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Clinical Findings in Workers Exposed to Pentachlorophenol

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Abstract. Comparative findings are presented on the health and exposure status of groups of individuals in Hawaii with and without occupational exposure to pentachlorophenol (PCP). Occupational exposure to PCP occurred through employment at firms engaged in the treatment of wood with either PCP alone or PCP plus other compounds as preservative chemicals. Mean serum levels were 0.32 ppm for 32 control individuals, 1.72 ppm for 24 workers exposed to PCP and other wood preservative chemicals, and 3.78 ppm for 22 workers exposed to PCP as the sole preservative chemical.

Age-standardized prevalence rates were significantly higher among the PCP-exposed than among the controls for low-grade infections or inflammations of the skin and subcutaneous tissue, protective membrane of the eyes and the mucosa membrane of the upper respiratory tract. Strong to moderate statistical associations were observed between PCP exposure and increased occurrence of bands (immature leucocytes) and basophils, increased plasma cholinesterase, alkaline phosphatase, gamma globulin and uric acid, and decreased serum calcium. Despite these statistical associations, laboratory values considered to be clinically abnormal were few and not significantly greater in occurrence among the PCP-exposed individuals.

During the seven year period between 1967 through 1973 a major effort was made to monitor the health of over 400 residents of Hawaii who were in large part occupationally exposed to pesticides. The work was done by the Hawaii Community Studies Project as part of a federally sponsored nationwide program of research initiated in 1965 by the U.S. Public Health Service to determine whether exposure to pesticides at home or at work was injurious to health (Acosta 1965).

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The volunteer participants of the Hawaii study were given extensive annual medical examinations which included a battery of biochemical, organ function, and behavioral tests. Comparative data on pesticide exposure were collected subjectively through information on worker contact with pesticides and objectively by repeated measurements of concentrations of organochlorine pesticides (including PCP) in blood samples collected from the participants. Concentrations of PCP also were determined in urine samples.

The large volume of data on this study has undergone processing and a number of the findings have been published (Klemmer 1971, Rayner *et al.* 1972, Takahashi *et al.* 1976, Rashad *et al.* 1976, Begley *et al.* 1977, Budy *et al.* 1977, Korsak and Sato 1977). Comparative findings are reported herein on groups of workers with and without occupational exposure to PCP. This publication has been preceded by a final report (Sato *et al.* 1978) issued to the Human Effects Monitoring Branch, U.S. Environmental Protection Agency and to the American Wood Preservers' Institute.

Clinical and Laboratory Methods

Physical examinations were conducted either by project physicians or by staff at local clinics and were given annually to all participants who elected to remain in the study for more than one year. Blood chemistry and urinalysis testing was done at the project laboratory or at local clinical laboratories by methods approved by the sponsoring agency (Smith 1969) and in conformity with a federally supervised quality control program. Pesticide residue analyses were done at the project laboratory by methods prescribed by the sponsoring agency (Burchfield *et al.* 1965, Thompson 1974). Earlier research done by the Hawaii project had led to the development of a sensitive method for the measurement of PCP in human urine (Bevenue *et al.* 1966, Bevenue *et al.* 1967a, 1967b). This, as well as the later development of a method for the measurement of PCP in blood (Bevenue, *et al.* 1968; Casarett *et al.* 1969), provided the Hawaii project with the analytical skill and the interest to recruit into the present study a number of workers with occupational exposure to PCP.

Characteristics of the Study Sample

The total sample size consisted of 422 individuals. Only the last set of data collected on each individual was used for the present evaluation. Within the total sample, 42 individuals were controls with no occupational exposure, 333 were farmers or pest control operators (PCOs) with mixed exposure to various pesticides, and 47 were employees of firms that processed lumber and other wood products by treatment with PCP and other wood preservative chemicals (hereinafter referred to as the wood-treatment group).

