GENERAL POPULATION EXPOSURE TO PENTACHLOROPHENOL

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General population exposure to pentachlorophenol (PCP) is considered universal. PCP is an ubiquituous chemical found in air, water, soil, food, and house dust as well as in many consumer products. Exposure has been estimated by measurement of PCP in the various media to which people are exposed as well as by modeling based on measurement of PCP in human urine, serum, and various body organs. Four recently published estimates of average population exposure are in reasonable agreement with values of 11 to 23 μg/day).

Previous exposure estimates have not considered exposure to PCP in house dust which appears to be considerable for some individuals and is currently under investigation. They have not considered the exposure of the most susceptible or highest risk population groups: children, pregnant women, the elderly, and the infirm. PCP body burdens are usually highest in the youngest occupants of homes with PCP-treated wood. PCP is a known teratogen, and recent studies indicate that PCP is a potential human carcinogen.

The knowledge gained during the 1970s and 1980s regarding the toxicity of commercial grade PCP has led to its restriction in many developed nations including the United States. In spite of the restrictions, production and use of PCP continues at a substantial fraction of the pre-restriction levels. And PCP already in the environment generally persists for more than five and up to thirty years.

The contribution of house dust and indoor air to human exposure, the distribution of PCP in homes not known to contain PCP-treated wood, and the effectiveness of various control strategies are not adequately understood at this time. Therefore, effective strategies to protect susceptible populations should, but cannot yet, be developed. Further research is necessary to improve our understanding of general population exposure to PCP and of its control in indoor environments.

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INTRODUCTION

General population exposures to commercial grade pentachlorophenol (PCP) and its common contaminants are substantial due to the widespread use and long environmental residence times of this pesticide. PCP is a broad-spectrum biocide which has been used not only for wood preservation but also as an herbicide and fungicide in a wide variety of consumer, agricultural, and industrial products.[1,2,3,4]

Commercial forms of PCP and Na-PCP (the water soluble salt) may be contaminated with PCDDs, PCDFs, and hexachlorobenzene (HCB), which are considered the principal sources of its toxicity. (In the remainder of this paper, PCP refers to commercial preparations of PCP and Na-PCP unless otherwise specified.) Commercial PCP preparations typically contain 85-90% PCP, 4-8% tetrachlorophenol (TCP), <0.1% trichlorophenol, 2-6% higher chlorophenols, and $\mu g/g$ concentrations of PCDDs and PCDFs which generally increase with the number of chlorine atoms. OCDD concentrations typically range from 500 to 2,500 $\mu g/g$.[1,2]

Production and Uses

Precise production quantity estimates are not available. In the early 1980s, production was estimated at 50 to 90 x 10^6 kg worldwide, 23 x 10^6 in the United States. More recent estimates have placed production at 17 to 30 x 10^6 worldwide, 12.7 x 10^6 in the United States.[1,5,6,7] The reduced production in recent years is attributed to restrictions on PCP use imposed by most developed countries, especially for agricultural and domestic applications. While PCP is used primarily for commercial wood preservation, its domestic use is of concern due to the possible health hazards associated with indoor application of wood preservatives containing PCP.[7]

PCP has been widely used in various wood finishes including stains and in other products incorporated into residential and commercial construction.[2] Until its withdrawal from the domestic consumer market in the mid-1980s, many widely-used wood stains and preservatives contained PCP.[2,4] It is estimated that 30,000 log homes had been built in the U. S. by 1978. At that time the logs were generally treated with 5% PCP in an organic solvent to control fungal discoloration.[8] No estimate of the current number of PCP-treated log homes is available. The food chain contains significant PCP residues due to its wood preservative use for buildings and agricultural containers; and contamination of water, air and soil where food is produced; and contamination of water where fish are obtained for human consumption.[1,2,7]

Human Exposure

Human exposure results not only from PCP use as a wood preservative but also from contamination of soil, water, and food. The frequency of detected human PCP body burdens in a representative sample of the U. S. population was second only to DDT frequency among the pesticides monitored in the Second National Health and Nutrition Examination Survey, 1976-1980 (NHANES II). PCP was detected in 79% of the population 12-74 years of age at an estimated average urinary concentration of 6.25 x 10^{-3} mg/liter.[9,10] Body burdens (serum and urinary PCP concentrations) are elevated several-fold in portions of the U. S. and in other nations where wood preservation and other biocidal requirements are enhanced by warm, humid climate conditions.[11] (Compare data for Hawaii, Spain, and Nigeria to other locations reported in Table I.)

