MORTALITY AND INDUSTRIAL HYGIENE STUDY OF WORKERS EXPOSED TO POLYCHLORINATED BIPHENYLS

David P. Brown

Mark Jones

U.S. Department of Health, Education and Welfare

Public Health Service

Center for Disease Control

National Institute for Occupational Safety and Health

Division of Surveillance, Hazard Evaluations and Field Studies

Cincinnati, Ohio 45226

ACKNOWLEDGEMENTS

The authors would like to express their appreciation for the work of many individuals who helped to successfully complete this study including the -- guidance of Joseph Wagoner, Richard Lemen and Richard Waxweiler; the assistance of the clerical and secretarial staff in the Biometry Section; the data entry and data analysis provided by the Southwest Ohio Regional Computer Center; the cooperation of the companies and labor unions chosen for the study; and for the information provided by the New York State Department of Health.

ABSTRACT

Due to the demonstrated toxic effects from polychlorinated biphenyls (PCB's) on exposed animals, the National Institute for Occupational Safety and Health (NIOSH) conducted a retrospective cohort mortality study of two worker populations who manufactured electrical capacitors. The two study cohorts included 968 workers from Plant 1 and 1599 from Plant 2 who were employed for at least three months in areas of the plants where PCB's were used.

The vital status of over 97 percent of the two cohorts was determined as of January 1, 1976 and 38,890 person-years were accumulated. All-cause mortality was lower than expected (163 obs. vs. 174 exp.) as well as all cancer mortality (39 obs. vs. 40.6 exp.). Rectal cancer (4 obs. vs. 1.07 exp.) and liver cancer (3 obs. vs. 0.88 exp.) excesses were noteworthy although not statistically significant. In one of the plants, the observed mortality due to cirrhosis of the liver was also elevated. The results are discussed with regard to the detailed industrial hygiene surveys conducted in each plant.

INTRODUCTION

Polychlorinated biphenyls (PCB's) are a class of compounds composed of biphenyl molecules with a varying number of substituted chlorine atoms. In commercially prepared PCB's, the weight-percent of chlorine has varied between 21 and 68 percent. In some preparations, there has also been some degree of contamination by chlorodibenzofurans. (1)

The primary use of PCB's has been as a liquid insulating material in electrical capacitors and transformers, and the greatest potential for occupational exposure has been in the manufacturing and repair of these components. PCB's have also been used in heat exchange units, hydraulic systems, vacuum pumps, gas transmission turbines, plasticizers, adhesives, pesticide extenders, paints, and carbonless copying papers.

As of 1971, PCB's were sold only for use in closed systems. According to the Toxic Substances Control Act of 1976, specific rules and regulations were promulgated to limit the manufacture and use of PCB's. This Act stipulated that all U.S. production of PCB's end January 1, 1979 and that all U.S. sale and distribution of PCB's end July 1, 1979. However, continual exposure to PCB's will occur among workers who maintain transformers and capacitors, and among the general population mainly through contaminated food.

During the past few years, there has been a great deal of interest in studying the health effects among individuals exposed to PCB's. This interest has been stimulated by: (a) the tendency for PCB's to accumulate in tissues and in certain organs, (2, 3) (b) the stability of PCB's and their persistence in the environment, (4, 5) and (c) the demonstrated long term effects in exposed laboratory animals (6-13) - including liver tumors and other liver diseases. Much of this interest was expressed at the National Conference on Polychlorinated Biphenyls in November, 1975 (14) and the toxicity of PCB's has been extensively reviewed in the NIOSH Criteria Document on PCB's. (15)

In order to determine whether or not past occupational exposure to commercially produced PCB's has caused any long term health effects, NIOSH initiated an epidemiologic study among workers in two capacitor manufacturing plants. In conjunction with this study, detailed industrial hygiene surveys were also conducted by NIOSH.

DESCRIPTION OF FACILITIES

The two facilities chosen for the study were selected after preliminary walk-through surveys were conducted at numerous types of plants where PCB's were used. Both of the plants manufacture electrical capacitors. These plants were selected because of their large workforce, the early dates (1938 & 1946) at which PCB's were

first used at these plants, the potential for exposure to PCB's with little potential for exposure to other known toxic contaminants, and the availability of records necessary to identify individuals to be included in the study population. At the time the study was initiated both plants were still using PCB's. Plant 1 is located in New York State and is divided into two manufacturing facilities within close proximity. One facility produced small industrial capacitors using PCB's since 1946 and the other produced large PCB filled power capacitors since 1951. The type of PCB's used has varied over the years from "Aroclor" (Aroclor is a Monsanto tradename) 1254 (54 percent chlorine) to 1242 (42 percent chlorine) to 1016 (41 percent chlorine). Several other kinds of oils were used, but in a small number of capacitors. These oils included castor oil, dibutyl sebaceate, diotylthalate and mineral oil.

