dose), and by excretion of PCP or its metabolites into the bile (73,74). The identity of radiochemical in the feces (equivalent to ~25% of dose) was not determined. The dynamics of elimination of ^{14}C was biphasic in both males and females at 10 mg/kg; the rapid phase had half-lives of 17 and 13 hrs, respectively, and over 90% of the dose was eliminated then (73). Comparison of the elimination rates of PCP and other environmental chemicals (75) showed PCP to be among the most readily cleared.

Similarly, Jacobson and Yllner (76) reported the fate of [^{14}C] PCP in mice dosed by subcutaneous or intraperitoneal injection; 73-83% was excreted in the urine over 4 days as tetrachlorohydroquinone, PCP, and glucuronide conjugate, with 8-21% of the dose in the feces. Rabbits excreted PCP and glucuronide conjugate but no tetrachlorohydroquinone (Kobayashi et al., 1970).

The rhesus monkey excreted PCP at a much slower rate. A single oral dose of 10 mg/kg of [^{14}C] PCP in corn oil was eliminated with a half-life of 40 hrs in the male and 90 hrs in the female (78). Only PCP was excreted in the urine -- no conjugate or tetrachlorohydroquinone was detected -- and even after 15 days, 11% of the dose still was retained in the body (1% in liver, 7% in intestines and 3% in other organs). Tetrachlorohydroquinone was detected in the urine of human workers occupationally exposed to PCP (74), but Casarett et al (79) found that after the cessation of work-related exposure, the concentration of PCP in urine had decreased only by 60% after 18 days.

The uptake, clearance, and metabolism of PCP in aquatic animals has been reviewed by Kobayashi (80). As a typical example, PCP was absorbed rapidly by rainbow trout (Salmo gairdneri) exposed to a concentration of 0.025 mg/L in the water; after 24 hrs, liver, blood, fat, and muscle contained 16, 6.5, 6.0 and 1.0 ppm PCP, respectively (81), which was eliminated with half-lives of 9.8, 6.2, 23 and 6.9 hrs into clean water. Bile contained high concentrations (250 ppm), mostly as glucuronide, but no other metabolites were detected. However, goldfish (Carassius auratus) (80,82) and shellfish (77) excreted the sulphate conjugate. After 120 hrs of exposure to 0.1 ppm of PCP, the goldfish had concentrated it about 1000-fold (primarily as glucuronide in the gall bladder), although a return to clean water resulted in rapid clearance mainly as PCP conjugates; clearance from the gall bladder was much slower than from other body tissues (83). Bioconcentration was similar into other fish species (75,80). Other invertebrates -- mollusks, crustaceans, and Culex mosquito

larvae -- also accumulated PCP and metabolized it to PCP acetate, tetrachlorophenol, tetrachlorohydroquinone, and/or conjugates (80,87).

4.2 Uptake and Metabolism of PCP in Plants

Surprisingly little information exists on plant metabolism of PCP, although the compound is very phytotoxic. Miller and Aboul-Ela (84) applied [^{14}C] PCP to cotton plants but did not investigate the metabolic products. Limited translocation of radioactivity was reported and the kernels of bolls, which were closed at spraying, contained residues of radioactivity at harvest. Oats grown in PCP-treated soils absorbed only small amounts of radioactivity, while foliar application of PCP to sugar cane resulted in almost complete recovery eventually from the leaves, while root application in cultures deposited most of the compound in the roots (85).

Haque et al. (86) reported preliminary results on the growth of rice in soil treated with [^{14}C] PCP. After one week, the plants had absorbed about 3% of the applied radioactivity, half of it strongly bound. Most (90%) extracted radioactivity represented unchanged PCP, but 9% was in the form of unidentified conjugates and about 1% was identified as 2,3,4,6-tetrachlorophenol (although the presence of the 2,3,5,6-isomer could not be excluded). Algae metabolized rather than concentrated PCP and converted it into similar products (87), and it seems probable that, once absorbed, plants will metabolize PCP by the routes common to other living things (Sections 4.1 and 4.3).

4.3 Transformation of PCP by Microorganisms

As reviewed recently by Kaufman (88), PCP generally is metabolized rapidly by most microorganisms. Several cultures which degrade PCP have been isolated from soil: Kirsch and Etzel (89) obtained a mixed culture from soil sampled from the grounds of a manufacturer of wood products (biodegradation was indicated by release of $^{14}CO_2$ during a 24 hr exposure to [^{14}C] PCP), and continuous-flow enrichment provided an unidentified bacterium which metabolized PCP as a sole source of organic carbon (90). Watanabe (91) isolated a PCP-decomposing Pseudomonas from soil perfused with PCP solution which was able to grow on and degrade PCP, all 5 chlorine atoms being liberated. Pseudomonas degraded [^{14}C] PCP rapidly and released $^{14}CO_2$ as well as the intermediate metabolites tetrachlorocatechol and tetrachlorohydroquinone (92). Using bacterial isolates from wood-preservative wastewater, Reiner et al. (93) also implicated chloranil and 2,6-dichlorohydroquinones as metabolic intermediates.

Duncan and Deverall (94) showed that several species of fungi also depleted PCP from treated wood blocks, and slow chloride release and detoxication of PCP occurred via the fungal enzymes laccase, tyrosinase, and peroxidase (95). Cserjesi (96) found that PCP disappeared during 12 days' incubation with cultures of 3 species of the fungus Trichoderma; Trichoderma virgatum growing in malt extract was shown to methylate PCP to pentachloroanisole (97), although this did not account for the total loss of pentachlorophenol in the medium. Similarly, several fungal species also caused methylation of tetrachlorophenol to tetrachloroanisole, a compound causing a musty taste in poultry products (98), and tetrachlorodihydroxybenzenes and their methyl ethers were isolated as PCP degradation products formed by Aspergillus (99).

Ability to degrade PCP may not be a uniform feature among microorganisms. Alexander and Aleem (100) found no degradation of PCP in a mixed population grown from a soil suspension, no degradation was observed in acclimated active sludge (101), and overall degradation to CO_2 was limited during another 5-day test with activated sludge (75). However, Kirsch and Etzel (89) and others (1) have shown that PCP is readily biodegradable in water from an activated sludge plant. Indeed, adaptation of microbial populations to PCP may play an important role in the degradation. Successive additions of PCP to paddy soils hastened depletion (Watanabe and Hayashi, (102), and readdition of PCP after its initial degradation result in an accelerated rate of subsequent PCP degradation (91). PCP-decomposing microorganisms increased within 6 weeks after an initial application by about 3 orders of magnitude, the number in soils that had received PCP for 3 years reaching 10^5/g or more while that in soil without PCP was 10^2/g.