Toxicity

PCP has been recognized as a fetotoxin since the mid-1970s, and as a teratogen since 1974.[12,13] In 1989, the National Toxicology Program found evidence of carcinogenic activity in male and female $B6C3F_1$ mice in separate feed studies of technical-grade pentachlorophenol and of Dowicide EC-7^R. For technical grade PCP, there was "clear evidence" in the male mice and "some evidence" in the female mice for carcinogenicity. For Dowicide^R EC-7 there was "clear evidence" in both sexes.[5] Its toxic action has been attributed to the common contaminants of its commercial preparations.[2,4]

The National Academy of Science's proposed acceptable daily intake (ADI) for PCP is 180 μg/day.

HUMAN EXPOSURE ESTIMATES

General population intake of PCP has been estimated by various methods. The resulting estimates range from 11 to 23 μg/person/day. Several recent estimates are described below.

A Six-Compartment Environmental Partitioning Model

Using a six compartment environmental partitioning model, Hattemer-Frey and Curtis explored transport and accumulation of PCP within and between various environmental media. They applied the results of the model with measured background environmental concentrations to estimate rates of exposure and obtain an average, long-term daily PCP uptake estimate of 16 μg/day.[14]

In performing their analysis, Hattemer-Frey and Travis calculated an inhalation uptake of 0.003 μg/day from air based on rainwater PCP concentrations in Hawaii (an assumed PCP concentration of 1.4×10^{-7} μg/g (1.55×10^{-3} μg/m^3) and inhalation of 15 m^3 of air/day.[15] They estimated uptake from water at 0.012 μg/day based on a PCP water concentration of 8.2×10^{-6} μg/g. The model did not include estimating uptake by skin absorption or inhalation of PCP from dust or indoor air. They concluded that "the food chain, especially fruits, grains, and vegetables, accounts for 99.9% of human exposure to PCP."[14]

One-compartment Linear Pharmacokinetic Model

Geyer et al used a one-compartment pharmacokinetic model based on PCP concentrations in human adipose tissues of West Germans to derive an estimated average PCP uptake from ingestion of 19 μg/day. This value was based on an assumption of uptake by ingestion only. The authors said their estimate is in agreement with values calculated from published measurements of urinary PCP in West Germany (16 μg/day). The authors concluded that "the main route of PCP uptake by the non-occupationally exposed human in the FRG is mainly by ingestion and not by inhalation or percentaneous [sic] absorption of PCP."[16]

Hattemer-Frey and Travis compared the uptake estimate from the environmental partitioning model to one they obtained by calculating long-term average intake of PCP using a linear, one-compartment pharmacokinetic model (after Geyer et al). Their estimate was based on a 20-day half-life for PCP in the human body, 14.7 kg of body fat (21%) for the average human weighing 70 kg, with 17.4 μg PCP per kg fat, the mean concentration for the general population of the U. S. This calculation resulted in an estimated daily PCP uptake of 23.3 μg/day.[14]

Exposure Estimates from Urine Concentration Models

Many investigators report that PCP urine concentrations are indicative of uptake.[1,14,17,18] Mean or median urinary PCP concentrations are approximately 1 mg/liter for exposed workers, ca. 0.04 mg/liter for for persons exposed non-occupationally, and ca. 0.01 mg/liter for the general population.[7]

Residents of log homes and workers or residents of other buildings where PCP has been used are defined as "non-occupationally exposed." Crosby proposed that daily PCP intake can be estimated at equilibrium by equation (1).

$$\text{Intake } (\mu g/\text{day}) = \frac{\text{urine concentration } (\mu g/\text{liter}) \times \text{urine vol } (\text{liter/day})}{0.86} \quad (1)$$

where 0.86 is a correction factor for the fraction of total body burden that is eliminated in urine.[1] Uhl et al confirmed the correction factor at 0.86 by administering oral doses to volunteers without known PCP exposure.[18]

Tables I and II list selected urinary PCP concentrations, sera PCP concentrations, and calculated daily PCP uptake for various population groups. There are some problems with comparing results from different investigations and time periods. Analytical improvements in recent years have lowered the detection limits. The use of different methods and reduced detection limits make comparability of diverse analytical data questionable, "particularly when urinary-PCP levels have been determined without hydrolysis."[7] Tables I and II list studies in reverse chronological order from (top to bottom); the newness of reports may be a crude indicator of the quality of analysis. The uptake calculations are made using equation (1) and assuming a 1.4 L daily urine output for a 70 kg person (ICRP reference man).[19]

Table I presents data for various population groups without known PCP exposure. The range of average calculated uptake is 5.5 to 10.3 μg/day excluding reports from areas where PCP use is elevated due to climatic conditions conducive to microbial growth. When reports from Hawaii, Nigeria, and Barcelona, Spain, are included, the maximum calculated average intake value is 40.7 μg/day (range = 28 to 4,347 μg/day).

