American Journal of Industrial Medicine 17:411–421 (1990)

Chloracne Associated With Employment in the Production of Pentachlorophenol

Michael A. O'Malley, MD, MPH, Arvind V. Carpenter, DDS, DrPH, Marie Haring Sweeney, MPH, Marilyn A. Fingerhut, PhD, David A. Marlow, BS, William E. Halperin, MD, MPH, and C.G. Mathias, MD

To evaluate the association between exposure to pentachlorophenol (PCP) and the occurrence of chloracne, we studied the medical and personnel records for individuals employed in the manufacturing of PCP. Forty-seven cases of chloracne were identified among 648 workers (7.0%) assigned to PCP production at a single plant between 1953 and 1978. The annual incidence rate varied considerably, ranging from 0 (in 1953) to 1.46 (in 1978). No linear trend in the risk of chloracne was observed with the duration of employment in the pentachlorophenol department. Workers with a documented episode of direct skin contact with PCP had a significantly increased risk of chloracne compared with workers who did not have a documented episode of direct skin contact (cumulative incidence ratio = 4.6; 95% confidence interval 2.6–8.1). Our results confirm that chloracne is associated with exposure to PCP contaminated with hexachlorinated, heptachlorinated, and octachlorinated dibenzo-*p*-dioxins and dibenzofurans.

Key words: dioxin, occupational exposure, wood preservatives, epidemiology, hexachlorodibenzo-*p*-dioxin

INTRODUCTION

Pentachlorophenol (PCP) is a widely used wood preservative that has had a variety of applications in wood, leather, and paper industries. PCP has also had limited use in homes and gardens as fungicide, algicide, insecticide, and disinfectant. An estimated 17,000 workers are currently exposed to PCP in the United States [Sieber, 1989]. In the United States, PCP is produced by direct chlorination of molten phenol, using progressively elevated temperatures and a catalyst to drive the reaction [Cirelli, 1978; WHO, 1987]. During the process, a small proportion of various chlorophenols react with each other to yield polychlorinated dibenzo-*p*-dioxins and dibenzofurans. The major types of dioxins in PCP are hexachlorinated, heptachlori-

Industrywide Studies Branch, Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, Cincinnati.
Address reprint requests to Ms. Sweeney, Industrywide Studies Branch, Division of Surveillance, Hazard Evaluations, and Field Studies, NIOSH, Mail Stop R-15, Cincinnati, OH 45226.
Dr. O'Malley is now at the California Department of Food and Agriculture, Sacramento, CA 95814.
Dr. Halperin is now at Harvard University School of Public Health, Boston, MA 02159.
Dr. Mathias is now at Dermatology Department, Group Health Associates, Cincinnati, OH 45215.
Accepted for publication November 20, 1989.

Published 1990 by Wiley-Liss, Inc.

nated, and octachlorinated isomers. The major types of furans in PCP are tetrachlorinated, pentachlorinated, hexachlorinated, heptachlorinated, and octachlorinated isomers [Plimmer, 1973; Goldstein et al., 1977; WHO, 1987]. PCP does not contain 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.

Exposure to pentachlorophenol contaminated with dioxins and furans has been associated with chloracne in rabbits and in case reports of human exposure [Baader and Bauer, 1951; Johnson et al., 1973; Markel and Lucas, 1975; Sehgal and Ghorpade, 1983; Lambert et al., 1986; Cole et al., 1986]. When applied to rabbit ear, commercial PCP produced a positive response, whereas pure PCP gave a negative response in these bioassays [Johnson et al., 1973]. Chloracne is a persistent acneiform condition characterized by comedones, keratin cysts, and sometimes pustules. Chloracne occurs mainly on the face; particularly around and under the eyes and behind the ears. In severe cases, the lesions may cover the rest of the face, neck, and shoulders, genitalia, chest, and lower trunk. Chloracne occurs subsequent to acute or chronic exposure to a variety of chlorinated aromatic compounds [Crow, 1978; Moses and Prioleau, 1985]. Published reports associate the occurrence of chloracne with an exposure to 2,4,5-trichlorophenol (2,4,5-TCP), 2,4,5-trichlorophenoxy-acetic (2,4,5-T) acid, or their derivatives [Kimmig and Schulz, 1957; May, 1973; Reggiani, 1980; Suskind, 1985], which usually contain the contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). No large epidemiologic studies on workers who produced PCP have been published. One study that described skin absorption of PCP during the formulation and subsequent use of wood preservatives containing PCP found no overt case of chloracne among 209 exposed and 101 unexposed workers [Jones et al., 1986].