4.4 Transformation of PCP in Soil

4.4.1 Adsorption onto soil. The degree of adsorption of a pesticide affects both its rate of degradation and its tendency to disperse by leaching. PCP is adsorbed to a moderate degree under acidic conditions (as neutral molecules) but moves quite rapidly in ionized form under neutral or alkaline conditions (103). Adsorption is greatest at a soil pH of 4.6-5.1, and no adsorption occurs above pH 6.8 (104). However, in a series of herbicides tested (105), PCP was the most strongly adsorbed. According to Watanabe (106), PCP is strongly adsorbed by volcanic ash soil, but the organic fraction probably is more important in most instances.

Four months after [^{14}C] PCP soil application at a high rate (22 kg/ha) under controlled conditions, only 0.5% of the applied ^{14}C was detected in leachate, (F. Korte, unpublished, 1979), indicating that leaching was not a significant pathway of dispersion. Data referred to by Arsenault (1) also indicate that dispersion by leaching occurs to only a limited

extent; degradation may take place before the potential for leaching can be achieved, although Wong and Crosby (47) reported the occurrence of PCP in wellwater in Northern California locations near a wood-treatment plant.

4.4.2 Degradation in soil. Early studies showed that the toxicity and biocidal activity of even massive PCP applications disappeared in soil (107,108). Herbicidal activity in soil persists for approximately 3 weeks (109) [although PCP also has been cited as "very persistent" (110)], and both chemical or biological degradation were implicated. Most studies on PCP degradation in soil have been reported in Japanese literature (102,111-113); other studies have been referred to by Arsenault (1), and Kaufman (88) also has reviewed the subject.

The rate of decomposition of PCP in laboratory soils is more rapid in those of high organic content than in those of low organic content, and greater when moisture content is high and when soil temperature approach the optimum for microbial activity (114); half-lives usually are on the order of 2-4 weeks. The degradation of PCP in a paddy soil at 28°C also shows a half-life of about 3 weeks, and reducing conditions increase the rate of degradation slightly, whereas sterilization impedes it (115). Reductive dechlorination by microorganisms produces tetrachlorophenols, trichlorophenols, dichlorophenols, and m-chlorophenol, meta-chlorines being the most stable. Methyl ethers of most of the phenols also isolated.

Kuwatsuka and Igarashi (116) confirmed these generalizations in ten different soil types. PCP degradation rates were higher under anaerobic (flooded) conditions than under aerobic (upland) conditions, the half-life of PCP at 30°C varied from 20-120 days under aerobic conditions and from 10-70 days under anaerobic conditions, and the rate of breakdown increased with the organic matter content -- in one soil of very low organic matter, 100% of the PCP was recovered after 2 months incubation. PCP degradation was assumed to proceed by both chemical and microbial means, based on the effect of sterilization, soil temperature, and the nature of the degradation products. These products, detected by gas chromatography in extracts of the soil, included 2,3,4,5-, 2,3,4,6- and 2,3,5,6-tetrachlorophenol, 2,3,6-, 2,4,6-, 2,3,5-, and 2,3,4-trichlorophenol (or 2,4,5-trichlorophenol), and pentachloroanisole.

These results also are consistent with information obtained by use of [^{14}C] PCP in a silty clay loam (117). Under anaerobic conditions, no $^{14}CO_2$ was detected during a 24-day incubation period; 5.3% of the radioactivity subsequently was extracted was present as pentachloroanisole, whereas 7% was present as 2,3,5,6-tetrachlorophenol, 2,3,4,5-tetrachlorophenol, and 2,3,6-trichlorophenol, and 2.5% as polar material. Aerobic production of pentachloroanisole was greater than that under anaerobic conditions; aerobic soil gave less extractable ^{14}C-activity (14.7%), and more activity remained as bound residue (44.6%). Indeed, pentachloroanisole was suggested to be the principal residue adsorbed to PCP-treated field soils.

4.5 Uptake and Transformation of Dioxins and Dibenzofurans

Very little information exists on the uptake and metabolism of the dioxins actually found in technical PCP. However, much more data are available on 2,3,7,8-TCDD; although this isomer has not been detected in PCP, its properties seem to reflect those of the related compounds, especially the ones which have chlorines at the 2,3,7, and 8-positions, and may provide insight into the behavior of the PCP impurities.

No reports have appeared on the uptake and bioconcentration of PCP dioxins or dibenzofurans in higher animals, although HCDD was responsible for extensive outbreaks of chick edema (22), and HCDD, HpCDD and OCDD were detected in dairy cattle (4). In the rat, a single oral dose (50 µg/kg) of TCDD cleared with a half-life of 17 days (118), with retention primarily in the liver. After 21 days, the remaining body burden still represented 40% of the initial dose. Chronic feeding with a diet containing 7 and 20 ng/g of [^{14}C] TCDD (119) showed that whole-body retention represented about 40% of the total previous intake at any given time; when placed on a clean diet, the dosed animals last their TCDD with a half-life of about 12 days. Isensee and Jones (120) examined the distribution of TCDD among the snail, microcrustaceans, and fish of an "aquatic model ecosystem" and found it bioconcentrated in all instances to an extent of roughly 10^4 relatively independent of the concentration in water.

Several TCDD investigations (119,121) indicated that 2,3,7,8-TCDD was not metabolized in animals. Likewise, OCDD was not metabolized by the rat (122), but other chlorinated dioxins were hydroxylated at open 2,3,7, or 8-positions exclusively -- perhaps explaining the stability of the other dioxins examined earlier.

Higher plants, such as oats and soybeans, did not absorb appreciable TCDD from soil (123), and administration of the compound directly to leaves resulted in retention rather than translocation. Algae, however, rapidly absorbed and concentrated TCDD about 10,000-fold from water (120).