Table II presents data for individuals with known non-occupational exposure. The averages range from 11 to 137 μg/day. The calculated maximum PCP uptake rates for all listed urinary PCP concentrations range from 49 to 1,172 μg/day. No adjustment has been made for metabolic and weight differences between adults and children.

Hattemer-Frey and Travis compared their environmental partitioning estimate to one based on urinary PCP. Using the average urinary PCP concentration reported by Dougherty and Piotrowska, they calculated an average, long-term daily PCP intake of 11 μg/day.[20,14]

Estimating Uptake from PCP in Air

Ambient air in urban areas of developed countries typically contains several ng/m^3 while concentrations in less developed areas are typically an order of magnitude lower.[6] Recently published papers report PCP air concentrations near 0.001 μg/m^3 in remote areas (Bolivian Andes), about 0.005 to 0.01 g/m^3 in urban air (Antwerp).[21] Reported indoor air concentrations in Europe and the United States from residences and other buildings not known to be contaminated range from 0.01 to 0.1 μg/m^3.[17,22,23] Concentrations from 0.2 to 50 μg/m^3 have been reported from buildings containing PCP-treated wood.[22,23,24,25]

Estimates based on PCP intake from inhalation should apportion time spent between indoor and outdoor environments[26]. Assuming 3 hours outdoors and 21 hours indoors and inhalation of 15 μg/m^3 per day with PCP air concentrations of 0.005 to 0.01 μg/m^3 outdoors and 0.05 to 0.1 μg/m^3 indoors yields a range of uptake estimates from 0.67 to 1.33 μg/day and an average of 1.0 μg/day.

Inhalation Worst Case Estimates

An active adult spending 24 hours/day in a house with levels typical of many PCP-treated homes might represent a worst case scenario. Assuming a daily respiratory volume of 20 m^3/adult-day and a 100% uptake of PCP from the air (worst case assumption), a person living in a home with air concentrations in the 1 to 10 $\mu g/m^3$ range could receive 20 to 200 μg/day from inhalation. The worst case inhaled uptake for a child might be half the adult value, assuming one-quarter the body weight and twice the metabolic rate.

PCP Ingestion and Absorption from Dust - Worst Case Estimates

No data are available for average dust exposure or PCP absorption rates from dust. Assuming a maximum daily pre-schooler dust ingestion of 0.2 g/day with 1000 μg/g PCP, a pre-schooler would ingest 200 μg/day. Assuming an upper bound for dust contact of 50 mg/day for 2 - 5 year olds, and assuming 100% dermal absorption results in an uptake of 50 μg/day.

DISCUSSION

Little is known about the actual distribution of PCP exposures. Data from the U.S. indicate that persons living in Hawaii have several-fold higher average urine levels than persons living on the mainland. Based on the urine model, average exposures do not appear to vary greatly among developed nations. The limited data available suggest that exposures in some developing nations may be significantly higher than in the more developed nations.[7]

Virtually every reported measurement involving children indicates that younger members of the population experience larger exposures than older members. This is the case within families and within communities. The larger exposures by children are not adequately explained, although some authors have suggested that metabolic differences are involved. Serum and urinary PCP of children in PCP-treated log homes and in control groups are higher than in adults and usually highest in the youngest age groups.[7,8,22] It is likely that differences in exposure through skin absorption or inadvertent ingestion of PCP on surfaces and in house dust are an important route of exposure for children.

Several studies of occupants in residences containing PCP-treated wood indicate that women have higher body burdens than men. It is often assumed that this results from the higher percentage of time women spend in the homes, although few of the reported PCP studies provide information on the time budgets of the subjects. Since pregnant women are likely to spend large amounts of time at home, more information on residential exposure is important. Young children, the elderly, and infirm members of the population are also subject to larger exposures if the residential environment is an important source.