Plant 2 is located in Masachusetts where the use of PCB's to manufacture capacitors started in 1938. This plant also changed the type of PCB's used from "Aroclor" 1254 to 1242 to 1016. Until 1972, other types of capacitors which did not contain PCB's were made at this plant including mica, electrolytic and tubular. Castor oil was used in lieu of PCB's to produce the large power capacitors at this plant.

Both plants assemble the capacitors using the same general techniques, whether they are the small or large types. The following is a brief description of the assembly process:

A. Winding and Pre-assembly - The inner components of the capacitor are made of paper, foil and sometimes plastic film, wound together, which are subsequently loaded into metal casings. This job is done in an enclosed dust-free room where there is minimal exposure to PCB's.

B. Impregnation - The pre-assembled capacitors are filled or impregnated with the PCB's. Within this area there is potential for exposure to PCB's.

C. Final Assembly - The tops of the capacitors are closed using various techniques - crimpping (rubber stoppers) or soldering, which involves some exposure to PCB's. The capacitors are washed to remove excess PCB's by running them through a detergent wash or a degreaser such as trichloroethylene. Finally, they are sent through the final operations involving drying, testing, and painting.

Other areas of importance where there is potential exposure to PCB's in the plants, include the laboratory and the area where rejected

capacitors are rebuilt. Approximately 10 percent of the two workforces have been employed in jobs where there has been potential exposure to PCB's.

Historically, the workforce at Plant 1 has been approximately 50 percent white males and 50 percent white females. Plant 2 has had a less homogeneous workforce with two-thirds being female and reflects the general ethnic make-up of the area, which is largely Cape Verdean and Portuguese.

METHODS

A retrospective cohort study of mortality was conducted to determine whether or not individuals occupationally exposed to PCB's have experienced any increase in cause-specific mortality. The study cohorts were defined as all workers who accumulated at least three months of employment any time between January 1, 1946 and January 1, 1976 for Plant 1 and January 1, 1940 and January 1, 1976 for Plant 2, in areas of the plants where there was a potential for exposure to PCB's. These exposed jobs were designated by the companies and verified by the labor unions, and through the industrial hygiene surveys.

An effort was made to determine the vital status (alive or deceased) of each individual in the cohorts as of January 1, 1976. Vital status was determined through records maintained by Federal and State agencies, including the Social Security Administration, state motor vehicle registration, and state vital statistics offices. For those individuals who could not be located through these sources, U.S. Postal Mail Correction Services and other follow-up searches were used. For all those who were known to be deceased, death certificates were requested and causes of death were interpreted by a qualified nosologist according to the International Classification of Diseases (ICDA) in effect at the time of death and then converted to the 7th Revision of the ICDA. Those who had an unknown vital status were assumed to be alive as of January 1, 1976 so that the true risk of mortality was not overestimated. Those who died after January 1, 1976 were considered alive for purposes of analysis.

Person-years were accumulated for each worker starting at the point in time when three months of employment in exposed jobs was completed and ending at the date of death or the study end date (1/1/76), whichever occurred first. Using a modified life table computer program similar to that described by Cutler, (16) the person-years for each cohort were combined into five-year calendar and five-year age time periods and multiplied by the corresponding U.S. white male (for male cohort members) and U.S. white female (for female cohort members)

cause-specific mortality rates to yield the expected number of deaths. Person-years were additionally distributed by five-year exposure and five-year latency (number of years from date first employed in exposed jobs to date of death or study end date) categories. Observed and expected cause-specific deaths were compared and differences were tested using the Poisson distribution.

The detailed industrial hygiene surveys included personal time-weighted air samples of employees from selected job titles, as well as area air samples. In both plants, samples were taken for PCB's (Aroclor 1016), trichloroethylene, lead, tin, and zinc. In addition, samples for toluene, methyl isobutyl ketone (MIBK), aluminum and iron were taken at Plant 1. These surveys were designed to characterize the exposures occurring at the time of the survey and may not represent exposures of previous years, especially those of Plant 1 where exposures may have been reduced because of new production techniques which had recently been initiated.

RESULTS

There were a total of 2,567 workers who met the definition of the study cohort. Table 1 gives a breakdown of vital status ascertainment and the number of person-years within each sub-cohort. The vital status ascertainment was more than 97 percent complete.