What little information there is about the properties of dioxins and dibenzofurans in soil indicates that they are adsorbed very tightly and are virtually immobile (124). Although microbial degradation exists, the process is slow and restricted to relatively few microbial species (125). On the other hand, Arsenault (1) presented evidence for microbial degradation of OCDD, although the rate was slower than that for PCP. TCDD dissolved in a pesticide formulation was degraded on the soil surface when exposed to sunlight, but the part of the solution already absorbed into the soil remained unchanged; in comparison, the TCDD solution exposed to sunlight on a leaf or a glass surface was entirely degraded within a few hours. In the absence of solvent pure TCDD was stable under these conditions (63).

Half-life values for TCDD in Florida soil was 190 days, in Utah soil 330 days, and in Kansas soil 240 days (126), and almost no leaching occurred (<0.6% in 270 days in Utah soil). Kearney et al. (127), estimated that one year represented a typical TCDD half-life in soil, but later work indicated as short a time as 9 weeks (128). It seems likely that the other toxic dioxins would persist to an analogous degree.

4.6 Conclusions

PCP apparently is absorbed and degraded readily by most living things when exposure is at nontoxic levels (Figure 4.1). Under aerobic conditions, the principal detoxication

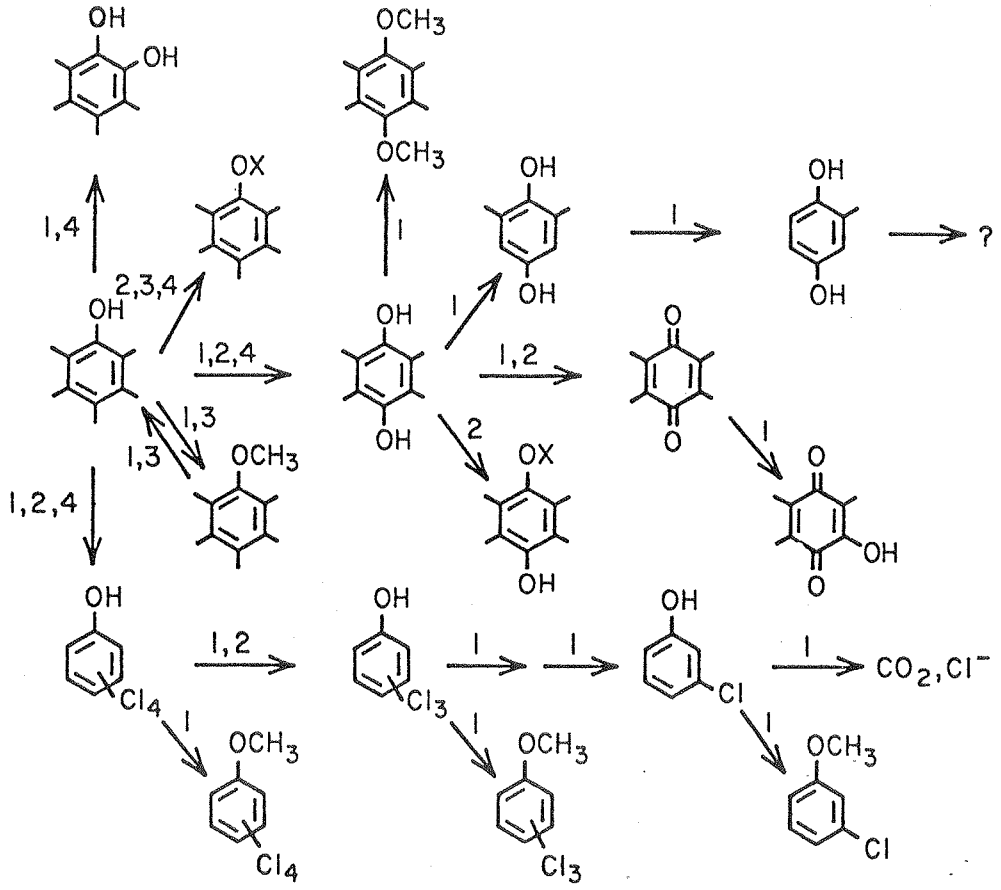

Fig. 4.1 Biodegradation of PCP. For clarity, Cl substituents are indicated only by lines. 1=Microorganisms, 2=Mammals, 3=fish and aquatic invertebrates, 4=green plants.

products are tetrachlorohydroquinone (and tetrachlorocatechol), water-soluble conjugates, and, in microorganisms at least, pentachloroanisole. Bioconcentration is not extensive. Although absorbed strongly to acidic soil, degradation by both microbial and purely chemical pathways results in usual half-lives of a few weeks. Breakdown is especially rapid under anaerobic (flooded) conditions, where the principal products are tetra-, tri-, and dichlorophenols. There is no evidence of biological conversion of PCP to dioxins or dibenzofurans.

In contrast, the dioxins and dibenzofurans -- to the extent they have been investigated -- are much more stable to metabolism and are much more highly bioconcentrated than is PCP. While the highly toxic TCDD has received the most scientific attention, it has not been found in commercial PCP; virtually no information exists on the bioconcentration or metabolic fate of the dioxins and dibenzofurans actually found as neutral PCP impurities. However, the most toxic of these -- the ones chlorinated at the 2,3,7, and/or 8-positions--can be expected to be both highly bioconcentrated and only very slowly biodegraded.

5. ANALYTICAL METHODS FOR PCP AND ITS IMPURITIES

Numerous methods have been reported for the detection and measurement of PCP. Most of the earlier ones, based on color reactions and reviewed by Bevenue and Beckman (2), now have been supplanted by gas chromatography. Analytical separations long have relied on several distinctive PCP properties: its volatility with steam; its relatively strong acidity which allows extraction into base or ion-exchange chromatography; its electropositive ring which enhances selective chromatographic adsorption and the absorption of UV light; and its ability to form esters, ethers, and colored derivatives.

5.1 Colorimetry and Spectrophotometry

As early as 1942, the nitric acid oxidation of PCP to yellow chloranil, the formation of colored complexes between PCP and 4-aminonantipyrine, and other color reactions provided the basis for most of the analytical methods (2). To cite only a few examples, PCP in surface water was determined by Colas (129) after extraction with chloroform and reaction with copper sulphate and pyridine; recovery was 97%, and the detection limit was 0.5 ppm. PCP was determined in air samples by Gromiec (130) using its reaction with safranin-O in alkaline solution to give a pink product which was extracted into chloroform and measured by spectrophotometry with a detection limit of 0.033 mg of PCP/m^3. 4-Aminoantipyrine was used by Kerner (131), Tsuda and Kariya (132) and Ueda et al. (133) for the determination of PCP in wood, fish, or body fluids following cleanup by steam distillation or acid-base separation, while PCP was determined in softwood and preservative solutions by Williams (134) using 4-aminophenazone, a derivative of 4-aminoantipyrine, after anion-exchange chromatography. In general, these procedures were cumbersome, not particularly specific, and rather insensitive (achieving at best the low microgram range), but they offered the advantage of relatively simple equipment.