The purposes of this study were to evaluate the occurrence of chloracne among PCP workers and to assess the risk of chloracne among workers who had records of direct skin contact with PCP.

METHODS AND MATERIALS

Plant Selection and Production Processes

A chemical plant located in southwestern Illinois was chosen for study because it had a long history of PCP production (1938–1978), good personnel records, and an extensive on-site medical program. The medical program provided medical surveillance examinations and primary care for occupational injuries and illnesses. Additionally, this plant never produced 2,4,5-TCP or 2,4,5-T acid. A study of other health effects in this population is currently being conducted by researchers at Northwestern University.

Beginning in 1938, pentachlorophenol was produced by direct chlorination of phenol, monochlorophenol, dichlorophenol, and/or 2,4,6- trichlorophenol in the presence of an aluminum catalyst. With the exception of some modifications in the packaging and finishing operations, PCP was produced using the same chemical process throughout the entire production period (1938–1978). It is not clear from the available data whether significant changes in industrial hygiene practices occurred or whether they would have affected the potential for development of chloracne. In addition, during 1938 to 1975, sodium pentachlorophenate (NaPCP) was also produced at the plant by reacting PCP with sodium hydroxide solution.

Other chemicals known to cause chloracne were also produced by the company

TABLE I. Categorization Based on Medical Records Review

Category 1	Diagnosis of chloracne recorded in plant records with supporting clinical information.
Category 2	Diagnosis of chloracne recorded without supporting clinical information.
Category 3	New onset of facial comedones with no clinical diagnosis of chloracne recorded. Inclusion in this category required the presence of normal skin in a previous examination.
Category 4	New onset of undescribed facial rash. Inclusion in this category required the presence of normal skin in a previous examination.
Category 5	New onset of comedones in an undescribed distribution, in combination with presence of normal skin in a previous examination.
Category 6	No evidence of chloracne.

in buildings or areas separate from PCP production. From 1929 to 1977, polychlorinated biphenyls (PCBs) were produced; and during 1960–1971, 2,4,5-trichlorophenoxyacetic acid (brought in from a facility in another state) was esterified.

Study Population Selection

Exposure to PCP was defined as ever having been employed in the PCP department as an hourly production worker between 1938 and 1978. From company personnel records, 926 workers were identified who met this criterion and thus became eligible for inclusion in the study.

Salaried workers were excluded from the study because employment records did not always reflect the department or the process in which the salaried individuals worked. Maintenance workers were excluded from the analysis for similar reasons, particularly because they may have been assigned to other processes, e.g., 2,4,5-T or PCB production, where chloracnegens other than pentachlorophenol were present.

Case Ascertainment

To determine the occurrence of chloracne among PCP production workers, company medical records, including preemployment and periodic medical surveillance examinations and reports of acute illnesses, and available worker compensation records were reviewed for information regarding chloracne and all other forms of skin illnesses or injuries. The following information was abstracted from each record of a skin examination: date of examination, type of record and health care provider, examination results, type and distribution of skin lesions, diagnosis, and treatment. On the basis of the information in the medical record, each individual was placed into one of six categories (Table I). For the purposes of this study, individuals in categories 1, 2, and 3 were considered cases of chloracne and workers in categories 4, 5, and 6 were considered noncases. Work histories were used to identify chloracne cases attributable to PCP exposure. An individual was excluded from the analysis if: chloracne was diagnosed while he was employed either in the 2,4,5-T ester or the PCB department; if the individual had a history of employment in either 2,4,5-T ester or PCB department within 2 years prior to the diagnosis (although diagnosed as a case of chloracne while working in PCP department); or if that person was diagnosed more than 2 years after leaving employment in the PCP department. This 2-year

period was intended to capture late reporting of cases that may have occurred shortly after leaving employment in the PCP department.

