Cancer Mortality Patterns Among Female and Male Workers Employed in a Cable Manufacturing Plant During World War II

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A cohort mortality study was conducted among 9028 (3042 women, 5986 men) workers potentially exposed to chlorinated naphthalenes (chloracnegens structurally similar to dioxins) and asbestos in the manufacture of Navy cable during World War II. Based on mortality through December 31, 1985, standardized mortality ratios (SMRs) for all cancers was 1.03 in women (95% confidence interval [CI] = 0.90 to 1.17) and 1.18 in men (95% CI = 1.10 to 1.26). There were no significant elevations in causes of death hypothesized a priori to be associated with chlorinated naphthalene exposure (malignant neoplasms [MN] of connective tissue, liver, and lymphatic and hematopoietic organs). An excess of MN of the connective tissue was suggested for workers with over 1 year of exposure and 25 years of latency (SMR = 3.54; 95% CI = 0.97 to 9.07). Among cancer sites not hypothesized to be related a priori, three showed concordant excesses among both genders (MN of stomach; rectum; and trachea, bronchus, and lung). No significant elevations occurred in hormonally related cancers among women. Cancer mortality among 460 individuals with chloracne (431 men, 29 women) was similar to that of the entire cohort, although the chloracne subcohort showed significant excesses in two rare causes of death (MN of esophagus, SMR = 3.26; "benign and unspecified neoplasms," SMR = 4.93). Use of county referent rates decreased SMRs for stomach, rectal, and buccal cavity cancer, suggesting a role for nonoccupational risk factors. It is difficult to draw conclusions

Chlorinated naphthalenes were the first agents to cause widespread outbreaks of chloracne in the United States and Europe.[1] Chlorinated naphthalenes consist of two fused aromatic rings with from one to eight of their hydrogens substituted with chlorine. Historically, chlorinated naphthalenes have been used in cable insulation, wood preservatives, capacitors, engine oils, and cutting and grinding fluids.[2] Domestic manufacture of chlorinated naphthalenes declined from over 7 million pounds in 1956 to 700,000 pounds in 1978, and ceased in 1980.[2] The toxicity of chlorinated naphthalenes has been evaluated in cattle exposed via contaminated feed[3,4] and in rats in laboratory studies,[5] but no carcinogenicity data are available.[2] Our interest in studying the mortality experience of workers exposed to chlorinated naphthalenes stems primarily from its toxicologic similarity with chlorinated hydrocarbons of current public health concern, ie, chlorinated dibenzodioxins and polychlorinated biphenyls (PCBs). Based on animal and human data on carcinogenicity of chlorinated dibenzodioxins[6–10] and PCBs,[11–15] we hypothesized a priori that chlorinated naphthalene exposure might be associated with an increased risk of soft tissue sarcoma, lymphoma, and malignant neoplasm (MN) of the liver. Recently, PCB concentration in mammary tissue of patients with breast cancer compared with patients with benign breast disease was reported[16]; another study did not find a significant association between PCB concentration in serum and risk of breast cancer.[17]

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about carcinogenicity of chlorinated naphthalenes because of study limitations, most importantly, concomitant asbestos exposure and the relatively short duration of exposure to chlorinated naphthalenes among most of the cohort.

The cohort under study was exposed to asbestos, and possibly carbon tetrachloride and PCBs, in addition to chlorinated naphthalenes. In epidemiologic studies, asbestos has been consistently associated with cancer of the lung and with mesothelioma; some studies have shown excesses in MN of the stomach, esophagus, colon, rectum, larynx, and buccal cavity and pharynx.[18–20] Carbon tetrachloride is an animal carcinogen; no studies of workers exposed to carbon tetrachloride in the absence of other potentially carcinogenic solvents have been conducted.[21] PCBs induce benign and malignant neoplasms in mice and rats after their oral administration[11,13,14] and have limited evidence for carcinogenicity in humans.[21]

The plant under study produced cable for use in naval vessels during World War II. As with other wartime manufacturing industries, the shortage of healthy young men and the need for substantially increased production resulted in the hiring of a large number of women workers to do traditionally male jobs. Thus, this cohort, which was exposed to several potentially carcinogenic substances, offers an opportunity to compare the cancer mortality experience of male and female workers.