The possibility of missing records from the personnel files that were used to assemble the Plant 1 cohort was questioned at the initiation of the study. In an effort to determine whether or not eligible workers were missing from the plant 1 cohort, a validity check was conducted by the New York State Department of Health (personal communication from Phil Taylor, NYSDH, April, 1980), similar to that described by Marsh et al. (17). Social Security Administration (SSA) quarterly earning statements (SSA form 941) from 1945-1965 were obtained and compared to the names appearing on the microfilmed personnel records which were used to assemble the cohort. The results of this comparison yielded 35 additional workers (3.5 percent of cohort) who should have been included in the plant 1 study cohort. The vital status of these missing workers is not known at this time, however, the NYSDH is currently trying to ascertain this information. Nevertheless, the validity check confirmed that only a small portion of the total population at risk was missing from the study cohort and the results should not be biased. According to plant officials, there was no reason to believe that the personnel file system at Plant 2 was missing records, and it appeared from our inspection that the personnel file system had been maintained intact.

Tables 2 and 3 summarize the number of deaths observed from the study cohorts and the number of deaths expected. The all-cause mortality is lower than expected in each cohort, with an SMR (SMR = observed

deaths/expected deaths x 100) of 99 (73 obs. vs. 73.2 exp.) for Plant 1 and an SMR of 89 (90 obs. vs. 100.84 exp.) for Plant 2. These SMR's may be affected by the "healthy worker effect". (18) There was no increase in observed mortality for any of the major causes of death listed in Table 2.

Table 3 lists the observed and expected number of deaths by specific cancer cause and for cirrhosis of the liver. When both cohorts were combined, the observed number of deaths was more than that expected for cancer of the rectum (4 obs. vs. 1.07 exp.) and liver cancer - ICD=155, 156A (3 obs. vs. 0.88 exp.). The only statistically significant difference (at $p<0.05$) in observed versus expected deaths occurred in females from Plant 2 for cancer of the rectum (3 obs. vs. 0.46 exp.; $p<0.05$). For both cohorts combined, there were 6 deaths due to cirrhosis of the liver, while 5.47 were expected. Five of these cases were from the Plant 2 cohort while 3.1 were expected. According to hospital reports, at least three of the six persons who died of cirrhosis of the liver were known to have consumed alcohol on a regular basis.

The relationship between latency and the mortality from all cancer, cancer of the rectum, liver cancer, and cirrhosis of the liver is shown in Table 4. For "all cancer" there is no apparent pattern in either cohort. However, for cancer of the rectum, there is a slight

increase with an increase in the latency period. The risk of mortality due to cirrhosis of the liver does not show a consistent increase with an increase in the latency periods, however there is a greater risk after a 20 year period.

The relationship between these same causes of mortality and length of employment in PCB exposed areas of the plants is given in Table 5. As indicated in the table there is no increase in mortality with increasing lengths of exposure, except for cirrhosis of the liver, however; the numbers in this comparison are small.

The industrial hygiene survey results of area and personal sampling for PCB's (Aroclor 1016) are summarized in Tables 6 and 7. Due to differences in the production processes, the results by specific jobs or work areas are not comparable between the two plants. However, relative comparisons can be made and the range of concentrations observed in Plant 1 were lower than those in Plant 2. In Plant 1, the time weighted average (TWA) personal air samples ranged from 24 $\mu g/m^3$ to 393 $\mu g/m^3$ and the TWA area air samples ranged from 3 $\mu g/m^3$ to 476 $\mu g/m^3$. In Plant 2, the TWA personal air samples ranged from 170 $\mu g/m^3$ to 1260 $\mu g/m^3$ and the TWA area air samples ranged from 50 $\mu g/m^3$ to 810 $\mu g/m^3$. The current OSHA standard and ACGIH TLV for chlorodiphenyl (42 percent chlorine) is 1000 $\mu g/m^3$. There is no current OSHA standard of ACGIH TLV for Aroclor 1016.

Trichloroethylene was measured near the degreasers in both plants. Out of eleven area air samples from Plant 1, all were below 35 ppm except for two which measured 195 ppm and 321 ppm. At Plant 2, three area air samples were taken which ranged from 53.4 ppm to 77.5 ppm. The OSHA standard for trichloroethylene is 100 ppm based on a 8 hour time weighted average.

Even though most exposures to trichloroethylene were usually below the TLV, an attempt was made to exclude workers from the study who were employed around the trichloroethylene degreasers.

Area air samples were measured for tin, lead and zinc near the soldering operations. There were no detectable levels for tin at either plant. Out of four samples collected for lead and zinc at Plant 1, lead was detected in one sample at a level of 12 $\mu g/m^3$, zinc was detected on two samples at levels of 8 and 24 $\mu g/m^3$. At Plant 2, fifteen samples were collected for lead and zinc. All but one of these samples showed no detectable levels for lead, the one detectable sample was 41.2 $\mu g/m^3$. Six of the fifteen samples found concentrations of zinc ranging from 2.3 to 94.1 $\mu g/m^3$. The current OSHA standard for lead and zinc oxide (reported as zinc) are 50 $\mu g/m^3$ and 5 mg/m^3 respectively.