Direct Skin Contact

Plant medical and available worker compensation records were also reviewed for reports of direct skin contact with molten PCP or its sodium salt, which was a liquid, during employment in the PCP department. Records abstracted included medical reports of burns or acute cutaneous irritation due to PCP or NaPCP. The records of skin contacts to PCP provided independent evidence of direct exposure to PCP. For chloracne cases, episodes of direct skin contact were excluded from the analysis if they occurred after the diagnosis of chloracne.

Analysis

The cumulative incidence of chloracne, the overall incidence rate, and the annual incidence rate were calculated for workers with medical records. About one third of all PCP production workers had no medical records. Because we could not identify chloracne cases among workers without medical records, we excluded these workers from the analyses. Furthermore, a large proportion (97%) of workers employed prior to 1953 had no medical records (176 of 181), whereas only about 10% of workers who were employed in 1953 or later had no medical records (79 of 745). Hence, to calculate the cumulative incidence, we excluded from the analysis workers with medical records who terminated their employment prior to 1953. Similarly, we began calculating person-years at risk (PYAR) in 1953 even for workers who began employment prior to 1953. Thus, cumulative incidence was calculated by dividing the number of chloracne cases by the total number of workers with medical records who were employed in 1953 or later. The overall incidence rate was calculated by dividing the number of cases by the total number of person-years employed in PCP during 1953–1978. Because each person was considered to be at risk for chloracne for 2 years after leaving the PCP department, PYAR for each individual included total years of employment in the PCP department plus 2 years. If the worker left the company at the time he/she left the PCP department, then PYAR included only the years in the PCP department. Annual incidence rate of chloracne was calculated by dividing the number of new chloracne cases per year by the number of PYAR in that particular year, including PYAR contributed by individuals who had left the PCP department within the previous 2 years. For cases, the duration of exposure was defined as the number of calendar days worked in the PCP department prior to the onset of chloracne, whereas for noncases, the total duration of employment in the PCP department was used. To evaluate the effect of duration of exposure, incidence rates of chloracne were calculated for five exposure categories by dividing the observed number of cases in a category by PYAR in that category. Even though these exposure categories were chosen arbitrarily, the rationale reflects the knowledge that chloracne occurs within a short time after exposure. For each category, PYAR were calculated using a modified life-table analysis system developed at National Institute for Occupational Safety and Health [Waxweiler et al., 1983]. The standardized incidence ratios (incidence rate among the exposed group divided by the incidence rate among the referent group) were calculated for each exposure category. The "0.5 year or less" group constituted the referent. A test for trend was conducted [Hakulinen, 1981].

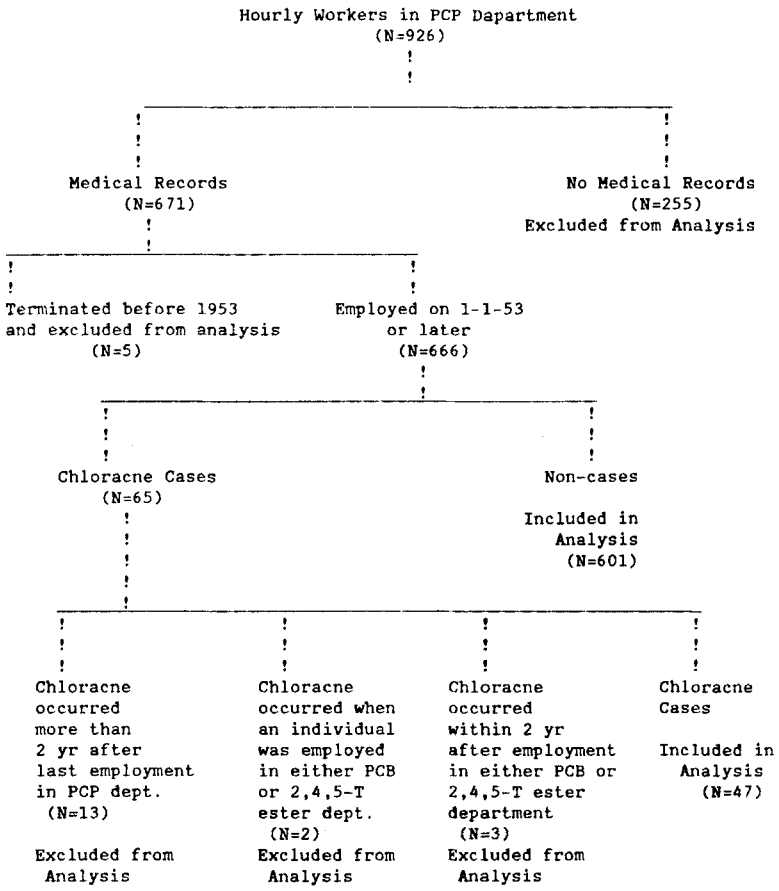

Fig. 1. Description of the study population.