Background

The plant, located in Westchester County, New York, used chlorinated naphthalenes (sold under the trade name Halowax) to insulate electrical cable from 1939 to 1944. The production areas and equipment used in the impregnation of cables with chlorinated naphthalenes no longer existed in the 1980s when the cohort was identified by the National Institute for Occupational Safety and Health. Although information about the process was limited, historical records document that chlorinated naphthalenes were melted in open vats through which wires coated with asbestos were drawn to saturate them.

In the early 1940s, numerous cases of chloracne were recognized and documented in worker compensation claims. In January 1943, a series of industrial hygiene studies was initiated in an attempt to contain the outbreak. Industrial hygiene reports contain measurements of air concentrations of chloride ion at approximately 20 to 30 in-plant sampling locations. These reports document that exposure was not confined to areas of the plant where chlorinated naphthalenes were used. For example, elevated levels were found in the dispensary and the engineering department. The industrial hygiene reports also mention the presence of Arochlor (chlorinated biphenyls) and carbon tetrachloride as possible explanations for high chloride ion concentrations in specific areas of the plant, but no information is available to determine the extent of worker exposure to these compounds. According to the industrial hygiene records, use of chlorinated naphthalenes was phased out in the summer of 1944.

We studied the cohort of workers employed from January 1, 1940, through December 31, 1944 (Halowax use was discontinued in 1945). Since the majority of the work force employed during World War II did not continue employment after the war ended (only 3254 persons remained employed after January 1, 1945), we did not evaluate processes and exposures at the plant from 1945 on.

Methods

Personnel and payroll records were used to identify individuals who worked at the plant from January 1, 1940, through December 31, 1944, and to code information necessary for vital status follow-up. Because personnel records did not contain detailed work history information, the information coded was limited to beginning and ending dates of employment.

Chloracne cases were identified from review of an archived set of plant medical records of approximately 3700 individuals who terminated employment before 1955. Seven hundred sixty records that pertained to occupational dermatitis were microfilmed. These consisted mainly of New York State worker's compensation claims. To be considered a case of probable chloracne, the medical record either had to state that the person had chloracne or include one of the following: (1) "fumes from Halowax caused skin to break out," (2) "waxy compound caused skin to break out," or (3) no notation of Halowax or chloracne but a description of comedones, pustules, and cystic lesions. Chloracne cases were identified independently by two sets of coders who reviewed the 760 medical records of dermatitis cases. Discrepancies between the two sets of coders were resolved by one of the authors (AS).

Vital status of all workers in the study was determined as of December 31, 1985. Person-years were ended at the date last observed if a study subject could not be followed through 1985. Follow-up was extended to December 31, 1989, for the chloracne subcohort to increase the statistical power of the analysis for this small subgroup. Sources used to determine vital status were the Social Security Administration, the Internal Revenue Service, US Post Office cards mailed to last known address, the Veterans Administration, the Health Care Financing Administration, and the National Death Index. The mortality experience of the cohort was analyzed using a modified life table system developed by the National Institute for Occupational Safety and Health.[22,23] Life table analyses were conducted for the total cohort and for men and women separately, using US mortality rates for the comparison as well as local county rates for some analyses. We present 95% confidence intervals and two-sided *P* values throughout this article. Confidence intervals and *P* values were calculated using an exact method

(if either the observed or expected frequency was less than 8) or an approximate method (if observed and expected frequencies were 8 or more).[24]

Results

A total of 9028 workers were included in the study (3042 women, 5986 men). Women workers tended to be younger when they started employment and included a higher proportion of individuals whose race was classified as "nonwhite" (Table 1). There were very few women employed at the plant before the United States entered World War II in December 1941 (Table 1). Women tended to have a shorter duration of employment (Table 1). There were 460 cases of chloracne; all but 6 with unknown dates of diagnosis occurred between January 1, 1940, and December 31, 1944; four hundred ten (89%) occurred in 1942 and 1943. The temporal distribution of chloracne cases among male and female employees was similar, but the "attack rate" (defined as total chloracne cases divided by total number of employees) was much higher among male workers (431/5986; 7.2%) than among female workers (29/3042; 1.0%).