Both personal and area samples were taken around the welding operations for measuring aluminum and iron at Plant 1. The aluminum samples ranged from non-detectable to 233 $\mu g/m^3$ and the iron samples from 47 $\mu g/m^3$ to 123 $\mu g/m^3$. The ACGIH TLV for aluminum (Al_2O_3) is 10 mg/m^3 and the current OSHA standard for iron oxide (measured as iron) is 10 mg/m^3.

Twelve personal samples were collected for toluene and MIBK during painting operations at Plant 1. Toluene concentrations ranged from 0.48 to 22 ppm and MIBK ranged from 2 to 5 ppm. The current OSHA standard for toluene is 200 ppm and 100 ppm for MIBK.

Although the exposures to PCB's at the time of the surveys (Plant 1 - April, 1977; Plant 2 - March, 1977), were relatively higher in Plant 2, the, historic levels of exposure may have been more equivalent. It is these exposures that occurred 20 to 30 years ago that are more relevant when considering the occupational cancer risk among the study cohorts. The PCB mixtures used during these time periods were Aroclor 1254 and 1242, whereas, Aroclor 1016 was first used in 1971. In addition, several different stabilizers have been added to the PCB's (1 percent or less by weight) used at Plant 1 since the early 1960's. These include potential carcinogens such as diglyceride ether-disphenol-a and more recently, vinyl cyclohexene dioxide. It is not known which stabilizers have been used at Plant 2.

There are few previous epidemiologic studies that have examined the long term health effects of humans exposed to PCB's. Individuals poisoned by rice oil heavily contaminated with PCB's (Yusho Disease) have been studied extensively years after the incident took place in Japan in 1968. (19, 20) However, the rice oil contaminant also contained polychlorinated dibenzofurans, and quarter phenyls in higher concentrations than that found in commercially prepared PCB's. A high prevalence of skin and eye conditions were noted in the Yusho patients. In addition, there were clinical and laboratory findings that included changes in the microanatomy of liver cells and a decreased concentration of bilirubin in the serum of these individuals. (21, 22)

Early reports regarding the health effects from occupational exposure to PCB's include chloracne (23), digestive disturbances, eye irritation, liver injury and impotence. (24, 25) Most of these findings have been reported as case histories.

In a recent study of volunteers conducted by the Mount Sinai School of Medicine (26), 326 workers who were employed at Plant 1 were examined. The most prevalent symptoms noted were dermatological, and those of the central nervous system. There was a low prevalence of

abnormal liver findings on physical examination. However, a subgroup exposed to PCB's were found to have liver enzyme changes different from those of a normal, non-exposed group. In addition, abnormal SGOT levels were associated with plasma levels of PCB's. There was a relatively high prevalence of decreased lung capacity among a subgroup of 243 workers tested. (27)

In a preliminary report, Bahn (28) reported an increase in deaths due to malignant melanoma (2 obs. vs. 0.04 exp.) and cancer of the pancreas among 51 research and development employees and 41 refinery plant employees at a New Jersey petrochemical facility. These individuals were considered to have had some exposure to Aroclor 1254 during various periods between 1949 and 1957, along with exposure to other toxic and potentially carcinogenic compounds.

In a summary of case histories (G. Roush. Written communication to NIOSH, September, 1976) among approximately 300 workers employed in the manufacturing of PCB's, no malignant melanomas or pancreatic cancers were observed. However, among the death certificates of 50 former workers at this manufacturing facility, seven cases of lung cancer were observed whereas 2.7 cases were expected. The findings were preliminary and were not adjusted for age or smoking.

The previously reported findings of an increased risk for mortality due to malignant melanoma, cancer of the pancreas, and lung cancer among workers exposed to PCB's were not corroborated in the present study. There were no observed deaths due to malignant melanoma and only 1 observed death from pancreatic cancer while 1.77 were expected. There were 7 observed deaths from respiratory system cancer, whereas 7.69 were expected. The only categories of cancer in which the number of observed deaths were greater than expected were for cancer of the rectum and cancer of the liver and only slight increases for cancer of intestine except rectum, and breast cancer. When both cohorts and sex groups were combined none of the excesses were statistically significant at $p<0.05$. However, the excess in liver cancer is noteworthy because it is consistent with the toxicology data observed in laboratory animals exposed to PCB's, where effects have been noted in the liver (6-13). The slight increase in deaths due to cirrhosis of the liver in the Plant 2 cohort is also consistent with the notion that PCB's have a toxic effect on the liver.