A cumulative incidence ratio (cumulative incidence of chloracne among workers with documented reports of direct skin contact divided by the cumulative incidence among workers without such documented reports) was calculated to evaluate the association between chloracne and a history of documented direct skin contact with PCP.

RESULTS

Of the 926 hourly workers in the study cohort, 671 (72.5%) had a medical or worker compensation record available. The remaining 255 workers were not included in the analysis (Fig. 1). Five of the 671 workers with medical records terminated employment prior to 1953 and were also excluded from all analyses. The mean year of birth was 1919 for workers without medical records compared with 1923 for workers with medical records. The mean year of hire was 1946 and 1952, and the mean year of termination was 1951 and 1978 for workers without and with medical records, respectively. Workers without medical records were employed in PCP, on average, for 0.6 year compared with 1.4 years for the workers with medical records.

TABLE II. Distribution of Chloracne Cases by Year of Diagnosis

Year of diagnosis	Number	Percent
Total	47	100.0
1953–1955	0	0.0
1955–1959	2	4.2
1960–1964	13	27.7
1965–1969	4	8.5
1970–1974	7	14.9
1975–1978	21	44.7

Of the 666 workers with medical records who were employed in 1953 or later, 65 had a diagnosis of chloracne in their medical records. Eighteen of these 65 workers were excluded from analyses because chloracne was diagnosed more than 2 years after leaving the PCP department or the workers were potentially exposed to PCBs or 2,4,5-T esters (Fig. 1). Thus, 47 cases were identified that were thought to be associated with PCP exposure. Of these 47 chloracne cases, 14 had a diagnosis of chloracne accompanied by supporting clinical description (category 1), 32 cases had a recorded diagnosis, but no supporting clinical description was available (category 2), and one worker had new onset of comedones without a definite diagnosis of chloracne (category 3). No case of chloracne was diagnosed before 1955, whereas almost half of the cases were diagnosed between 1975 and 1978 (Table II).

There were 601 individuals who had worked in the PCP department during 1953–1978 but did not have chloracne. The mean duration of employment at the plant was longer for noncases (21 years) than for cases (17 years); however, the mean duration of exposure (i.e., employment in PCP department) was longer for cases (3.2 years) than for noncases (1.2 years).

The overall cumulative incidence of chloracne was 7.2% (47/648), and the overall incidence rate for chloracne was 2.1% (47/2,252 PYAR). The annual incidence rate varied considerably and ranged from 0 (in 1953) to 1.459 (in 1978). The annual number and the annual incidence rate of chloracne show an increase in the early 1960s and 1970s (Fig. 2).

Duration of Exposure

Using the life-table analysis system, we calculated PYAR for each of the five duration of exposure categories. Standardized incidence ratios for 0.51 to 1.0 year, 1.01 to 5.0 years, 5.01 to 10.0 years, and more than 10 years were 4.37, 1.99, 4.32, and 1.42, respectively, when compared with 0.5 year or less (Table III). It did not appear that increased duration of exposure was associated with increased risk of chloracne (test for trend $p > 0.2$).

Direct Skin Contact with PCP

Episodes of direct skin contact with PCP were reported throughout the history of the plant, with periodic increases in the number and the incidence rate of skin contacts. Records indicate that while assigned to the PCP department, 50 of the 648 workers in this study experienced one or more episodes of direct skin contact with PCP or NaPCP (a total of 89 episodes). Of these 50 workers, 13 (26.0%) workers