The wood-treatment group of workers obtained exposure to PCP through two different processes for the preservative treatment of wood (Casarett *et al.* 1969). One process involved the open vat dipping of wood products in a kerosene solution containing approximately 5% PCP and sometimes antibloom agents other than kerosene. Workers undergoing the greatest exposure in this process were those engaged in the filling and periodic cleaning of the vat, the fork lift operators, and those who otherwise came in direct contact with either the chemical solution or the treated wood. Twenty-one of the workers in the wood-treatment group were employed in this type of treatment process which used PCP exclusively as the preservative chemical. The second wood treatment process involved pressure treatment of wood products with mixtures of different formulations which contained PCP salts or acids of chromium, fluorine, arsenic, copper, boron, tin, and dieldrin. The greatest worker exposure by this process occurred through inhalation of fumes upon the opening of the retort, from tank cleaning operations, from contact with spilled formulations, and/or direct contact with the treated wood. Twenty-six of the workers in the wood-treatment group were employed in this type of treatment process and thus obtained a mixed exposure to PCP and other preservative chemicals.

Table 1. Blood serum concentrations (ppm) of PCP in individuals in different exposure groups

	No. of individuals	Mean	Standard deviation	Minimum	Maximum
Open-vat wood treaters	22	3.78	3.998	0.15	17.4
Pressure-tank wood treaters	24	1.72	2.023	0.02	7.70
PCO/Farmer group	280	0.25	0.882	<0.01	8.40
Controls	32	0.32	1.259	0.02	7.20

Table 2. Urine concentrations (ppm) of PCP in individuals in different exposure groups

	No. of individuals	Mean	Standard deviation	Minimum	Maximum
Open-vat wood treaters	18	0.95	1.93	<0.01	7.80
Pressure-tank wood treaters	23	0.27	0.56	<0.01	2.40
PCO/Farmer group	210	0.01	0.05	<0.01	0.40
Controls	32	0.03	0.18	<0.01	1.00

More complete detail on the preservative chemicals and procedures used in each treatment are given in the final report on which this publication is based (Sato *et al.* 1978).

Results

The mean concentrations of PCP found in the blood serum and urine samples collected from individuals in each group of the entire study sample are given in Tables 1 and 2. The mean serum concentrations of PCP were considerably higher in the wood treatment group (3.78 ppm for the open vat treaters and 1.72 ppm for the pressure tank treaters) than in either the PCO/farmer group (0.25 ppm) or the control group (0.32 ppm). The mean urine concentrations were also highest in the wood-treatment group (0.27 ppm to 0.95 ppm).

The degree of statistical significance found between the mean PCP values shown in Tables 1 and 2 was determined by analysis of variance following logarithmic transformation to adjust for skewness in this data. Highly significant differences were found in PCP concentrations in serum and urine of the different exposure groups. Furthermore, the mean concentrations found in workers in the open vat treatment of wood were significantly higher than those found in workers in the pressure chamber treatment of wood. The latter finding is in agreement with similar results obtained earlier by Casarett *et al.* (1969).

To ensure complete uniformity in the comparative evaluation of all clinical laboratory data, the technique of listwise deletion (SPSS Manual 1975) was used to screen out participants whose data sets were incomplete by one or more of the variables noted in Table 3. This reduced the sample size to 189 males of which 17 were wood treaters (7 open vat treaters; 10 pressure tank treaters), 155 were pest control operators and farmers, and 17 were controls.

Despite this reduction of the sample, the final size was considered ample for further statistical computations. The advantage of the use of the listwise deletion approach resided in the assurance that it provided for compatibility