In most occupational health studies where cancer mortality is being assessed, latency is an important variable; the hypothesis being that there is an increased risk of mortality once a certain time period after initial exposure has elapsed. In this study, this hypothesis is difficult to examine due to the small number of deaths. None of the causes of death analyzed according to latency clearly demonstrated

this association. Rectal cancer showed a slight increase with an increase in latency and cirrhosis of the liver showed an increase in risk with an increase in latency after 20 years.

There was no relationship between increasing lengths of employment in PCB exposed jobs and the risk of mortality due to cancer or cirrhosis of the liver.

When the cancer mortality is examined by plant, it is evident that most of the excesses occur in plant 2, especially among the female group. This finding may be related to heavier exposures to PCB's at plant 2 as indicated by the industiral hygiene results. In addition, there was an opportunity for earlier exposures at plant 2, potentially allowing for a longer latency period. However, this difference in mortality may be a function of the size of the cohorts (plant 1 only has half the number of person-years as plant 2) and thus simply be a statistical quirk.

A potential confounding variable or interaction variable in this study is the possible effect of alcohol ingestion on the observed increase (at Plant 2) in mortality from cirrhosis of the liver. However, this cannot be properly assessed in the present study since not enough is known about the ingestion of alcohol among the entire study cohort.

Due to a relatively small number of deaths, conclusions drawn from the results of this study are tentative.

Despite these study limitations, observed excesses for liver cancer and cirrhosis of the liver are consistent with previously reported findings on experimental animals exposed to PCB's, and suggest that there may be an association between these causes of death and occupational exposure to PCB's (Aroclor 1254 and 1242). The observed excess in cancer of the rectum related to PCB workers was unexpected and needs further investigation.

REFERENCES

1. Hutzinger, O., Safe, S., Zitko, V. 1974. The Chemistry of PCB's. Cleveland, The Chemical Rubber Co. Press.

2. Yobs, A.R. 1972. Levels of polychorinated biphenyls in adipose tissue of the general population of the nation. Environ. Health Perspect., Experimental issue No. 1: 79-81.

3. Price, H.A., Welch, R.L. 1972. Occurrence of polychorinated biphenyls in humans. Environ. Health Prospect., Experimental issue No. 1: 73-78.

4. Jensen, S., Johnels, A.G., Olsson, M. and Otterlind, G. 1969. DDT and PCB in marine animals from Swedish waters. Nature, 224:247-250.

5. Jensen, S. 1972. The PCB story, Ambio, 1:123-131.

6. Von Wedel, H., Holla, W.A., Denton, J. 1943. Observations on the toxic effects resulting from exposures to chlorinated naphthalene and chlorinated phenyls with suggestions for prevention. Rubber Age 54:419-26.

7. Miller, J.W. 1944. Pathologic changes in animals exposed to a commercial chlorinated diphenyl. Public Health Rep. 59:1085-93.

8. Bruckner, J.V., Khanna, K.L., Cornish, H.H. 1974. Polychlorinated biphenyl-induced alteration of biologic parameters in the rat. Toxicol. Appl. Pharmacol. 28:189-99.

9. Kimbrough, R.D., Linder, R.E., Gaines, T.B. 1972. Morphological changes in livers of rats fed polychlorinated biphenyls. Arch. Environ. Health 25:354-64.

10. Kimbrough, R.D., Linder, R.E., Burse, V.W., Jennings, R.W. 1973. Adenofibrosis in the rat liver -- With persistence of polychlorinated biphenyls in adipose tissue. Arch. Environ. Health 27:390-95.

11. Kimbrough, R.D., Linder, R.E. 1974. Induction of adenofibrosis and hepatomas of the liver in BALB/cJ mice by polychlorinated biphenyls (Aroclor 1254). J. Natl. Cancer Inst. 53:547-52.

12. Allen, J.R., Abrahamson, L.J., Norback, D.H. 1973. Biological effects of polychlorinated biphenyls and triphenyls on the subhuman primate. Environ. Res. 6:344-54.

13. Vos, J.G., Notenboom-Ram, E. 1972. Comparative toxicity study of 2,4,5,2',4',5'-hexachlorobiphenyl and a polychlorinated biphenyl mixture in rabbits. Toxicol. Appl. Pharmacol. 23:563-78.

14. Proceedings of the National Conference on Polychlorinated Biphenyls. EPA, Office of Toxic Substances, Washington, D.C., March, 1976.

15. NIOSH, CDC, PHS, DHEW. 1977. Criteria for a recommended standard...Occupational Exposure to polychlorinated biphenyls (PCB's).

16. Cutler, S.J., Ederer, F. 1958. Maximum Utilization of the life table methods in analyzing survival. J. Chron. Dis., 8:699-709.