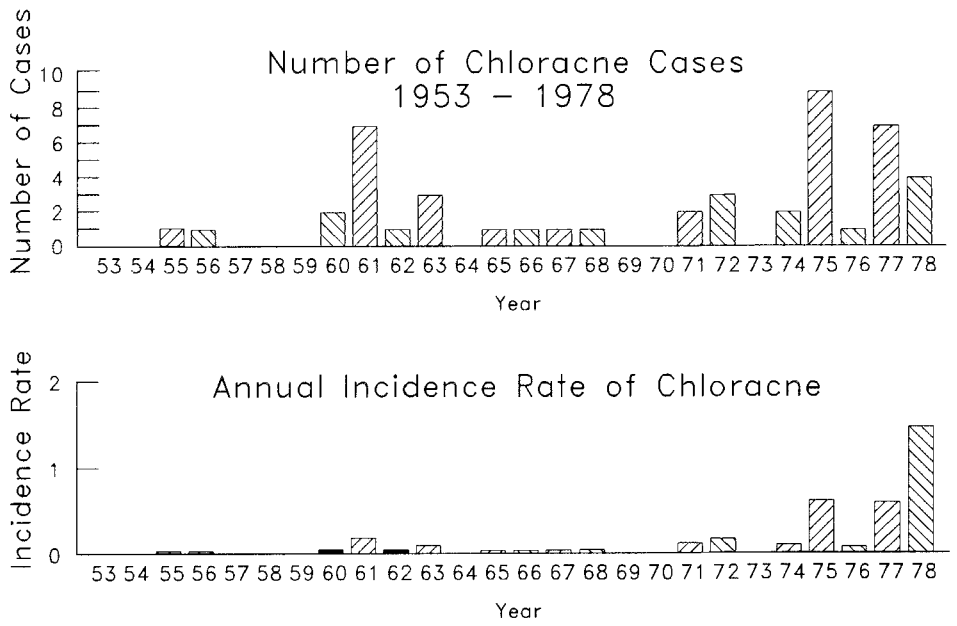

Fig. 2. Chloracne cases and annual incidence rate by year.

TABLE III. Standardized Incidence Ratio for Chloracne by Duration of Employment in Pentachlorophenol Department Using Person-Years at Risk

Duration of employment in PCP (years)	Total no. of person-years at risk	Number of chloracne cases	Standardized incidence ratio[a] (95% CI)[b]
0.50 or less	1040	14	1.00
0.51–1.0	289	9	4.37 (1.51–12.65)
1.01–5.0	645	12	1.99 (0.86–4.60)
5.01–10.0	193	8	4.32 (1.51–12.32)
> 10.0	85	4	1.42 (0.41–4.90)

[a]Age, calendar-year, sex, and race standardized incidence ratio using 0.5 year or less as the referent category.
[b]95% confidence intervals.Chi-square (1 df) for linear trend = 1.38; $p > 0.2$.

developed chloracne subsequent to the skin contact (Table IV). Thus, workers with independent records of direct skin exposure had overall a fourfold increase in the risk of developing chloracne compared with workers who did not have records of direct skin contact (CIR = 4.6; 95% CI = 2.6–8.1).

Eight of the 13 cases had only one episode of direct skin contact with PCP prior to the diagnosis of chloracne, three cases had two episodes; and two cases had three

TABLE IV. Relationship Between Direct Skin Contact to PCP and Occurrence of Chloracne

Direct skin contact with PCP	Chloracne cases	Total workers	Cumulative incidence	Cumulative incidence ratio (95% CI)[a]
No	34	598	0.057	1.0
Yes	13	50	0.260	4.6 (2.1–8.1)

[a]95% confidence intervals.

episodes; thus, on average, a case had 1.5 episodes of direct skin contact. The interval between the latest episode of direct skin contact and the diagnosis of chloracne for these 13 cases ranged from about 7 weeks to about 14 years. Four of the 13 cases occurred within 6 months of skin contact, four occurred between 1 and 2 years after the skin contact, two occurred between 2 and 3 years after contact, and three occurred more than 10 years postcontact.

We repeated our analysis using the eight workers who developed chloracne within 2 years of direct skin contact. The cumulative incidence ratio was 2.5, with a 95% confidence interval of 1.2 to 5.0.

DISCUSSION

In our study, we found 47 cases of chloracne occurring over a 25-year period among workers who had exposure only to PCP in the 2 years prior to the diagnosis, a cumulative incidence of 7.2%. In addition, a positive association was found between the incidence of chloracne and the reported episodes of direct skin contact with PCP.