Among the most sensitive and accurate of the spectrophotometric methods was that reported by Fountaine et al. (135). Surface-water samples were extracted with chloroform, cleaned up by acid-base separation, and PCP determined directly from the UV spectrum. Recovery was 98%, sensitivity 2 ng/ml, and there was little interference from other water pollutants.

5.2 Gas Chromatography

PCP was determined directly by Higginbotham et al. (136) in fats, oils, and foodgrade fatty acids after treatment with concentrated sulphuric acid and clean-up by acid-base separation, but the procedure was not suitable for quantitative measurements due to low and inconsistent recoveries. Barthel et al. (137) successfully determined PCP in human tissues and clothing by direct gas chromatography with electron capture (EC) detection and DEGS treated with phosphoric acid as stationary phase. The recovery was 90-100%, and the detection limit in blood was about 0.02 ppm, corresponding to 0.02 ng of PCP.

However, EC gas chromatography combined with chemical derivatization is the method currently preferred. Diazomethane commonly is used to methylate PCP to the corresponding methyl ether; for example, the method has been applied to the determination of PCP in blood and urine (137a,138), fish, soil, and water (139,140), wood dust (141), and stone fruits (142). The Rivers modification of the original Bevenue procedure uses benzene as the extraction solvent, and methylation is carried out directly in the benzene extract. The recovery is 89-99% and the detection limit $1:10^8$. Methylation in benzene also was used by Renberg (139), but a clean-up over Sephadex was included prior to derivatization and produced detection limits from $1:10^{12}$ in water to $1:10^9$ in fish. This procedure, based on the work of Bevenue et al. (143), is presently the official method recommended and used in the USA by EPA and FDA for regulatory analysis (144,145).

In addition, other modifications of the procedure have been reported. Dimethyl sulphate was used in dilute base by Wakimoto et al. (146) for the methylation of PCP for analysis in water and soil samples; recoveries were 84-116% for water and 86-99% for soil, and detection limits were $1:10^{11}$ ppb for water and $1:10^9$ for soil. Ethyl-PCP was used by Shafik (147) and by Lindström and Nordin (148) for human tissue and for bleach liquors from pulp mills, respectively. Methyl, ethyl, propyl, n-butyl, isobutyl, amyl and isoamyl-PCP were prepared by Cranmer and Freal (149) and their behavior on seven different phases determined as well as their p-values in three different solvent systems.

The determination of PCP as an alkyl ether has two drawbacks: hazardous reagents such as diazomethane or another diazoalkane or dimethyl sulfate must be used, and carboxylic acids and other compounds with active H-atoms also will be methylated, giving rise to possible interference. Acylation of PCP circumvents both problems. Acetic anhydride can be handled more easily than the alkylating agents, and interference can be minimized. Acetylation of PCP with acetic anhydride to give acetyl-PCP has been used by several workers, including Chau and Coburn (150), Gebefügi et al. (151), Krijgsman and van de Kamp (152), and Rudling (153) and applied to environmental samples such as surface waters, industrial wastes, air, and fish. Acetylation was carried out in weakly alkaline aqueous solution after acid-base separation, with typical recoveries reported by Rudling (153) as 92-98% for surface waters and 81-91% for fish; the detection limit for surface waters (150) was $1:10^{11}$. Alternatively, Farrington and Munday (154) reported derivatization of PCP with 2,4-dinitrofluorobenzene which provided a limit of detectability of $5:10^{9}$.

5.3 Other Methods

Several specialized gas chromatographic modifications offer advantages. The five chlorines of PCP assist greatly the sensitivity of the Cl-specific microcoulometric detector (2), and in recent years the increasing use of the mass-spectrometer detector has provided a high degree of specificity (148,155). Glass capillary gas chromatography has permitted greatly improved separations of PCP from related contaminants (152,156).

Thin-layer chromatography was used by Challen and Kucera (157), Davies and Thuraisingham (158), and Malygina and Tsendrovskaya (159), among others, for the determination of PCP in wood, latex, air, or blood. Detection was either by exposure to UV light or by spraying with an ammoniacal silver nitrate solution.

An extension of the well-known thin-layer chromatographic determination of cholinesterase inhibitors with enzymatic detection was extended to PCP by Geike (160); PCP showed remarkable inhibitory properties towards several enzymes, including bovine liver esterase, and 10 nanograms of PCP could be detected.

High-pressure liquid chromatography (HPLC) has been applied routinely to PCP analysis. An official US regulatory method (144) typically employs a 254 nm UV detector, DuPont ODS Permaphase as stationary phase, and 30% aqueous methanol as mobile phase, thus combining the separation power of liquid chromatography with the sensitivity of spectrophotometry. Recent applications of HPLC to PCP determination reported by Faas and Moore (1979) and Armentrout et al. (1979) have brought detection sensitivity down to $1:10^{9}$. Dougherty and Piotrowska (163) and Kuehl et al. (164) applied negative chemical ionization (NCI) mass spectrometry to screening of environmental and tissue samples for PCP. Analysis of human urine samples involved acid hydrolysis of conjugates, extraction into hexane, and measurement of the NCI spectra; recoveries were 80-98%, and sensitivity was less than 1 ng.

5.4 PCP Impurities

The chlorinated dibenzodioxins and dibenzofurans occurring in commercial PCP have received increasing analytical attention (Sections 3.2 and 3.5). Of the large number of articles published recently on this subject, those of Buser (156) and Buser and Bosshardt (27) are typical. The neutrals from base extraction into petroleum ether were separated by alumina-column chromatography into a fraction containing PCBs or other chlorinated interferences and one containing the dioxins and dibenzofurans. The concentrate was then subjected to gas chromatography on either a small-bore packed column (OV-1, OV-17, or OV-101) or a glass-capillary and its components detected by EC or by mass fragmentometry using the molecular ion. Although responses generally were grouped by number of chlorines, individual isomers could also be determined. Recoveries typically were 80-95% at 0.1-1 µg (0.03-0.3 ppm). HCDD, HpCDD, and OCDD have been efficiently determined at the $1:10^{7}$ level by HPLC (165), and thin-layer chromatography also has been used (166).