Table 3. Means and standard deviations of variables used in the study

Variable	Normal range[a]	Total Group (n=189) Mean	S.D.	PCP Group (n=17) Mean	S.D.
Red blood cell cholinesterase	7.5– 17.0 μM/min/ml	14.1	3.0	10.9	5.3
Blood plasma cholinesterase	1.2– 5.5 μM/min/ml	4.8	1.0	5.0	0.9
Red blood cell count	4.6– 6.2 × 10^6	5.0	0.4	5.2	0.3
Hemoglobin	13 – 18 gm %	15.8	1.3	16.2	1.3
Hematocrit	40 – 54 %	45.8	3.6	47.8	3.5
White blood cell count	4.8– 10.8 × 10^3	5,827.0	1,596.1	6,117.7	1,389.4
Bands (immature leucocytes)	0 – 5 %	0.2	0.7	0.6	1.5
Lymphocytes	24 – 45 %	34.0	8.7	33.1	9.4
Monocytes	2 – 6 %	3.8	2.5	3.1	2.4
Atypical lymphocytes	0 %	0.5	1.3	0.2	0.6
Eosinophils	0 – 3 %	3.0	3.3	3.9	3.1
Basophils	0 – 1 %	0.4	0.7	0.7	1.0
Polymorphonucleocytes	40 – 70 %	58.1	9.6	58.4	10.7
Uric acid	2.5– 7.0 mg %	6.5	1.4	6.8	1.5
Bilirubin	0.2– 1.0 mg %	0.7	0.4	0.7	0.3
Blood urea nitrogen	8 – 22 mg %	14.9	3.4	13.5	3.7
Cholesterol	150 –250 mg %	232.3	42.6	214.3	44.6
Creatinine	0.9– 1.4 mg %	1.0	0.2	1.0	0.2
Total protein	6 – 8 mg %	7.8	0.5	7.9	0.5
Glucose	70 –110 mg %	107.4	14.8	103.3	6.7
A1 Globulin	1 – 3 %	4.2	1.1	4.1	1.0
A2 Globulin	6 – 14 %	8.0	1.4	7.6	0.9
B Globulin	8 – 16 %	9.3	2.5	9.4	1.2
G Globulin	9 – 22 %	14.6	2.5	14.7	1.8
LDH[b]	200 –1500 UV Units (W-L)	409.3	81.2	425.1	90.7
CPK[b]	0 – 12 Sigma Units	5.5	9.9	7.4	6.4
SGOT[b]	12 –36UV Units (W-L)	29.4	11.4	32.5	11.6
SGPT[b]	6 – 53UV Units (W-L)	30.3	19.6	34.8	16.6
Alkaline phosphatase[b]	0.8– 2.3 BLB	1.4	0.5	1.7	0.7
LDH[c]	90 –200 mu/ml	142.3	29.5	143.3	21.5
SGOT[c]	10 – 50 mu/ml	40.0	24.9	41.0	16.6
Alkaline phosphatase[c]	16 –107 u	60.5	16.7	69.1	17.7
Calcium	5.5– 11 meq/L	9.7	0.4	9.6	0.4
Phosphorus	2.5– 4.8 meq/L	3.3	0.4	3.3	0.3
Blood PCP residue level	—	0.3 ppm	0.9	1.6 ppm	1.7
Urine PCP residue level	—	0.01 ppm	0.09	0.17 ppm	0.25
Age	—	43.7 yr	12.1	33.6 yr	9.7

[a] Normal range used by the Pathology Laboratory of Straub Clinic, Honolulu, HI, or in Smith (1969)
[b] Gilford Spectrophotometric Method (Smith, 1969)
[c] Auto-Analyzer Method, Straub Clinic, Honolulu, HI

among the various statistical techniques used in this study. It was assumed that the criterion variables shown in Table 3 were not independent and form mutually dependent subsets. Therefore, all tests were performed on the same population, *i.e.*, cases with a complete variable set.

Statistical Methods and Findings

Preliminary Statistics

The mean and standard deviation values for the clinical laboratory variables, age, and PCP residue levels are noted in Table 3. The values placed directly to

Table 4. Ethnic composition of the sample

	PCP group		Non-PCP group		
	n	%	n	%	N
Caucasian	4	23.5	23	13.4	27
Chinese	2	11.8	13	7.6	15
Filipino	5	29.4	3	1.7	8
Hawaiian extraction	2	11.8	9	5.2	11
Japanese	1	5.8	124	72.1	125
Others	3	17.6	0	—	3
	17		172		189
			$X^2 = 89.9$	$p < .001$	

Table 5. Pearson's correlation coefficients used as T-test comparisons between the PCP-exposed group and non-PCP exposed group with respect to age, blood PCP residue, urine PCP residue, and blood dieldrin and DDT + DDE residue levels

	Coefficient values	t-values	Significance
Age	−.263	3.728	.001
PCP: blood	.484	7.564	.001
PCP: urine	.422	6.365	.001
Dieldrin: blood	−.020	0.274	N.S.
DDT + DDE: blood	−.125	1.723	N.S.

the right of each variable in the table provide a comparative range adjudged to be clinically normal. Ethnic differences among the PCP-exposed group and non-exposed group are shown in Table 4. The ethnic distribution among the two groups are significantly different.

The mean values for age, PCP blood residue level, and PCP urine residue level were found to be significantly different between the PCP-exposed group and the non-PCP exposed group (Table 5). PCP residue levels were significantly higher in the exposed group, and the age of the exposed group was significantly lower than the non-PCP exposed group. Also, persons occupationally exposed to PCP did not appear to have significant exposures to DDT (+ DDE) or dieldrin, even though the pressure tank treaters were exposed to dieldrin from 1970.

