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Mortality and Industrial Hygiene Study of Workers Exposed to Polychlorinated Biphenyls

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ABSTRACT. Because of the demonstrated toxic effects on animals resulting from exposure to polychlorinated biphenyls (PCBs), the National Institute for Occupational Safety and Health conducted a retrospective cohort mortality study of 2,567 workers in two plants where PCBs were used in the manufacture of electrical capacitors. All workers included in the study were employed for at least 3 months in areas of the plants where PCBs were used. The vital status of 98% of the two cohorts was determined, and 39,018 person-years were accumulated. All-cause mortality was lower than expected (163 obs. vs 182.4 exp.) as well as all cancer mortality (39 obs. vs 43.8 exp.). Excess mortality was noted for rectal cancer (4 obs. vs 1.19 exp.) and liver cancer (3 obs. vs 1.87 exp.), although neither excess was statistically significant. In one of the plants the observed mortality due to cirrhosis of the liver was also elevated. The results of detailed industrial hygiene surveys conducted in each plant are also presented.

POLYCHLORINATED BIPHENYLS (PCBs) are a class of compounds composed of biphenyl molecules with a varying number of substituted chlorine atoms. In commercially prepared PCB mixtures, the weight-percent of chlorine has varied from 21 to 68%. In some preparations, there has also been some degree of contamination by chlorodibenzofurans.[1]

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

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

During the past few years, interest in the health effects among individuals exposed to PCBs has been stimulated by: (a) the tendency for PCBs to accumulate in tissues and certain organs;[2,3] (b) the stability of PCBs and their persistence in the environment;[4-6] and (c) the demonstrated long-term toxic effects, including liver tumors and other liver diseases, in exposed laboratory animals.[7-13] Much of this interest was expressed at the National Conference on Polychlorinated Biphenyls in November, 1975,[14] and the toxicity of PCBs has been extensively reviewed in the NIOSH Criteria Document on PCBs.[15] In comparison to

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the accumulated information on acute toxic effects in humans and adverse effects in animals, little is known about the chronic effects from long-term exposure in man.

To determine whether past occupational exposure to commercially produced PCBs 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 to document the levels of exposure to PCBs and other chemicals.

Description of Facilities

Both of the plants chosen for study manufacture electrical capacitors and were selected because: (a) each had a large work force; (b) PCBs had been used for more than 30 yr; (c) there was considerable potential for exposure to PCBs with little potential for exposure to other known toxic contaminants; and (d) the records necessary to identify individuals to be included in the study population were readily available. At the time the study was initiated, both plants were still using PCBs. Plant 1 is located in New York State and is divided into two manufacturing facilities within close proximity. One facility that has used PCBs since 1946 produced small industrial capacitors and the other facility has produced large PCB-filled power capacitors since 1951. The type of PCBs used has varied during the years from "Aroclor" (Monsanto trade name) 1254 (54% chlorine) to 1242 (42% chlorine) to 1016 (41% chlorine). In addition, several other kinds of oils were used, but in a limited number of capacitors.

Plant 2, located in Massachusetts, began to use PCBs to manufacture capacitors in 1938. This plant also changed the type of PCBs used from "Aroclor" 1254 to 1242 to 1016. Until 1972, other types of capacitors which did not contain PCBs were made at this plant. Castor oil was used in lieu of PCBs to produce the large power capacitors at this plant.

Both plants assembled small and large type capacitors using the same general techniques. The following briefly describes the assembly process.

Winding and pre-assembly. The inner components of the capacitor were made of paper, foil, and sometimes plastic film; wound together; and subsequently loaded into metal casings. This job was done in an enclosed dust-free room where there was minimal exposure to PCBs. Therefore, the workers in these jobs were not considered "exposed" when choosing the study cohort.

Impregnation. The pre-assembled capacitors were filled or impregnated with the PCBs. Within this area there was potential for exposure to PCBs, and therefore, those employed in this area were considered "exposed" when choosing the study cohort.

Final assembly. The tops of the capacitors were closed by crimping, rubber stoppers, or soldering, which involved some exposure to PCBs. The capacitors were washed to remove excess PCBs by running them through a detergent wash or a degreaser such as trichloroethylene. Finally, they were sent through the final operations involving drying, testing, and painting. Those employed in several of these jobs were considered "exposed" when choosing the study cohort.