Among the other PCP impurities (Section 3.2), the phenoxyphenols (predioxins) have been determined by gas chromatography after methylation (31); the chlorinated cyclohexadienones by gas chromatography (35) or HPLC (52); and HCB by a variety of methods including TLC, HPLC, and EC-gas chromatography (167), the latter with a sensitivity of 0.2 pg. However, quantitative measurement of the individual PCP neutral impurities has been largely restricted to analysis of the technical pesticide itself.

5.5 Analytical Interferences

As with other chemicals, the problem of analytical interferences increases as measurement levels decrease beyond the ppm range. At low PCP levels, Bevenue and Ogata (168) reported that analytical-grade reagents such as sodium hydroxide can contribute substantially to observed PCP residues, as can laboratory glassware and even carefully cleaned sample storage containers. Detrick (169) and Arsenault (1) describe PCP interferences which arose from the general laboratory atmosphere; samples originally thought to contain 0.2-0.4 ppm of PCP eventually were shown to contain less than 0.001 ppm. A number of substances interfere with PCP measurement by gas chromatography, including chloronaphthalenes, PCB (chlorinated

biphenyls), pesticides such as diuron [3-(3,4-dichlorophenyl)-1,1-dimethylurea], and p-methoxytetrachlorophenol which is both a natural fungal metabolite and the product of incomplete methylation of the PCP metabolite tetrachlorohydroquinone.

Analysis of dioxins and dibenzofurans appears even more sensitive to interference. Chlorinated diphenyl ethers, biphenyls, and other substances interfered with both gas chromatographic and mass spectrometric measurements (27,170), and the application of rigorous pre-analytical cleanup is essential. Another major source of interference is the conversion of phenoxyphenols (predioxins) and cyclohexadienones to corresponding dioxins during the analytical process (Section 3.3).

5.6 Conclusions

PCP and its salts and impurities are readily detected and measured by a wide variety of methods. While the less sensitive and specific colorimetric procedures are going into disuse, UV spectrophotometry still is convenient and useful down to the nanogram ($1:10^9$) range. Thin-layer chromatography can be used for semiquantitative work down to this level, and HPLC also is receiving attention.

However, electron-capture gas chromatography presently is the preferred method, especially for the more volatile methyl or acetyl derivatives, and detection limits of $1:10^{11}$ to $1:10^{12}$ are common. Resolution is greatly improved by use of glass capillary columns. These same methods also prove satisfactory for determination of PCP impurities, even including individual dioxin and dibenzofuran isomers whose identities have been confirmed by a mass spectrometer detector.

While measurement of PCP is straightforward, the measurement of most of its neutral impurities is not. First, the equipment required for accurate determination usually is complex and expensive, and the methods themselves are not yet really routine. Second, the number of impurities in technical PCP alone is large, and the opportunities for interference from other contaminants in environmental samples are considerable. Third, the possibility of conversion of phenoxyphenols and cyclohexadienones into either dioxins or PCP during analysis can lead to major inaccuracy unless compensated. The search for the toxic 2,4,5-T impurity, 2,3,7,8-TCDD, has required analytical sensitivity approaching $1:10^{12}$, but the detection level for the toxic PCP impurities still is several orders of magnitude above that figure.

6. RESIDUES OF PCP AND ASSOCIATED COMPOUNDS

6.1 Residues in the Atmosphere

Despite a seemingly low vapor pressure, PCP is quite volatile. It evaporates from the surface of water (85), from treated wood (20), and from painted surfaces (54). For example, volatilization of PCP from painted wood within an enclosed indoor swimming area resulted in its transfer to the water of the swimming pool and the air-conditioning condensate (54). As expected, the PCP levels in the air of an experimentally-treated room varied greatly with temperature and ventilation (1-160 $\mu g/m^3$) (151).

PCP also was found at levels up to 1.7 $\mu g/m^3$ in the air of a wood-treatment plant (171), at least a part of which may have been on minute wood-dust particles (141). PCP also has been associated with atmospheric dust in the outdoor environment, being found at levels of about 7 ng/m^3 in the particulate fraction of air from a city (Antwerp) and at 0.25 and 0.93 ng/m^3 in a similar fraction from an altitude of 5,200 m in the Bolivian Andes (172).

While the relatively low vapor pressure of dioxins and dibenzofurans suggests that they might not produce measurable levels of vapor, they were found (together with PCP) on airborne dust in a sawmill (141) at levels up to 6 ppm. At the low levels to be expected in air samples, compensation for possible analytical interferences will be essential (Section 5.5).

6.2 Residues in Water

It is not surprising that PCP was detected in the raw effluent from a series of wood-treatment plants (173) at levels ranging from 25 to 150 mg/L, but receiving waters outside another plant contained 10-18 mg/L near the outfall and 0.05 mg/L at a distance of 3 km downstream (174). PCP and its degradation products have been shown to persist in bottom sediments long after an accidental spill (175,176).

Domestic and industrial sewage contained µg/L amounts of PCP (177,178), as did river water including that from the Willamette (178) and Weser (155). Wong and Crosby (47) detected low levels of PCP in domestic well-water in Northern California, and Buhler *et al.* (178) found it at 0.1-0.7 µg/L in Oregon drinking water. The atmospheric transport of PCP is reflected in its occurrence in rain and snow (177).

Chlorinated dioxins and dibenzofurans have not been detected in water samples (155), but HCB is widely distributed in aquatic environments although usually attributable to sources other than PCP. However, the insolubility of these neutral contaminants in water suggests that

they would be more likely to precipitate and become mixed with or adsorbed onto sediments to form a reservoir. No work on this subject appears to have been reported.

6.3 Residues in Food

For many years, PCP has been one of a number of pesticides routinely monitored in US raw food in the FDA Market Basket Survey. For example, in 1973-74, 10 out of 360 composite food samples contained PCP at 0.01-0.03 ppm -- one in dairy products, one in cereals, one in a vegetable, and 7 in sugar (179). The next year, 13 out of 240 composites contained PCP (0.01-0.04 ppm), again mostly in sugars (180). However, Doherty and Piotrowska (163) reported that all of a series of random samples of Florida food contained PCP at levels of $1{:}10^6$ to $1{:}10^9$, principally in grain products. PCP also was found in chicken (154) and in fish (181) at low levels, and chloranisoles have been found in chicken (98,182). PCP has been used widely in food processing and packaging (Table 2.4), but there are no data on any resulting food residues.