Table 6 shows that blood dieldrin residue levels were not significantly associated with either blood or urine PCP residue levels. DDT + DDE blood residue levels, on the other hand, showed a significant association with blood PCP residue levels, but not urine PCP residue levels. An explanation for the apparent association between blood DDT + DDE and blood PCP residue levels is that agricultural and pest control workers used both types of pesticidal agents in their work. Referring to Table 5, it is noted that among the wood-treatment group, there is no apparent cross-contamination with dieldrin or DDT + DDE exposure.

Table 6. Simple Pearson's correlations among blood and urine concentrations of PCP and organochlorine pesticide blood residue concentrations in the study sample of 189 individuals

	PCP-blood	PCP-urine	Dieldrin	DDT + DDE
	Coefficient values			
PCP-blood	1.000			
PCP-urine	.817**	1.000		
Dieldrin	.011	−.016	1.000	
DDT + DDE	.122*	.004	.083	1.000
Significance *P ≤ .05 **P ≤ .01				

Table 7. Significant associations between the PCP-exposed group and clinical laboratory variables, after correlating for age and ethnicity (n = 189)

Variable	Beta	F (1,181)	Significance
Bands	0.239	8.253	$P < .005$
Plasma cholinesterase	0.189	4.898	$P < .050$
Alkaline phosphatase (Gilford)	0.179	6.046	$P < .025$
Alkaline phosphatase (Auto analyzer)	0.192	4.969	$P < .050$

Regression Analysis

Since it was found that age and ethnic differences existed between the PCP-exposed group and the non-PCP-exposed group (Tables 4 and 5), control of the effects of these variables were sought when comparing clinical laboratory variable values between the two groups.

Noting that ethnicity and PCP exposure are classificatory variables, the methodology of choice would be multivariate analysis of variance. The numbers, however, in the sub-cells were too small to allow sub-class analysis. Therefore, it was decided to use multiple regression analysis with a mixture of continuous (age and the clinical laboratory variables) and dichotomous pseudo-variables (PCP group membership and ethnicity).

The results of this analysis are shown in Table 7. PCP exposure appears to be highly associated with increased numbers of bands, increased blood plasma cholinesterase levels, and increased alkaline phosphatase levels, after controlling for the effects of age and ethnicity.

Canonical Correlation Analysis

By using the technique known as canonical correlation analysis, an attempt was made to account for inter-relationships among the clinical laboratory variables (criterion variables) and the predictor variables: PCP-exposure, blood residues of PCP, *p,p'*-DDT, *p,p'*-DDE and dieldrin, urine residues of PCP, and age and ethnicity.

The aim of canonical analysis (Hotelling 1936) is to derive a linear combi-

nation of variables in each set in such a way that the correlation between the linear combinations is a maximum. Many pairs of linear combinations (canonical variates) can be derived. The first pair of canonical variates is selected so as to have the highest inter-correlation possible. A secondary pair of canonical variates is then selected to account for a maximum amount of the relationship between the two sets of variables left unaccounted for. Since each successive stage of canonical variates account for residue variance, the variate pairs are uncorrelated with each other.

The results of this analysis are summarized in Table 8. At state 2 of the canonical correlation analysis, relationships between PCP-exposure variables and certain clinical laboratory variables were uncovered. PCP exposure appears by this technique to be associated with increased bands, gamma globulin, basophils, uric acid, plasma cholinesterase, and reduced serum calcium.

Analysis of Illness Conditions

All illness conditions determined at the time of medical examination and review were converted to code numbers as designated by a manual (ICDA 1968) on the International Classification of Diseases. The prevalence rates of these coded illness conditions, age standardized by the direct method (Barclay 1958), were determined among the wood treatment and control groups of the original sample of 422 individuals described under Characteristics of the Study Sample. The study sample in this analysis consisted of 47 individuals with occupational exposure to PCP (mean age 37.4 ± 12.7 years) and 42 control individuals (mean age 40.4 ± 10.6 years).

Age standardized prevalence rates for a number of the illness conditions found are given in Table 9. Because of the small sample size, related illness conditions were occasionally grouped together. Absent from this Table are other disease conditions which occurred at prevalence rates too low for meaningful analysis.