Other areas where there was potential exposure to PCBs in the plants included the laboratory and the area where rejected capacitors were rebuilt. Approximately 10% of the two work forces were employed in areas where there had been potential exposure to PCBs. Those employed in these jobs were considered "PCB exposed" for purposes of choosing the study cohorts.

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

METHODS

Mortality study. A retrospective cohort mortality study was conducted to determine whether individuals occupationally exposed to PCBs have experienced any increase in cause-specific mortality. The study cohorts were defined as all workers who accumulated at least 3 months of employment at any time in areas of the plants where there was a potential for exposure to PCBs. These "exposure jobs" were designated by the companies and verified by the labor unions (at Plant 1), and by the NIOSH industrial hygiene surveys to represent the high-exposure jobs. Trichloroethylene (TCE) was used as a degreaser in both plants. Therefore, if the work history records indicated that an employee had potential exposure to TCE, the individual was not included in the cohort. This included very few workers.

An effort was made to determine the vital status (living 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 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, therefore the true risk of mortality was not overestimated. Those who died after January 1, 1976, were considered to be alive for purposes of analysis.

Person-years were accumulated for each worker starting after 1940 when 3 months of employment in exposed jobs were 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,[14] the person-years for each cohort were combined into 5-yr calendar time periods and 5-yr age groups 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 5-yr exposure and 5-yr latency (number of years from date first employed in exposed

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Table 1.—Vital Status of PCB Workers

	Plant 1			Plant 2			
	Males	Females	Total	Males	Females	Total	Grand Total
Known to be alive	520	360	880	833	836	1,469	2,349
Known to be deceased	55	18	73	28	82	90	183
Unknown vital status	8	7	15	14	26	40	55 (2%)
Total	583	385	968	875	924	1,599	2,567
Person-years	7,825	5,185	13,010	9,229	16,779	26,008	39,018

jobs) categories. Observed and expected cause-specific deaths were compared and differences were tested using the Poisson distribution.

Industrial hygiene survey. The detailed industrial hygiene surveys included personal time-weighted air samples from selected job titles, as well as area air samples. In both plants, samples were taken for PCBs (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 recently initiated.

RESULTS

Mortality study. A total of 2,567 workers met the definition of the study cohort. Table 1 gives a breakdown of the vital status ascertainment and the number of person-years within each sub-cohort. The vital status ascertainment is 98% complete.

The possibility that records might be missing from the personnel files used to assemble the Plant 1 cohort was cited at the beginning of the study. In an effort to determine whether eligible workers were missing from the Plant 1 cohort, a validity check was conducted by the New York State Department of Health[20] using methodology 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 that were used to assemble the cohort. The results of this comparison yielded 35 additional workers (3.5% of cohort) not included in the Plant 1 study cohort. This small portion of the population at risk that is missing from the study cohort should not seriously bias the results. A similar validity check was not done at Plant 2, as it appeared from our inspection that the personnel file system had been maintained intact.

Table 2 shows the distribution of the cohorts by duration of employment in jobs where PCB exposure occurred. The distribution within the two plants is somewhat similar, with the exception of the female workers in Plant 2, where

Table 2.—Duration of Employment among Cohort Members in PCB Exposure Jobs

Plant 1	Males N (R.F)*	Females N (R.F)	Total N (R.F)
3-6 mo	137 (23.5)	79 (20.5)	216 (22.3)
6 mo-1 yr	88 (15.1)	59 (15.3)	147 (15.2)
1-2 yr	93 (16.0)	92 (23.9)	185 (19.1)
2-3 yr	53 (9.1)	41 (10.6)	94 (9.7)
3-10 yr	165 (28.3)	82 (21.3)	247 (25.5)
10 yr	47 (8.1)	32 (8.3)	79 (8.2)
Total	583	385	968
Plant 2	**Males N (R.F)**	**Females N (R.F)**	**Total N (R.F)**
3-6 mo	211 (31.3)	207 (23.4)	418 (26.1)
6 mo-1 yr	127 (18.8)	161 (17.4)	288 (18.0)
1-2 yr	118 (17.5)	175 (18.9)	293 (18.3)
2-3 yr	64 (9.5)	82 (8.9)	146 (9.1)
3-10 yr	123 (18.2)	188 (20.3)	311 (19.4)
10 yr	32 (4.7)	111 (12.0)	143 (8.9)
Total	675	924	1599

*R.F = Relative frequency.