The dioxin and dibenzofurans associated with PCP have been detected in liver and fat of quarantined cows in Michigan (4); HCDD was present at 0.01-1.3 ng/g, HpCDD at 0.03-12 ng/g, and OCDD at 0.23-47 ng/g. An extensive search for HCDD and OCDD in milk from these animals failed to show their presence at levels above $1{:}10^{10}$ (183), but HCB occurs widely in food (179,180). Firestone (184) found chlorodioxins in commercial gelatin and food-grade fatty acids, apparently derived from preservative treatment of hides.

PCP receives relatively little use as an agricultural pesticide, although its presence in sugar may come from its registered use on cane. However, its occurrence in other food commodities almost certainly is derived by contact with treated storage bins and farm structures, the wood shavings used as bedding, or through processing.

6.4 Other PCP Residues

Other sources of human exposure to PCP have been mentioned by Arsenault (1) and Detrick (169), and an extensive discussion of the subject has recently become available (6). Indeed, from the uses listed in Tables 2.1 and 2.2, one can imagine many potential sources of occupational exposure (Table 6.1), but in no instances have complete residue measurements

TABLE 6.1. Potential sources of occupational exposure to PCP and PCP impurities (4)

Manufacture and shipping of industrial chlorophenols
Sawmills
Wood-treatment plants
Carpentry and other timber and wood-working
Termite control
Agricultural pesticide application
Greenhouses
Industrial cooling towers and evaporative condensers
Treatment and handling of wool
Treatment and handling of burlap, canvas, rope, leather
Paper manufacture
Petroleum and other drilling
Paint and adhesive manufacture and use
Telephone and electrical line work
Dyeing and cleaning of garments

been made. For example, from the results of air- and urine analysis, Wyllie et al. (171) speculated that contact of wood-treatment workers with PCP must be largely dermal, but no surface residue levels were reported; Ferguson (185) reported damage to seedlings grown in PCP-treated flats but gave no information on the residues on the boxes.

Likewise, the widespread use of PCP products must provide frequent opportunities for incidental human contact with the chemical and its impurities in the home, business, and outdoor environments (Table 6.2), but residue data are fragmentary. In the previously cited work of Gebefügi et al. (54), volatilization of PCP within a room whose woodwork had been painted 6 years previously with preservative paint deposited up to 26 ppm PCP on untreated wood surfaces, up to 23 ppm on furniture and curtains, and ppm-level residues on other common household items such as books and recording tape; the painted wood still contained up to 2,800 ppm of PCP. As other examples, Van Langeveld (186) detected PCP in 9 out of 65 toy paints tested in the Netherlands, Levin et al. (141) in Sweden found PCP and the associated dioxins and dibenzofurans in sawdust, Kutz et al. (185) found it in US house dust, Metcalfe found its dioxins in tallow (187), and its use in a US laundry product resulted in the tragic death of babies (137). Surprisingly, however, no general survey of

such residues as sources of PCP release and exposure has been reported to date.

TABLE 6.2. Some possible sources of incidental exposure to PCP and PCP impurities (4)

Smoke from sawmills and burning scrap lumber
Sawdust (fuel, floor covering, particle-board, etc.)
Treated lumber and plywood
Home-treatment of lumber for termite control
Burlap, canvas, and rope
Wool and other textiles
Leather products
Paper products
Contact with adhesives, paint, and painted surfaces.
Used telephone poles and railroad ties
Ornamental wood-chips
Fat trimmed from treated hides (used as feed additive)
Water treated for mollusk control

6.5 Residues in Humans

PCP has been widely detected in human urine (Table 6.3), as well as in blood and fat

TABLE 6.3. PCP levels in human urine

Source (Number of Samples)	Urine; Av ng/ml (range)	Reference
Hawaii (130), occupational	1802 (3-35, 700)	211
Hawaii (117), non-occupational	40 (0-1840)	211
Idaho (6), occupational	164 (41-761)	171
Idaho (1), non-occupational	3.4 (2.6-4.2)	171
Florida (60), non-occupational	20 (10-80)	163
Florida (4), occupational	120 (24-265)	149
Japan (20), general	(10-50)	212
USA (416), general	6.3 (0-190)	11
Florida (6), general	4.9 (2.2-10.8)	149

(137a,138,147,171,189). Of particular interest is the discovery of PCP in human semen and sperm (190). Low levels (mean value 6.3 µg/L) were detected in 85% of urine samples from the general US population, and the urine of people occupationally exposed reached values as high as 37 mg/L. The half-life of PCP in plasma of exposed individuals was 33 hours (1.3 days), and 86% of an administered dose was lost *via* urine (191). However, accurate measurements at these low levels requires special attention to possible analytical interferences (Section 5.5).

Extensive efforts have been made to relate human urinary levels to exposure (3,6). The existing data show clearly that excretion rate is directly related to intake and that human exposure is commonplace. At equilibrium,

$$\text{Exposure level (mg/kg/day)} = \frac{\text{urine level (mg/L) x daily urine volume (L)}}{\text{body weight (kg) x 0.86}}$$

Although exposure could be expected to be highly variable, both measured and calculated values for workers indicate a strong inhalation as well as dermal component (6, 171). Urinary excretion of a single above average dose follows the expression $C_t = C_o e^{-0.503t}$ where the concentration after a t time interval (C_t) is a log function of the maximum concentration C_o; that is, a surge to 65 µg/L would return to 5 µg/L in about 6 days. Typical (calculated) exposures for the general US population (0.14 µg/kg/day), for the population of Hawaii (1 µg/kg/day), and for wood-treatment workers (35 µg/kg/day) have not been substantiated by measurements.

In general, the PCP impurities have not been reported to occur in humans, with the exception of HCB which has been found widely in human fat and milk (192-194). However, although the differences in physical and chemical properties between PCP and dioxins indicate that they will not share the same pharmacokinetics, the distinct pharmacokinetic and metabolic differences between humans, rats, and monkeys in the case of PCP suggest caution in extrapolation

of data from animal models to man.