17. Marsh, G.M., Enterline, P.E., 1979. A Method for Verifying the completeness of cohorts used in occupational mortality studies. JOM, In Press.

18. McMichael, A.J., Haynes, S.G., Tyroler, H.A. 1975. Observations on the evaluation of occupational mortality data. J. Occup. Med. 17:128-131.

19. Kuratsune, M., Masuda, Y., Nagayama, J. 1976. Some of the Recent Findings Concerning Yusho, in Proceedings of the National Conference on Polychlorinated Biphenyls, November 19-21, 1975, Chicago, EPA-560/6-75-004. U.S. Environmental Protection Agency, Office of Toxic Substances, 1976, pp. 14-29.

20. Urabe, H. 1974. [Foreward, The fourth reports of the study of "Yusho" and PCB.] Fukuoka Acta Med. 65:1-4, (Jap).

21. Hirayama, C., Irisa, T., Yamamoto, T. 1969. [Fine structural changes of the liver in a patient with chlorobiphenyls intoxication.] Fukuoka Acta Med. 60:455-56 (Jap).

22. Hirayama, C., Okumura, M., Nagai, J., Masuda, Y. 1974. Hypobilirubin in patients with polychlorinated biphenyls poisoning. Clin. Chim. Acta. 55:97-100.

23. Meigs, J.W., Albom, J.J., Kartin, B.L. 1954. Cloracne from an unusual exposure to Aroclor. JAMA 154:1417-18 .

24. Schwartz, L. 1936. Dermatitis from synthetic resins and waxes. Am. J. Public Health 26:586-92.

25. Drinker, C.K., Warren, M.F., Bennett, G.A. 1937. The problem of possible systemic effects from certain chlorinated hydrocarbons. J. Ind. Hyg. Toxicol. 19:283-99.

26. Fischbein, A., Wolff, M.S., Lilis, R., Thornton, J., Selikoff, I.J. 1979. Clinical findings among PCB-exposed capacitor manufacturing workers. New York Academcy of Sciences. 320:703-715.

27. Warshaw, R., Fischbein, A., Thornton, J., Miller, A., Selikoff, I.J. 1979. Decrease in Vital Capacity in PCB-Exposed Workers in a Capacitor Manufacturing Facility. New York Academy of Sciences. 320:277-284.

28. Bahn, A.K., Rosenwaike, I., Herrmann, N., Grover, P., Stellman, J., O'Leary, K.: Melanoma after exposure to PCB's. N. Engl. J. Med. 295:450, 1976.

Table 1

Vital Status of PCB Workers

	Plant 1			Plant 2			Grand Total
	Males	Females	Total	Males	Females	Total	
Known to be alive	520	360	880	633	836	1,469	2,349
Known to be deceased	55	18	73	28	62	90	163
Unknown vital status	8	7	15	14	26	40	55(2%)
Total	583	385	968	675	924	1,599	2,567
Person-Years	7,800	5,181	12,981	9,191	16,718	25,909	38,890

Table 2

Observed and Expected Deaths (O/E) According to
Major Causes Among PCB Workers

Cause of Death	Plant 1		Plant 2		Total	(SMR)
(7th Revision ICD No.)	Males	Females	Males	Females		
All Malignant Neoplasms (140-205)	9/ 8.97	4/ 6.62	3/ 6.13	23/18.90	39/ 40.62	(96)
Nervous System (330-334, 345)	3/ 3.08	1/ 1.92	2/ 1.78	5/ 5.52	11/ 12.30	(89)
Circulatory System (400-468)	26/22.05	7/ 6.64	14/13.87	13/19.30	60/ 61.86	(97)
Accidents (800-962)	7/ 5.86	1/ 1.06	3/ 7.26	2/ 3.46	13/ 17.64	(74)
All Other Causes	10/12.06	5/ 4.94	6/ 9.53	19/15.09	40/ 41.62	(97)
All Causes	55/52.02	18/21.18	28/38.57	62/62.27	163/174.04	(94)

Table 3

Observed and Expected Deaths (O/E) According to Specific Cancer Causes and Cirrhosis of the Liver Among PCB Workers