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Table 3.—Observed and Expected Deaths (O/E) According to Major Causes among PCB Workers

Cause of Death (7th Revision ICD No.)	Plant 1 Males	Plant 1 Females	Plant 2 Males	Plant 2 Females	Total	(SMR)	95% Confidence (CI)
All malignant neoplasms (140-205)	9/ 9.70	4/ 7.36	3/ 6.83	23/ 20.90	39/ 43.79	(89)	(63 - 122)
Nervous system (230-334, 340)	3/ 3.54	1/ 1.97	2/ 1.84	5/ 5.60	11/ 12.85	(86)	(44 - 157)
Circulatory system (400-450)	30/ 22.31	7/ 6.83	14/ 16.15	13/ 10.64	69/ 62.93	(95)	(73 - 125)
Accidents (800-962)	7/ 6.88	1/ 1.17	3/ 7.43	3/ 3.67	13/ 18.29	(71)	(38 - 122)
All other causes	10/ 12.80	5/ 5.84	6/ 10.28	19/ 16.19	40/ 44.70	(89)	(64 - 122)
All causes	55/ 53.97	18/ 22.77	28/ 40.51	62/ 65.10	163/ 182.38	(89)	(76 - 104)

more employees had worked for 10 or more yr, and in male workers where there was a high frequency of short-term (3-6 months) employees.

When the two cohorts are examined by year first employed in jobs where PCB exposure occurred, the females in Plant 2 are seen to have had an earlier initial date of exposure. In Plant 1, 49.4% of the males and 45.1% of the females were first employed in PCB exposure jobs before 1955. In Plant 2, 49.3% of the males and 69.6% of the females were first employed in PCB exposure jobs before 1955.

Tables 3 and 4 summarize the number of deaths observed (obs.) from the study cohorts and the number of deaths expected (exp.). The all-cause mortality is lower than expected in each cohort, with an SMR [Standardized Mortality Ratio (SMR = observed deaths/expected deaths X 100)] of 95 (73 obs. vs 76.7 exp.) for Plant 1 and an SMR of 85 (90 obs. vs 105.6 exp.) for Plant 2. These SMRs may be influenced by the "healthy worker effect."[18] There is no increase in observed mortality among the total cohort for any of the major causes of death listed in Table 3.

Table 4 lists the observed and expected number of deaths by specific cancer cause and for cirrhosis of the liver. When both cohorts are combined, the observed number of deaths is more than that expected for cancer of the rectum (4 obs. vs 1.19 exp.) and liver cancer—ICDA = 155, 156A (3 obs. vs 1.07 exp.). The only statistically significant difference ($P < .05$) in observed versus expected deaths occurred in females from Plant 2 for cancer of the rectum (3 obs. vs 0.50 exp., $P < .05$). For both cohorts combined, there are 6 deaths due to cirrhosis of the liver, while 5.60 were expected. Five of these cases are from the Plant 2 cohort, while 3.2 were expected. According to hospital reports, at least 3 of the 6 persons who died of cirrhosis of the liver were known to have consumed alcohol regularly.

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 5. For "all cancer" there is no apparent pattern in either cohort. For cancer of the rectum, there is a slight increase with an increase in the latency periods. All of the deaths due to liver cancer occur before 20 yr of latency and there is no trend of increasing risk 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; there is however, a greater risk after a 20-yr period.

The relationship between these same causes of death and length of employment in PCB exposure areas of the plants is given in Table 6. 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.

Industrial hygiene survey. The industrial hygiene survey results of area and personal sampling for PCBs (Aroclor 1016) are summarized in Tables 7 and 8. Because of 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 are 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 $\mu g/m^3$. The TWA personal air samples in Plant 2 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 $\mu g/m^3$.

Trichloroethylene was measured near the degreasers in both plants. Of 11 area air samples from Plant 1, all were less than 35 ppm, except for two which measured 195 ppm and 321 ppm. As Plant 2, three area air samples were taken which ranged from 53.4 ppm to 77.5 ppm.