The age standardized prevalence rates for conjunctivitis, chronic sinusitis, and chronic upper respiratory conditions (ICDA code numbers 500 through 504) were significantly higher among PCP-exposed than among control individuals. Significance was determined if the standardized prevalence ration (Table 9) was greater than or equal to 2.0. Infection of the skin and subcutaneous tissue (ICDA code numbers 680 through 686), and gout, also occurred among the PCP-exposed at somewhat higher prevalence rates, but the significance of this increase was borderline. Palpitation and tachycardia occurred at significantly higher rates in control individuals. All other disease conditions listed, although often occurring at high prevalence rates, did not differ appreciably between PCP-exposed and control individuals.

Conjunctivitis was found among the PCP-exposed in only those individuals who were engaged in the pressure treatment of wood and who, therefore, had encountered a mixed exposure to PCP and other chemicals used in wood preservation.

In the early stages of the analysis of the illness conditions shown in Table 9, it was noted that the ICDA code number 401 had been used for unconfirmed and borderline cases of essential, benign hypertension. Re-coding was then done, based upon either an elevation of both systolic and diastolic blood pres-

Table 8. Summary table of the canonical structure matrix: r's with P ≤ .01

Predictor variables		Criterion variables	
		Stage 1	
*p p'*DDE	.850	Cholesterol	.590
*p p'*DDE	.376	A2 Globulin	.306
Japanese	.350	G Globulin	.283
Urine PCP	−.441	Glucose	.270
Blood PCP	−.409	B.U.N.	.229
Dieldrin	−.341	A1 Globulin	.229
Caucasian	−.237	RBC cholinesterase	.224
Hawaiian	−.232	Albumin	−.534
		RBC	−.490
		Calcium	−.344
		Hematocrit	−.240
		Monocytes	−.231
		Stage 2	
Urine PCP	.827	Bands	.598
Blood PCP	.777	G Globulin	.392
PCP occupation	.537	Basophils	.312
Hawaiian	.323	Uric acid	.255
Filipino	.261	Plasma cholinesterase	.232
Japanese	−.237	Calcium	−.253
		Stage 3	
Hawaiian	.531	B.U.N.	.457
Caucasian	.438	WBC	.412
Chinese	.322	Creatinine	.364
Urine PCP	.303	Bilirubin	.287
Blood PCP	.267	G Globulin	.233
pp'DDE	.231	Eosinophils	−.262
Japanese	−.753	Basophils	−.255
Dieldrin	−.238	Plasma cholinesterase	−.127
		Stage 4	
Japanese	.530	Bilirubin	.507
Caucasian	−.793	Creatinine	.388
Controls	−.556	RBC	.316
Urine PCP	−.371	Uric acid	.304
Blood PCP	−.319	B.U.N.	.259
		Hematocrit	.258
		Hemoglobin	.241
		Bands	−.340
		Glucose	−.283
		Plasma cholinesterase	−.281
		A1 Globulin	−.267

sure to above 140/90 or a history of elevated blood pressure and a current regime of antihypertensive medication. The results for hypertension (Table 9) indicate a small but insignificant increase in the prevalence rate among PCP-exposed individuals.

Table 9. Age standardized prevalence rates of specific illness conditions among 42 controls and 47 individuals occupationally exposed to PCP

Condition	ICDA number	Controls	PCP Exposed	SPR[a]
Thyroid disease	(240–246)	4.8	3.6	0.8
Gout	274	7.1	12.4	1.7
Neuroses	(298–309)	23.8	17.2	0.7
Conjunctivitis (non-bacterial)	360	2.4	7.6	3.2
Refractive error	370	42.9	40.0	0.9
Pterygium	372	21.4	24.7	1.2
Cataract, glaucoma, other eye disease	(374,375,378)	9.5	14.2	1.5
Hypertension, essential benign	401	11.9	15.5	1.3
Palpitation and tachycardia	(782.1,782.2)	7.1	2.4	0.3
Acute upper respiratory infections	(460–465)	33.3	38.3	1.2
Bronchial asthma	493	14.3	10.6	0.7
Chronic sinusitis	503	7.1	24.4	3.4
Chronic upper respiratory conditions	(500–504)	11.9	33.3	2.8
Hay fever-allergic rhinitis	507	42.9	40.3	0.9
Chronic upper respiratory conditions plus allergic rhinitis	(500–504,507)	50.0	55.0	1.1
Impaired pulmonary function tests	519	16.7	17.0	1.0
Chronic bronchitis and productive cough	692	33.3	37.6	1.1
Infections of skin and subcutaneous tissue	(680–686)	4.8	7.6	1.6