Cause of Death	Plant 1		Plant 2		Total	(SMR)
(7th Revision ICD No.)	Males	Females	Males	Females		
All Malignant Neoplasms (140-205)	9/8.97	4/6.62	3/6.13	23/18.90	39/40.62	(96)
Stomach (151)	0/0.48	0/0.20	1/0.28	0/ 0.58	1/ 1.54	(65)
Intestine exp. Rectum (152, 153)	1/0.79	0/0.67	0/0.51	3/ 1.93	4/ 3.90	(103)
Rectum (154)	1/0.28	0/0.16	0/0.17	3/ 0.46*	4/ 1.07*	(374)
Biliary Pass Liver Liver not specified (155, 156A)	1/0.19	0/0.14	0/0.11	2/ 0.44	3/ 0.88	(341)
Pancreas (157)	0/0.50	1/0.24	0/0.32	0/ 0.71	1/ 1.77	(56)
Respiratory System (160-164)	5/3.15	1/0.66	0/2.14	1/ 1.74	7/ 7.69	(91)
Breast (170)	------	1/1.83	------	6/ 4.94	7/ 6.77	(103)
Lymphatic & Hematopoietic (200-205)	0/1.00	0/0.50	0/0.83	2/ 1.56	2/ 3.89	(51)
Other	1/3.58	1/2.22	2/1.77	6/ 6.56	10/14.13	(71)
Cirrhosis of Liver (581)	1/1.66	0/0.71	2/1.22	3/ 1.88	6/ 5.47	(110)

* $p<0.05$

Table 4

Observed and Expected Deaths According to Latency[1]
Among PCB Workers

I. All Cancers

Latency (years)	Plant 1			Plant 2			Plants 1 & 2		
	O	E	SMR	O	E	SMR	O	E	SMR
< 10 yrs.	6	4.83	124	6	7.29	82	12	12.12	99
10-<20 yrs.	3	6.07	49	16	10.24	156	19	16.31	116
> 20 yrs.	4	4.65	86	4	7.50	53	8	12.15	66
II. Cancer of Rectum (ICD = 154)									
< 10 yrs.	0	0.13	----	0	0.19	-----	0	0.32	---
10-<20 yrs.	0	0.17	----	2	0.26	769	2	0.43	465
> 20 yrs.	1	0.13	769	1	0.18	555	2	0.31	645
III. Liver Cancer (ICD = 155, 156A)									
< 10 yrs.	1	0.09	1111	1	0.15	666	2	0.24	833*
10-<20 yrs.	0	0.13	----	1	0.23	435	1	0.36	277
> 20 yrs.	0	0.11	----	0	0.16	-----	0	0.27	---
IV. Cirrhosis of Liver (ICD = 581)									
< 10 yrs.	1	0.79	127	1	0.95	105	2	1.74	115
10-<20 yrs.	0	0.98	----	1	1.32	76	1	2.30	43
> 20 yrs.	0	0.59	----	3	0.85	353	3	1.44	208

[1] Latency = number of years from date first employed in exposed job.

* $p<0.05$

Table 5

Observed and Expected Deaths According to Length of Exposure Among PCB Workers

Length of Employment	Plant 1			Plant 2			Plants 1 & 2		
	O	E	SMR	O	E	SMR	O	E	SMR
I. All Cancers (ICD = 140-205)									
≤ 5 yrs.	11	11.49	96	20	17.97	111	31	29.46	112
5-9 yrs.	1	2.59	39	2	3.65	55	3	6.24	48
10-14 yrs.	0	0.84	----	3	2.01	149	3	2.85	105
15-19 yrs.	1	0.59	169	1	0.89	112	2	1.48	135
≥ 20 yrs.	0	0.08	----	0	0.51	----	0	0.59	----
II. Cancer of Rectum (ICD = 154)									
≤ 5 yrs.	1	0.32	313	0	0.45	222	2	0.77	260
5-9 yrs.	0	0.08	----	0	0.10	----	0	0.18	----
10-14 yrs.	0	0.03	----	2	0.05	4000	2	0.08	2500
15-19 yrs.	0	0.02	----	0	0.02	----	0	0.04	----
≥ 20 yrs.	0	0.001	----	0	0.01	----	0	0.01	----
III. Liver Cancer (ICD = 155, 156A)									
≤ 5 yrs.	1	0.24	417	2	0.40	500	3	0.64	469
5-9 yrs.	0	0.06	----	0	0.08	----	0	0.14	----
10-14 yrs.	0	0.02	----	0	0.05	----	0	0.07	----
15-19 yrs.	0	0.01	----	0	0.01	----	0	0.02	----
≥ 20 yrs.	0	0.001	----	0	0.01	----	0	0.01	----
IV. Cirrhosis of the Liver (ICD = 581)									
≤ 5 yrs.	1	1.76	57	2	2.22	90	3	3.98	75
5-9 yrs.	0	0.38	----	1	0.46	217	1	0.84	119
10-14 yrs.	0	0.11	----	1	0.22	455	1	0.33	303
15-19 yrs.	0	0.09	----	1	0.12	833	1	0.21	476
≥ 20 yrs.	0	0.02	----	0	0.07	----	0	0.09	----