Area air samples were measured for tin, lead, and zinc near the soldering operations. There were no detectable levels for tin at either plant. Of four samples collected for lead and zinc at Plant 1, lead was detected in one sample at a level of 12 $\mu g/m^3$, and zinc was detected on two samples at levels of 8 and 24 $\mu g/m^3$. At Plant 2, 15 samples were collected for lead and zinc; all but one (41.2 $\mu g/m^3$) of these samples showed no detectable levels for lead. Six of the 15 samples revealed concentrations of zinc ranging from 2.3 to 94.1 $\mu g/m^3$.

Both personal and area samples were taken in the area

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Table 4.—Observed and Expected Deaths (O/E) According to Specific Cancer Causes and Cirrhosis of the Liver among PCB Workers

Cause of Death (7th Revision ICD No.)	Plant 1		Plant 2		Total	(SMR)	95% Confidence Interval
	Males	Females	Males	Females			
All malignant neoplasms (140-205)	6/ 8.70	4/ 7.36	3/ 6.83	23/ 20.80	36/ 43.79	(82)	(63 - 122)
Stomach (151)	0/ 0.51	0/ 0.22	1/ 0.31	0/ 0.62	1/ 1.66	(60)	
Intestine exp. rectum (153, 153)	1/ 0.82	0/ 0.70	0/ 0.54	3/ 1.97	4/ 4.00	(90)	(27 - 254)
Rectum (154)	1/ 0.31	0/ 0.18	0/ 0.20	3/ 0.90*	4/ 1.39	(336)	(92 - 860)
Biliary tract liver Liver not specified (155, 156A)	1/ 0.23	0/ 0.10	0/ 0.15	2/ 0.51	3/ 1.07	(280)	(58 - 820)
Pancreas (157)	0/ 0.53	1/ 0.27	0/ 0.35	0/ 0.75	1/ 1.90	(53)	
Respiratory system (160-164)	3/ 3.32	1/ 0.71	0/ 2.22	1/ 1.83	7/ 7.90	(89)	(35 - 181)
Breast (170)	.....	1/ 1.66	.....	0/ 4.06	7/ 6.84	(102)	(41 - 211)
Lymphatic and hematopoietic (200-205)	0/ 1.10	0/ 0.59	0/ 0.84	2/ 1.71	2/ 4.34	(46)	
Other	1/ 2.06	1/ 2.15	2/ 2.12	4/ 7.13	10/ 14.76	(68)	(32 - 134)
Cirrhosis of liver (301)	1/ 1.00	0/ 0.73	2/ 1.26	3/ 1.92	6/ 1.66	(107)	(39 - 233)

*$P < .05$

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Table 2.—Observed and Expected Deaths According to Latency* among Male and Female PCB Workers

I. All Cancers

Latency (yr)	Plant 1 O†	Plant 1 E‡	Plant 1 SMR	Plant 2 O	Plant 2 E	Plant 2 SMR	Plants 1 + 2 O	Plants 1 + 2 E	Plants 1 + 2 SMR
<10 yr	6	5.27	114	8	7.76	77	12	13.03	93
10-<20 yr	3	6.61	45	16	10.91	147	19	17.52	108
>20 yr	4	5.07	79	4	8.17	49	8	13.24	60

II. Cancer of Rectum (ICD = 154)

<10 yr	0	0.15	...	0	0.21	...	0	0.36	...
10-<20 yr	0	0.19	...	2	0.29	690	2	0.48	417
>20 yr	1	0.15	667	1	0.21	476	2	0.36	556

III. Liver Cancer (ICD = 155, 196A)

<10 yr	1	0.12	833	1	0.19	526	2	0.30	667
10-<20 yr	0	0.16	...	1	0.27	370	1	0.43	233
>20 yr	0	0.12	...	0	0.21	...	0	0.33	...

IV. Cirrhosis of Liver (ICD = 581)

<10 yr	1	0.80	125	1	0.95	105	2	1.73	114
10-<20 yr	0	1.01	...	1	1.35	74	1	2.36	424
>20 yr	0	0.61	...	3	0.89	341	3	1.49	201

* Latency = number of years from date first employed in exposed job.
† O = observed deaths.
‡ E = expected deaths.

of welding operations for measuring aluminum and iron at Plant 1. The aluminum samples ranged from nondetectable to 233 $\mu g/m^3$, and the iron samples ranged from 47 $\mu g/m^3$ to 123 $\mu g/m^3$.