Outdoor Exposures

The diverse, ubiquotous historical use and long environmental residence times of PCP make possible a large variety of potential exposures.[1,2] PCP has been extensively used to treat fence posts, utility poles, wood decks, and stairs. Playground equipment, picnic furniture, and outdoor wood seating may also be sources of PCP exposure via skin absorption and inadvertent ingestion, especially where warm weather results in clothing ensembles with less skin coverage.

Indoor Environmental Contamination

Little is known about the distribution of PCP in indoor air, on interior surfaces, and in house dust in the United States. No systematic studies of the occurrence of PCP in the U. S. building stock have been performed. Studies in buildings treated with PCP have been reported ranging from 0.04 $\mu g/m^3$ to 50 $\mu g/m^3$. [7,17,24,25]

In a pilot study in four Seattle residences, Roberts et al found a mean concentration of 4.8 $\mu g/g$ in house dust. This study evaluated methods for assessing exposure to pesticides in house dust. PCP was found in house dust at a level of 4.8 $\mu g/g$ (mean). The authors hypothesized the source as being from decks outside two of the homes. These decks were treated with PCP three and five years previously.[27] House dust concentrations at levels to 1000 $\mu g/g$ levels were reported in another study.[28]

Krause et al surveyed 217 households in the Federal Republic of Germany to determine pesticide use and occupant exposure. They found that PCP and lindane were "freely used for domestic purposes" in 90% of the households. The quantity of commercial grade PCP used varied from 50 g to 10 kg per household. Indoor air samples from 104 households showed a median PCP concentration of 5 $\mu g/m^3$. Dust samples from houses where PCP had been used contained a median PCP concentration of 13 $\mu g/g$ compared to 0.008 $\mu g/g$ in control houses. In a follow-up study seven years later, PCP urine concentrations in the exposed group remained higher (three times) than in the control group.[17]

Gebefügi et al found that the surfaces of all analyzed materials in a test house in the Federal Republic of Germany where PCP had been applied six years earlier were contaminated. Samples of the treated wood contained 1,570-2,750 $\mu g/g$ in the topmost 1.5 mm layer and 120-340 $\mu g/g$ in the layer 3.0 to 8.0 mm below the surface. Samples of untreated wood contained 15-26 $\mu g/g$ in the topmost 1.5 mm layer and 2.5-7 $\mu g/g$ in the layer 3.0 to 8.0 mm below the surface. Wallpaper, carpet, and curtains contained 2.2 to 14.2 $\mu g/g$, and other objects in the room contained similar concentrations.[29]

Skin Absorption, House Dust, and Urinary PCP

In an investigation in Italy, the PCP in dust from houses of six tannery workers varied between 2 and 11 $\mu g/g$. The PCP house dust concentrations in homes with treated interior wood ranged from 20 to 166 $\mu g/g$. The investigators found a correlation between PCP in house dust and in occupants' urine when the urine levels exceeded "base values ($\leq$ 20 $\mu g/L$)" [pre-exposure values].[30]

Some authors have reported positive correlations between PCP air levels and PCP house dust levels.[30] Krause et al found a strong correlation between PCP house dust concentrations and the PCP urine concentrations of occupants but no correlation between PCP urine concentations and PCP air concentrations.[17] Further studies of PCP in house dust are warranted due to the ease of sample collection and the reliability of house dust as an indicator of exposure.[28,30]

CONTROL MEASURES FOR PCP

Mitigation measures for general population exposure have not been systematically studied. Measures recommended in the past to control PCP in indoor air have not proven as effective as previously believed. Specifically, there is some evidence that sealing PCP-treated wood has not been as effective as expected in reducing PCP concentrations in indoor air.[23,25]

Levin and Hahn questioned the efficacy of sealing wood that had been pressure-treated with PCP. And they reported cracking and the "blooming" of PCP crystals in PCP-treated structural "glu-lam" timbers sealed with two coats of polyurethane varnish.[25] Hosenfeld et al did not show a significant difference in PCP air concentrations between sealed (0.218 $\mu g/m^3$) and unsealed (0.173 $\mu g/m^3$) PCP-treated log homes. However, neutralization of PCP-treated logs with a chemical made for the purpose of reducing emissions did result in a significant reduction in air levels from 0.308 $\mu g/m^3$ (not neutralized) to 0.136 $\mu g/m^3$.[25] Cline et al reported a significant decrease in body burdens of residents living in PCP-treated log homes that had been sealed. No details were given regarding the sealant or its application method.[8]

Neither regulatory nor technical assistance programs are in place in the U. S. that can significantly reduce population exposure to PCP indoors from pre-regulation applications. The recent new restrictions on the use of PCP cannot significantly reduce general population exposure in the short term due to the widespread distribution and relatively long persistence indoors.