Previously, the occurrence of chloracne among individuals exposed to PCP had been observed in case reports and in a cross-sectional study [Baader and Bauer, 1951; Markel and Lucas, 1975; Sehgal and Ghorpade, 1983; Baxter, 1984; Cole et al., 1986; Lambert et al., 1986]. The background rate of acneiform dermatitis resembling chloracne is difficult to determine because the clinical definition requires knowledge of exposure. Chloracne has not been shown to occur in nonexposed people [Suskind and Hertzberg, 1984]. Chlorinated dioxins and dibenzofurans have been found to be associated with the development of chloracne [Johnson et al., 1978; Crow, 1978; Kunita et al., 1985]. Available laboratory analyses demonstrated that PCP produced at the plant contained the hexa, hepta, and octachlorinated congeners of dibenzo-*p*-dioxins and dibenzofurans (Table V). Thus, our finding that exposure to PCP and its contaminants is associated with the occurrence of chloracne is consistent with previous case reports and animal evidence.

There are many problems in conducting an epidemiologic study with medical records as the primary data source. The limitations include: 1) missing or inconsistent diagnoses; 2) changes in clinical definition of the disease over the study period; 3) documentation of onset of disease versus date diagnosis; or 4) missing records.

To minimize biases that might be introduced by inconsistent diagnoses or changes in the clinical definition of chloracne on the part of the plant physicians, we

TABLE V. Levels of Hexa-, Hepta-, and Octa-Chlorinated Dibenzo-*p*-Dioxins and Dibenzofurans in Samples of PCP Produced Between 1970–1976

	Dibenzo-*p*-dioxins N = 24		Dibenzofurans N = 8	
Chlorinated isomer	Mean (ppm)[a]	Range (ppm)	Mean (ppm)	Range (ppm)
Hexa-	29	1–260	324	190–470
Hepta-	217	23–540	336	80–670
Octa-	721	15–1880	220	130–430

[a]PPM = parts per million.

defined the diagnostic criteria for chloracne before reviewing the medical records, and we developed a method of coding the diagnoses in a standardized fashion. These criteria were relatively simple to apply and did not require extensive clinical documentation, which was absent from many records. In this study, one physician and three nurses were involved in coding the medical records, and these persons reviewed each other's work so that interobserver variability was minimal. In addition, only four physicians and nurses were employed at the plant between 1953–1978; therefore, variability in reporting chloracne diagnosis is most likely very low.

For the purposes of this study, we also used the first mention or notation of chloracne or skin rash in the medical record as the date of diagnosis of the condition. However, we acknowledge that the date of diagnosis may not always reflect the actual date of onset. Given the nonfatal nature of chloracne, individuals may not seek medical attention immediately after it becomes apparent. Although the substitution of date of diagnosis for date of onset is a limitation of the analysis, date of diagnosis as used in this study is the only objective indicator of the general period of onset of chloracne for all employees, and thus is used as such.

About 28% of the workers employed in the PCP department had no medical records. Because we could not identify chloracne cases among workers without medical records, we excluded these workers from the denominator in calculating the cumulative incidence and the incidence rate. Because medical records were not available prior to 1953, we included only workers who were employed in 1953 or later and began calculating PYAR in 1953, even for workers employed in PCP earlier than that time. Thus, we believe that the risk estimates we calculated provide a reasonable incidence rate during 1953–1978.

Evaluation of the relationship between episodes of direct skin contact and chloracne has several limitations. It may be that episodes of direct skin contact to PCP were underreported, because some workers were not seen at the company clinic. The same possibility applies to chloracne. Although there is no way to document the likelihood that individuals would report to the clinic following skin contact, we can assume that workers with relatively extensive contact and severe irritation would be likely to seek care. Given the temporal relationship between the diagnosis of chloracne and the latest documented skin contact (less than 6 months for four cases and less than 2 years for eight cases), we would infer that the skin contact increases the risk of chloracne. The current analysis did not include other information on the level of exposure to PCP. We are currently constructing a job-exposure matrix that will provide more quantitative exposure measurements.

In summary, this study confirms the hypothesis generated from case reports and animal data that exposure to PCP contaminated with hexachlorinated, heptachlorinated, and octachlorinated dibenzo-*p*-dioxins and dibenzofurans is associated with the occurrence of chloracne.