Table 6

Concentrations of PCB's (Aroclor 1016) at Plant 1 - Taken April, 1977

A. Power Capacitor Manufacturing Facility

Personal Air Samples				Area Air Samples			
Job Titles	No. of Samples	Total Sampling Time (minutes)	TWA* (g/m)	Location	No. of Samples	Total Sampling Time (minutes)	TWA* (g/m)
Recovery Repair	2	840	298	Test & Paint	2	840	41
Salvage Operator	1	426	155	Assembly	2	851	29
EMF Operator	1	431	115	Shipping	1	426	16
Treat Helper	2	867	80	Storage	1	427	14
Treat Operator	2	731	66	Winding	1	420	3
Repair	1	422	50				

B. Small Capacitor Manufacturing Facility

Moveman (Sealing Area)	2	689	393	Soldering	2	782	476
Moveman (Testing Soldering Area) 827 115			3	1306	220	Assembly	2
Testing	3	1290	218	Shipping	2	838	56
Packer	3	1287	199	Winding	2	828	54
Treat Operator	2	845	160	Can Manufacturing	2	836	51
Rework & Final Assembly	2	824	152	Cover Manufacturing	2	834	45
Maintenance	1	404	150				
Rework Tester	1	433	140				
Rework Packer	1	435	132				
Rework Tester Solder	1	271	24				

* TWA is calculated over the total sampling time period.

Table 7

Concentrations of PCB's (Aroclor 1016) at Plant 2 - Taken March, 1977

	Personal Air Samples				Area Air Samples		
Job Titles	No. of Samples	Total Sampling Time (minutes)	TWA* (g/m)	Location	No. of Samples	Total Sampling Time (minutes)	TWA* (g/m)
Degreaser	1	381	1,260	Impregnation	2	176	810
Solder	3	884	1,060	Pump Room	3	1079	490
Tanker	9	2120	850	Testing	5	1424	320
Moveman (Soldering Area)	3	752	720	Pre-assembly	4	1213	140
Heat Soak Operator	3	872	630	Shipping	2	741	90
Tester	3	917	290	Winding	4	637	70
Pump Mechanic	1	377	280	Cover Manufacturing	3	1089	60
Floorman (pre-assembly)	6	1683	170	Office	2	741	50

* The TWA is calculated over the total sampling time period.

Unidentified Speaker: I know your numbers are small, but were you able to do separate analyses by sex? In particular, were there liver cancers in women?

Mr. Brown (NIOSH): I did separate analyses by sex. I will have to go back to check to see about the liver cancers in the women. I am not sure.

Unidentified Speaker: In both plants, TCE was used as a degreaser and it is known that TCE has induced liver cancer in laboratory animals. How did you separate those exposed to TCE from those exposed to PCB's to determine whether or not some of the excess is attributable to TCE?

Mr. Brown (NIOSH): Based on the work histories that we got from the plant, we could tell who worked around the TCE degreaser and we eliminated them from our study.

NIOSH

PROCEEDINGS OF THE
FIRST NCI/EPA/NIOSH COLLABORATIVE WORKSHOP:
PROGRESS ON JOINT ENVIRONMENTAL AND
OCCUPATIONAL CANCER STUDIES

MAY 6-8, 1980

SHERATON/POTOMAC, ROCKVILLE, MARYLAND

The papers included in these Proceedings were printed as they were submitted to this office.

Appropriate portions of the discussions, working groups and plenary session were sent to the participants for editing. The style of editing varied, as could be expected. To the extent possible, we have attempted to arrive at a consistent format.

PROCEEDINGS OF THE FIRST NCI/EPA/NIOSH COLLABORATIVE WORKSHOP: PROGRESS ON JOINT ENVIRONMENTAL AND OCCUPATIONAL CANCER STUDIES

MAY 6-8, 1980

SHERATON/POTOMAC, ROCKVILLE, MARYLAND

Proceedings were developed from a workshop on the National Cancer Institute's, the Environmental Protection Agency's and the National Institute for Occupational Safety and Health's Collaborative Programs on Environmental and Occupational Carcinogenesis.

PROCEEDINGS OF THE FIRST NCI/EPA/NIOSH COLLABORATIVE WORKSHOP: PROGRESS ON JOINT ENVIRONMENTAL AND OCCUPATIONAL CANCER STUDIES

Editors

H. F. Kraybill, Ph. D.
Ingeborg C. Blackwood
Nancy B. Freas

National Cancer Institute

Editorial Committee

Thomas P. Cameron, D.V.M.
Morris I. Kelsey, Ph. D.
National Cancer Institute

Wayne Galbraith, Ph. D.
C. C. Lee, Ph. D.
Environmental Protection Agency

Kenneth Bridbord, M. D.
National Institute for Occupational Safety and Health

Technical Assistance

Sara DeLiso
Donna Young
National Cancer Institute