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.

Although the exposures to PCBs at the dates of survey (Plant 1—April 1977, Plant 2—March 1977), were relatively higher in Plant 2, the historic levels of exposure may have been more equivalent. The exposures that occurred 20 to 30 yr ago 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 1991. In addition, several different stabilizers have been added to the PCBs (1% or less by weight) used at Plant 1 since the early 1960s. These include potential carcinogens such as diglyceride ether-diphenol-a and, more recently, vinyl cyclohexene dioxide. It is not known which stabilizers have been used at Plant 2.

DISCUSSION

There are few previous epidemiologic studies that have examined the long-term health effects on humans exposed to PCBs. Individuals poisoned by rice oil heavily contaminated with PCBs (Yusho Disease) have been studied extensively years after the incident took place in Japan in 1968.[29,30] However, the rice oil contaminant also contained polychlorinated dibenzofurans and other contaminants in higher concentrations than those found in commercially prepared PCBs. 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.[31,32]

Early reports regarding the health effects from occupational exposure to PCBs include chloracne,[33] digestive disturbances, eye irritation, liver injury, and impotence.[34,35] Most of these findings have been reported as case histories.

In a recent study of volunteers conducted by the Mount Sinai School of Medicine,[36] 326 workers who were em-

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Table 6.—Observed and Expected Deaths According to Length of Exposure among Male and Female PCB Workers

Length of Employment	Plant 1 O*	Plant 1 E†	Plant 1 SMR	Plant 2 O	Plant 2 E	Plant 2 SMR	Plants 1 + 2 O	Plants 1 + 2 E	Plants 1 + 2 SMR
I. All Cancers (ICD = 140-205)									
3 mo -5 yr	11	12.21	90	20	16.76	100	31	30.39	100
5-9 yr	1	2.95	34	3	4.10	40	3	7.85	43
10-14 yr	0	1.80	---	3	2.28	132	3	3.28	91
15-19 yr	1	0.89	145	1	1.04	96	2	1.73	116
≥ 20 yr	0	0.11	---	0	0.63	---	0	0.74	---
II. Cancer of Rectum (ICD = 154)									
3 mo -5 yr	1	0.36	280	1	0.48	208	2	0.83	241
5-9 yr	0	0.09	---	0	0.11	---	0	0.20	---
10-14 yr	0	0.02	---	2	0.09	3333‡	2	0.09	2222‡
15-19 yr	0	0.02	---	0	0.03	---	0	0.05	---
≥ 20 yr	0	0.001	---	0	0.02	---	0	0.02	---
III. Liver Cancer (ICD = 155, 196A)									
3 mo -5 yr	1	0.29	345	2	0.45	444	3	0.74	405
5-9 yr	0	0.08	---	0	0.11	---	0	0.19	---
10-14 yr	0	0.02	---	0	0.06	---	0	0.08	---
15-19 yr	0	0.00	---	0	0.02	---	0	0.04	---
≥ 20 yr	0	0.000	---	0	0.02	---	0	0.02	---
IV. Cirrhosis of the Liver (ICD = 581)									
3 mo -5 yr	1	1.79	56	2	2.36	88	3	4.85	74
5-9 yr	0	0.29	---	1	0.48	208	1	0.87	115
10-14 yr	0	0.13	---	1	0.24	416	1	0.36	278
15-19 yr	0	0.10	---	1	0.13	769	1	0.23	435
≥ 20 yr	0	0.00	---	0	0.08	---	0	0.10	---

*O = observed deaths.
†E = expected deaths.
‡P < .01

played 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 PCBs were found to have liver enzyme changes different from those of a normal, non-exposed group. In addition, abnormal serum glutamic oxaloacetic transaminase (SGOT) levels were associated with plasma levels of PCBs. There was a relatively high prevalence of decreased lung capacity among a subgroup of 243 workers tested.[27]

In a preliminary report, Baker[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 exposed 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 among approximately 300 workers employed in the manufacturing of PCBs,[29] no malignant melanomas or pancreatic cancers were observed. However, among the death certificates of 50 former workers at this manufacturing facility, 7 cases of lung cancer were observed whereas 2.7 cases were expected. The findings were preliminary and were not adjusted for age or smoking.