Remedial Action Research Needs

More research on effective control of PCP indoors is necessary to reduce general population exposure to PCP already incorporated in habitable structures. Remedial actions used to control radon levels in indoor air may be effective in reducing some PCP exposure from wood preservatives used in foundation, crawl space, and basement construction. A better understanding of the distribution of exposures will assist in targeting mitigation efforts at population groups at serious risk and at the most highly exposed individuals.

CONCLUSION

Recent published estimates of human exposure to PCP have failed to consider important exposure routes including air and dust. Some authors have attributed most PCP uptake to exposure through dietary contamination without adequately considering other important routes of exposure. There has been inadequate investigation of the mechanism of exposure and uptake, especially in younger members of the population.

Several important issues related to the control of PCP require further elucidation before effective remedial action can be initiated. The increasing evidence of PCP toxicity supports the need for improving our understanding of total human exposure to PCP and its effective control.

Table I. Levels of PCP in sera and urine and calculated PCP uptake for the general population (no known exposure).

Sample size	Country	Serum, mg/liter mean (range)	Urine, mg/liter mean (range)	Ref/Year	Calculated PCP uptake, µg/day mean (range)[a]
207	West Germany		0.013 (0.004 - 0.021)	[17]/1989	21 (6 - 34)
34	USA	0.040 (0.015 - 0.075)		[8]/1989	
143	USA		0.0034 (0.001 - 0.017)	[8]/1989	5.5 (0.2 - 28)
197	USA[b]		0.014	[31]/1989	22.8
6	Italy		0.0086 (0.0049 - 0.0125)	[30]/1987	14
100	Spain	0.0219 (0.0025 - 0.1165)		[32]/1987	
50			0.025 (0.004 - 0.136)		40.7 (6.5 - 221)
40	Nigeria	na[c] (trace-0.0213)	na (0.025 - 0.23)	[33]/1985	na (40.7 - 374)
418	USA	na (ND - 2.67)	0.0063 (na - 4,347)[d]	[10]/1983	10.3
37	USA		0.0044	[34]/1984	7.16
32	USA[e]		0.030	[11]/1980	48.8[f]
42	USA	na (0.0043 -0.0679)	na (0.007 - 0.011)	[22]/1980	

[a] Calculated by Levin using equation (1)
[b] Hawaii
[c] na = not applicable
[d] The authors reported no effort to screen study participants for occupational or other known PCP exposure in their random, stratified population sample.
[e] Arkansas children 2 - 6 years of age
[f] Not adjusted for weight or metabolic rate

Table II. Levels of PCP in serum and urine and estimated long-term daily PCP uptake of non--occupationally exposed persons.

Sample size	Country	Serum, mg/liter mean (range)	Urine, mg/liter mean (range)	Ref/Year	Calculated PCP uptake, µg/day mean (range)[a]
250	West Germany		0.044 (0.013 - 0.071)	[17]/1989	72 (22 - 115)
123	USA	0.0420 (0.069 - 1.340)		[8]/1989	
118	USA		0.0069 (0.001 - 0.340)	[8]/1989	11 (1.6 - 553)
12	Sweden		0.0132 (0.0023 - 0.0299)	[35]/1989	21 (3.7 - 49)
57	USA	0.0476 (0.007 - 0.168)	0.021 (0.005 - 0.179)	[23]/1986	34 (8 - 291)
146[c]	West Germany	0.038 (0.002 - 0.980)	0.030 (0.002 - 0.720)	[36]/1985	49 (3 - 1,172)
37	USA		0.050	[34]/1984	81
32	USA	0.330	0.0127	[22]/1980	21
5	USA	1.126 (0.580 - 1.750)	0.084 (0.0468 - 0.216)	[22]/1980	137 (76 - 352)

[a] Calculated by equation (1)

[b] Persons selected for the study on the basis of elevated urinary PCP or other risk factors.

[c] Persons who had used PCP-containing wood preservatives indoors in the prior 7 to 15 years. PCP was not used indoors after 1977.

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