6.6 Alternate Sources

Residues of PCP and associated compounds do not necessarily arise only from the production or uses of pentachlorophenol (Table 6.4). A number of other chemicals, including some

TABLE 6.4. Alternate sources of PCP residues

Source	References
1. Metabolism of HCB	195, 213-216
2. Metabolism of pentachlorobenzene	195, 216
3. Metabolism of PCNB fungicide	217, 218
4. Metabolism of BHC isomers	196, 197, 219
5. Chlorination of phenols in water	169, 198
6. Dechlorination of cyclohexadienones	52

common pesticides, are metabolized to PCP by plants, animals, and/or microorganisms, but the extent of this contribution to the environmental residues remains uncertain. In particular, metabolism of γ-BHC (lindane) has been shown to form PCP in rats (195), humans (196), and higher plants (197), and HCB is converted to PCP in rodents, chickens, fish, monkeys, and other organisms (Table 6.4).

Detrick (169) reported that chlorination of a 1 ppm aqueous solution of phenol produced detectable PCP, and Smith et al. (198) showed the aqueous chlorination of TCP to PCP. Although the suggestion (169) that PCP might be a natural product remains unsupported, several closely related compounds including 1,4-dimethoxy-and 1-hydroxy-4-methoxy-2,3,5,6-tetrachlorobenzene, and 2,3,5,6-tetrachloroanisole reportedly are biosynthesized by microorganisms (199-201).

Likewise, the higher chlorinated dioxins and dibenzofurans seem not to be naturally-occurring, although traces of TCDD have been reported as pyrolysis products from many common sources (202). However, pyrolysis or photolysis of phenoxyphenols undoubtedly is the principal alternate source of these compounds (Section 3.3). Environmental HCB has many alternate sources, including several pesticides (where it occurs as an impurity) and manufacturing processes such as electrolysis (203).

6.7 Waste Disposal and Decontamination

Considering the large worldwide production and use of PCP, the intentional distribution of PCP-containing wastes looms as an increasingly important issue. For PCP in waste-water, 96% is removed by an activated sludge process (204) and use of ozone in the presence of UV light has proved effective under experimental conditions (205). Other common methods of waste disposal such as open pits, lagoons, landfill sites, on-site burning, deepsea burial, or deep-well injection are not recommended because of the mobility and toxicity of PCP (206), although they obviously are in widespread present use (18). High-temperature combustion (800°C) is effective (207), although OCDD formation is detectable at high loads.

When purified by distillation, each metric ton of PCP results in 200 kg of still-residue containing dioxins, dibenzofurans, and other neutral and acidic impurities. These are indicated to be completely destroyed on a commercial scale by incineration at 1,200°C (4). TCDD contamination levels were sharply reduced by exposure to sunlight in the presence of an organic hydrogen-donor (olive oil) (208), but this procedure has not been reported for PCP impurities. HCB and other stable chlorinated hydrocarbons have been destroyed by commercial-scale high-temperature oxidation (206).

The disposal of treated wood and other PCP-containing products raises another problem. PCP is poorly combusted at low temperatures (207), so that attempts to burn treated wood results in volatilization of the PCP and conversion to dioxins (at least to OCDD) (48,59,209,210). Despite their seeming low volatility, the neutral PCP impurities may also be expected to volatilize or to be dispersed as airborne particles under these conditions.

6.8 Conclusions

PCP has been detected widely in the environment -- in air, water, soil, food, and in man. It seems probable that the volatility of PCP is the principal factor in its worldwide distribution, that the atmosphere is the main route of dispersal, and that inhalation represents a significant route of human exposure.

The levels of PCP in water and food are so low, and the compound is so readily metabolized, that ingestion must contribute only a small proportion of the human body load. On the other hand, such a large number of people reveal continuous low-level excretion of PCP, even when not occupationally exposed, that some other relatively continuous source must be presumed. In fact, exposure is almost inevitable, considering the extent to which PCP is used in wood preservation and in a wide variety of common household items. While no data seem to exist on PCP levels in the average home, an investigation of PCP distribution in an intentionally-treated room indicates that most people's exposure occurs rather incidentally within their own homes and work-places by inhalation and skin contact. In addition, continuous exposure to HCB and BHC isomers probably provides a significant proportion of the observed urinary PCP residues through metabolism. Further, the contribution of interferences to analytical error cannot be ignored.

Data on the environmental occurrence of the dioxin and dibenzofuran impurities presently are almost nonexistant, perhaps in part because suitable analytical techniques are both relatively new and expensive. As expected, dioxins are found as a direct consequence of wood treatment, but the attempts to detect them elsewhere in the environment (such as in milk) indicate that they have not been present down to the limit of detectability.

7. SUMMARY AND GENERAL CONCLUSIONS

PCP (pentachlorophenol) and its derivatives are produced on a multimillion kilogram annual scale and receive virtually worldwide use as preservatives, disinfectants, and pesticides which come into direct contact with humans. PCP also can be generated in the environment during water chlorination and by degradation of other pesticides. However, accurate and detailed data on specific use patterns or general levels of production, distribution, and consumption generally are not available, even in nations having highly developed regulatory systems. Clearly, such information is essential to clear understanding of any toxic hazards which may exist.

PCP is acutely toxic to all forms of life. Moreover, the technical product can contain up to several percent of neutral impurities which include the toxic chlorinated dibenzodioxins, dibenzofurans, hexachlorobenzene, and cyclohexadienones responsible for the observed chronic symptoms; the reported levels vary with the date, route, and source of manufacture and the method of analysis. PCP is readily absorbed via dermal, oral, and inhalation exposure in animals and man, but little information exists on plant uptake despite its obvious herbicidal properties. Technical PCP also contains up to 10% of other chlorinated phenols, mainly tetrachloroisomers, whose toxicological significance is poorly understood.

PCP is volatile, tightly adsorbed to most surfaces, and a relatively strong organic acid, but the influence of these properties varies with the pH of the surrounding environment; they do not apply to the anionic forms (such as NaPCP). Despite its high degree of chlorination, PCP is degradable in the environment. It is rapidly and completely degraded by exposure to sunlight, microbially degraded in soil, and undergoes metabolic detoxication by conjugation and/or dechlorination in animals and higher plants. Although no data are available on the chronic uptake of PCP from the environment by terrestrial animals (other than man), fish bioconcentrate it about 1,000-fold. Single oral doses are rapidly cleared at first, but as much as 11% of the initial dose is retained by mammals and 30% by fish and eventually is released at a much slower rate; the nature of this retention is not clearly understood. Soils, and presumably higher plants, also can retain a proportion of PCP as parent or metabolites, but consequent availability to other organisms remains uninvestigated. Clearly, extensive additional radiochemical investigations are warranted.