[a] Standardized prevalence ratio = (standardized rate for PCP exposed/standardized rate for controls)

Discussion

A detailed statistical evaluation of the clinical laboratory data revealed a strong association between PCP exposure and the increased occurrence of bands (immature leucocytes) and moderate associations between PCP exposure and increased plasma cholinesterase, alkaline phosphatase, gamma globulin, basophils, and uric acid, and decreased serum calcium. Despite these statistically significant associations, values considered to be clinically abnormal were few and were not significantly great in occurrence among PC individuals.

Bands were found to occur in only four individuals of the wood-treatment group. The actual count was two bands in each of three individuals and six bands in a fourth individual. The fourth individual had an apparent active skin inflammation, with the result that the presence of the bands was probably reflective of an infective or stress reaction. Since bands are commonly observed and many clinicians consider a 2 to 3% occurrence as normal, the observed occurrence in this study, while statistically significant, does not have any direct clinical relevance when these individuals are assessed separately.

Alkaline phosphatase and serum cholinesterase values were significantly higher among PCP-exposed individuals, although only a few of these values

were above their clinically normal range. The levels of other enzymes, including LDH, SGOT and SGPT, were not significantly increased. These findings fail to provide any biochemical indication of liver or other organ damage and there is little to suggest biliary tree obstruction resulting in increased alkaline phosphatase production. The only evidence of liver disease in the medical records of the PCP-exposed individuals was a single diagnosis of cholecystitis and two diagnoses of chemical jaundice based upon marginally elevated (1.1 mg%) bilirubin. Instances of abnormally elevated bilirubin were more numerous among control individuals, two of whom were also diagnosed as having chemical jaundice. The term chemical jaundice is used here to refer to an isolated laboratory observation of total bilirubin above the normal limit (0.2 to 1.0 mg%) that did not have supportive clinical or other laboratory findings that would provide a diagnosis of hepatic or biliary disease.

The significantly higher uric acid levels among PCP-exposed individuals probably was associated with the somewhat higher prevalence of gout in this group. Three of the five individuals with gout had abnormally higher uric acid levels. Four of the five individuals with gout were of Filipino extraction and, therefore, had a known predisposition to hyperuricemia (Healy and Bayani-Sioson 1971). Thus, the higher prevalence rate of this disease among the PCP-exposed is thought likely to be the result of the relatively higher proportion in this group of Filipinos.

Conjunctivitis, as found among wood treatment workers with mixed exposure to PCP and other wood preservative chemicals, was a low-grade irritation or inflammation of the outer protective membrane which was either allergic or catarrhal, probably caused by dust or chemical exposure. Since this condition did not occur among the wood treaters exposed only to PCP, it is possible that PCP had no causative activity.

The other disease condition found to be more prevalent among PCP-exposed than among control individuals was chronic sinusitis, alone or in combination with other chronic upper respiratory conditions. These patients presented low grade infections and did not require medical intervention; symptoms were distinct from acute respiratory infections. Clinically and symptomatically, these symptoms overlapped to some degree those of hay fever and allergic rhinitis. The prevalence rates of hay fever and allergic rhinitis were higher among the PCP-exposed group but not to any significant degree.

Contact dermatitis also occurred at a relatively high prevalence rate among both PCP-exposed and control individuals. Among the PCP-exposed, cases of dermatitis appeared rather non-specific although several individuals indicated the occurrence of sporadic "rashes" when in contact with PCP or other chemicals. Infections of the skin and subcutaneous tissue were chiefly characterized by boils and were somewhat higher in prevalence among the PCP-exposed group.

The information gathered in this study suggested that despite high chronic exposures to PCP, individuals in the wood treatment group of workers had not undergone any serious health effects from this exposure. The only evidence of tangible health effects, part of which could have been caused by exposures to chemicals other than PCP, were the low-grade infections or inflammations of the skin and subcutaneous tissue, of the protective membrane of the eye, and of the mucous membrane of the upper respiratory tract. No specific long-term effects could be elicited in the exposed group.

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