These previously reported findings of an increased risk of mortality due to malignant melanoma, cancer of the pancreas, and lung cancer among workers exposed to PCBs are not corroborated in the present study. There are no observed deaths due to malignant melanoma and only 1 observed death from pancreatic cancer while 1.89 are expected. There are 7 observed deaths from respiratory

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system cancer, whereas 7.69 are expected. The only categories of cancer in which the number of observed deaths are greater than expected are for cancer of the rectum and cancer of the liver and only a slight increase for breast cancer. When both cohorts and sex groups are combined, none of the excesses are statistically significant at $P < .05$. However, the excess in liver cancer is noteworthy because it is consistent with the toxicology data observed in laboratory animals exposed to PCBs, 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 PCBs 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 has elapsed after initial exposure. In this study, this hypothesis is difficult to examine because of the small number of deaths. None of the causes of death analyzed according to latency clearly demonstrates this association. Rectal cancer shows a slight increase with an increase in latency, and cirrhosis of the liver shows an increase in risk with an increase in latency after 20 yr.

There is no relationship between increasing durations of employment in jobs involving PCB exposure and the risk of mortality due to cancer or cirrhosis of the liver.

When 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 more exposures to PCBs at Plant 2, as indicated by the industrial 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.

Table 7.—Concentrations of PCBs (Aroclor 1016) at Plant 1 (April 1977)

	A. Power Capacitor Manufacturing Facility						
	Personal Air Samples				Area Air Samples		
Job Titles	No. of Samples	Total Sampling Time (min)	TWA* ($\mu g/m^3$)	Location	No. of Samples	Total Sampling Time (min)	TWA* ($\mu g/m^3$)
Recovery Repair	2	840	290	Test and Paint	2	840	41
Salvage Operator	1	436	155	Assembly	2	851	29
EMP operator	1	431	115	Shipping	1	426	16
Trust helper	2	867	88	Storage	1	427	14
Trust operator	2	737	66	Winding	1	420	2
Repair	1	422	50				
	B. Small Capacitor Manufacturing Facility						
Movement (Sealing area)	2	680	393	Soldering	2	782	476
Movement (Testing and soldering area)	2	1290	220	Assembly	2	827	118
Testing	2	1290	218	Shipping	2	836	56
Packer	3	1287	199	Winding	2	828	54
Trust operator	2	840	168	Can Manufacturing	2	836	51
Rework and final assembly	2	834	152	Cover Manufacturing	2	834	45
Maintenance	1	486	150				
Rework tester	1	430	140				
Rework packer	1	436	120				
Rework tester solder	1	271	34				

*TWA is calculated during the total sampling time period.

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Table 8.—Concentrations of PCBs (Aroclor 1016) at Plant 2 (March 1977)

	Personal Air Samples				Area Air Samples		
Job Titles	No. of Samples	Total Sampling Time (min)	TWA* ($\mu g/m^3$)	Location	No. of Samples	Total Sampling Time (min)	TWA* ($\mu g/m^3$)
Degreaser	1	381	1,200	Impregnation	2	176	816
Solder	3	884	1,000	Pump room	3	1079	400
Tanker	9	2120	850	Testing	5	1424	320
Movement (soldering area)	3	752	720	Pre-assembly	4	1213	140
Heat seal operator	3	872	850	Shipping	2	741	90
Tester	3	917	290	Winding	4	637	76
Pump Mechanic	1	377	280	Cover manufacturing	3	1080	60
Floorman (pre-assembly)	6	1083	170	Office	2	741	50

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

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.

CONCLUSIONS

Because a relatively small number of deaths were observed, conclusions drawn from the results of this study are tentative.

All-cause mortality is lower than expected, and there was no increase in mortality for the major causes of death that were examined. Among the cancer causes, there was increased cancer of the liver and rectum. Cirrhosis of the liver was also elevated in one of the plants. The slight excesses for liver cancer and cirrhosis of the liver are consistent with previously reported findings on experimental animals exposed to PCBs, and suggest that there may be an association between these causes of death and occupational exposure to PCBs (i.e., Aroclor 1254 and 1242). However, the findings for liver cancer do not reflect a relationship with latency that has been observed for other carcinogens found in the workplace. The observed excess in cancer of the rectum related to PCB workers was unexpected and requires further investigation.

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