The chlorinated dioxin and dibenzofuran impurities are of particular concern. Although the octachlorodibenzodioxin (OCDD) is not especially toxic, the hexa- and heptachloro compounds -- usually present at a total of over 30 ppm -- can be considered as potentially dangerous through oral or dermal exposure; no inhalation data have been reported. The most toxic homolog, 2,3,7,8-tetrachlorodibenzodioxin (TCDD), is absent within current limits of detection, although investigation suggests that its less toxic isomer(s) do occur in some PCP samples at levels up to 0.25 ppm. The composition of technical PCP -- at least that produced in the USA -- is known in detail, but relatively little toxicological or environmental residue data exist for the impurities except for HCB.

Their physical and chemical properties differ from those of PCP; although they, too, can be degraded by light and are adsorbed strongly, little is known about their environmental persistence and fate. They may be expected to bioconcentrate, be only slowly metabolized by animals or microorganisms, and accumulate in the livers of animals; however, although PCP has been sought (and found) in a variety of environmental situations, similar data on PCP impurities and conversion products are almost nonexistent. In addition, dioxins are known to be generated from PCP photochemically, by burning of treated wood, and via the corresponding predioxins, but any contribution of these sources to human exposure has not been evaluated.

Satisfactory methods are available for the analysis of PCP, at levels of $1:10^{10}$ or better, using acetylation or methylation followed by electron-capture gas chromatography. The impurities also may be determined at similar sensitivities with common electron-capture or mass-spectrometer detectors, but measurement in the range of $1:10^{11}$ or less requires unusually expensive and sophisticated mass spectrometry facilities. At low levels, however, results often are obscured by interferences arising from pesticides, other environmental contaminants, and even impurities from glassware and common laboratory reagents. While there is an obvious need for environmental analysis at sensitivity levels commensurate with toxicity, better verification of residue identity and careful standardization at residue levels will be essential to interpretation of possible hazards from traces of these highly toxic substances. Relatively little attention has been devoted to simplified analytical methods for residues of either PCP or its impurities.

Away from areas of its immediate use, most reported environmental residues of PCP fall in the $1:10^7$ to $1:10^{10}$ range. The occasional residues in food (typically 0.01-0.03 ppm) are found principally in milk, grain products, or sugar; PCP is seldom used on food crops, but its presence in water, rainfall, lumber, and bedding material may account for the rather widespread occurrence reported in human urine. PCP levels in treated wood are very high (typically 5,000 ppm), and the substance volatilizes readily from surfaces into the surrounding air from which it is redistributed and absorbed dermally and, especially, by inhalation. The PCP residues observed in human urine, even from people not occupationally exposed, could be due in part to this general environmental background as well as to metabolic generation from such common substances as lindane and hexachlorobenzene. Structural verification of PCP or its derivatives, definition of sources of analytical error, and relation of PCP residues to such alternate sources are essential early steps in authenticating possible human and animal exposures.

The exposure, uptake, and excretion of PCP by workers has been repeatedly confirmed. There is no question that PCP itself can represent a serious toxic hazard to unprotected, occupationally-exposed people; in fact, it may be considered dangerous under any conditions -- such as medicinal use, air in treated buildings, or on indoor wood surfaces -- where it is not subject to photochemical or microbial degradation. The extensive apparent occurrence of PCP residues in the general population and its observed retention in animals suggests possible widespread distribution and requires immediate further investigation. However, the toxicological significance of PCP impurities still remains circumstantial; so far, there is little evidence of environmental distribution, human exposure, or intoxication attributable to them. Although their expected low levels may complicate most efforts at detection, the fact that they are highly bioconcentrated -- especially in liver -- provides an obvious means to evaluate human and animal exposure.

While there actually is little evidence that low level environmental exposures to PCP present an imminent hazard to man or animals, the long history of acute intoxication by, and the wide use of, technical PCP -- often by untrained people -- reflect a clear hazard. On the other hand, PCP long has proven its usefulness as a cheap and effective preservative and pesticide, especially in the developing tropical nations. No truly satisfactory alternatives have been found for its major uses, and, for the present, improved safety may have to rely on better formulation, user education, and restriction of certain low-priority, high-contact uses such as indoor paints, laundry products, and cosmetics. For the future, the technology now exists to reduce neutral impurity levels to a fraction of 1% by purification. However, the chemical mechanisms by which these impurities form remains unclear; it seems probable that an understanding of them could lead to substantial processing improvements and greatly improved PCP quality without a concurrent problem of toxic waste disposal associated with present methods of purification.

8. RECOMMENDATIONS

The following recommendations are intended to implement three general objectives:

The exposure of humans and economic animals to PCP and its impurities must be defined and minimized.

The indiscriminate use of PCP products (e.g., most home uses) should be limited, in favor of essential uses.

The quality of technical PCP should be improved.

1. A worldwide survey of PCP production and use patterns should be established by an international agency in order to better define environmental inputs and potential exposure.

2. Further efforts to define PCP impurities should be encouraged, authentic standards of the major toxic impurities made available through commercial channels, and methods for their

specific determination simplified and standardized to permit more extensive analysis.

3. Commercial PCP from a wide range of identified existing sources should be analyzed for specific neutral impurities, including toxic dioxin and dibenzofuran isomers, and such analyses should become routine by manufacturers to provide a basis for estimating chronic health hazards and improving the quality of specific PCP products.

4. Commerically purified PCP and technical PCP should be compared and evaluated in relation to possible hazards under recommended conditions of use, rather than by composition only, to determine the desirability of purification in relation to the increased costs.

5. The chemistry of impurity formation during PCP production should be investigated in relation to possible improvement of the manufacturing processes, as an alternative to postmanufacture purification.

6. The presence of the chlorinated dioxins and other chlorophenyl ethers and their conversion products should be verified in man and animals and then surveyed in relation to possible sources of exposure.

7. The presence and fate of PCP and its impurities should be determined in sediments, air, water, and treated products under conditions of normal use in order to relate their persistence or movement to potential human exposure.

8. The presence of PCP and its metabolites in human urine and blood should be verified, their levels monitored, and the sources specifically identified with the purpose of controlling them.

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