ACKNOWLEDGMENTS

The authors thank Drs. Elizabeth Ward and Kyle Steenland for their comments on the epidemiologic aspects of the paper; Mr. James Morris for his help with the statistical analysis; Ms. Susan Nowlin, Ms. Patti Dill, and Ms. Kathleen Hicks for computer programming support; and Ms. Frances Guerra, Ms. Mary Chung, Ms. Kathy Masterson, and Ms. Mary Torok for typing this paper. This study was partially funded by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia.

REFERENCES

Baader HC, Bauer MHJ (1951): Industrial intoxication due to pentachlorophenol. Ind Med Surgery 20:286–290.

Baxter RA (1984): Biochemical study of pentachlorophenol workers. Ann Occup Hyg 28:429–438.

Cirelli DP (1978): Patterns of pentachlorophenol usage in the United States of America—an overview. In Rao KR (ed): "Pentachlorophenol: Chemistry, Pharmacology, and Environmental Toxicology." New York: Plenum Press, pp 3–18.

Cole GW, Stone O, Gates D, Culver D (1986): Chloracne from pentachlorophenol preserved wood. Contact Dermatitis 15:164–168.

Crow K (1978): Chloracne: The chemical disease. New Scientist 78:78–80.

Goldstein JA, Friesen M, Linder RD, Hickman P, Hass JR, Bergman H (1977): Effects of pentachlorophenol on hepatic drug-metabolizing enzymes and porphyria related to contamination with chlorinated dibenzo-p-dioxins and dibenzofurans. Biochem Pharmacol 26:1549–1557.

Hakulinen T (1981): A Mantel-Haenszel statistic for testing the association between a polychotomous exposure and a rare outcome. Am J Epidemiol 113:192–197.

Johnson RL, Gehring PJ, Kociba RJ, Schwetz BA (1973): Chlorinated dibenzodioxins and pentachlorophenol. Environ Health Perspect 5:171–176.

Jones RD; Winter DP, Cooper AJ (1986): Absorption study of pentachlorophenol in persons working with wood preservatives. Hum Toxicol 5:189–194.

Kimmig J, Schulz KH (1957): Occupational chloracne caused by aromatic cyclic ethers. Dermatologica 115:540–546.

Kunita N, Hori S, Obana H, Otake T, Nishimura H, Kashimoto T, Ikegami N (1985): Biological effects of PCBs, PCQs, and PCDFs present in the oil causing Yusho and Yu-Cheng. Environ Health Perspect 59:79–84.

Lambert J, Schepens P, Janssens J, Dockx P (1986): Skin lesions as a sign of subacute pentachlorophenol intoxication. Acta Derm Venerol (Stockh) 66:170–172.

Markel HL, Lucas JB (1975): Weyerhauser Treating Plant, DeQueen, AK. Health Hazard Evaluation Determination Report No. 74-117-251, U.S. DHEW, CDC, NIOSH, Cincinnati, OH.

May G (1973): Chloracne from accidental production of tetrachlorodibenzodioxin. Br J Ind Med 30: 276–283.

Moses M, Prioleau PG (1985): Cutaneous histologic findings in chemical workers with and without chloracne with past exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. J Am Acad Dermatol 12: 497–506.

Plimmer J (1973): Technical pentachlorophenol: Origin and analysis of base insoluble contaminants. Environ Health Perspect 5:41–48.

Reggiani G (1980): Acute human exposure to TCDD in Seveso, Italy. J Toxicol Environ Health 6:27–43.

Sehgal VN, Ghorpade A (1983): Fume inhalation chloracne. Dermatologica 167:33–36.

Sieber K (1989): National Occupational Exposure Survey. Vol. II: Sampling Methodology. NIOSH (in preparation).

Suskind RR (1985): Chloracne, "the hallmark of dioxin intoxication." Scand J Work Environ Health 11:165–171.

Suskind RR, Hertzberg VS (1984): Human health effects of 2,4,5-T and its toxic contaminants. JAMA 251:2372–2380.

Waxweiler RJ, Beaumont JJ, Henry JA, Brown DP, Robinson CF, Ness GO, Wagoner JK, Lemen RA (1983): A modified life-table analysis system for cohort studies. J Occup Med 25:115–124.

WHO (1987): Environmental Health Criteria 71: "Pentachlorophenol." Geneva: World Health Organization.