In the mortality study of the overall cohort, women were more likely to be lost to follow-up (Table 1), due to invalid or unmatched Social Security numbers. A substantially lower proportion of women was deceased (Table 1).

The standardized mortality ratios (SMRs) for cancers are shown in Table 2. Among the cancers hypothesized a priori to be associated with chlorinated naphthalene exposure, both women and men had nonsignificant excesses in MN of the liver, men but not women had nonsignificant excesses in MN of the connective tissue, and both genders had decreased mortality from MN of the lymphatic and hematopoietic organs. There were several MNs for which a significantly increased risk was noted in the overall cohort for which men and women were strikingly concordant in their SMRs. These were MN of the rectum; the stomach; and the trachea, bronchus, and lung. Women workers had elevated (but nonsignificant) SMRs for both kidney and bladder cancer; the SMRs for men were lower, but still above 1.00 (Table 2). In addition, men but not women had a significantly elevated SMR for MN of the buccal cavity and pharynx (SMR = 1.87), in particular, MN of "other parts of the buccal cavity" (SMR = 2.70).

SMRs for breast cancer and cancer of the "female genital organs" were near 1.00. A nonsignificant elevation was seen in MN of "other female organs." Deaths from MN of the male genital organs were decreased (Table 2).

The mortality experience of the chloracne subcohort is shown in Table 2. Men and women are combined in this analysis because there were too few women (n = 29) to support an independent analysis. Cancer mortality in the chloracne subcohort was generally quite similar to that of men in the overall cohort. SMRs for MN of the esophagus and "benign and unspecified neoplasms" were significantly elevated in the chloracne subcohort and not elevated at all in the overall cohort.

We also calculated SMRs based on Westchester County rates (Table 3). The county rate adjustment substantially decreased the SMR for MN of the rectum and stomach for both women and men and the SMR for MN of the buccal cavity and pharynx among men, suggesting that the excesses observed for these cancers may have been due, in part, to nonoccupational factors.

TABLE 1
Comparison of Demographic Characteristics, Work History, and Vital Status of Female and Male Workers at a Cable Manufacturing Plant

Characteristic	Women	Men	Chloracne Subcohort
Age at starting employment (years)			
<25	1359 (44.7%)	1493 (24.9%)	176 (36.3%)
≥25, <35	899 (29.5%)	1907 (31.9%)	170 (37.0%)
≥35	784 (25.9%)	2586 (43.2%)	114 (24.8%)
Median	26	32	
Range	15–73	15–73	
Race			
White	2388 (78.5%)	5300 (88.5%)	407 (88.5%)
Nonwhite	654 (21.5%)	686 (11.5%)	53 (11.5%)
Year of first employment			
<1940	36 (1.2%)	528 (8.9%)	33 (7.2%)
1940	8 (0.3%)	279 (4.7%)	24 (5.2%)
1941	3 (0.1%)	966 (16.1%)	113 (24.6%)
1942	842 (27.7%)	1607 (26.8%)	204 (44.3%)
1943	1186 (38.9%)	1922 (32.1%)	84 (18.3%)
1944	967 (31.8%)	684 (11.4%)	2 (0.4%)
Duration of employment during period chlorinated naphthalenes used (1940–1944)			
<6 Months	1344 (44.2%)	2129 (35.6%)	51 (11.1%)
≥6 Months, <2 years	1239 (40.7%)	1952 (32.6%)	162 (35.2%)
≥2 Years	459 (15.1%)	1905 (31.8%)	247 (53.7%)
Results of vital status ascertainment through 12/31/85 for total cohort and 12/31/89 for chloracne subcohort			
Alive	1717 (56.4%)	1952 (32.6%)	166 (36.1%)
Unknown	467 (15.4%)	357 (6.0%)	20 (4.3%)
Deceased	858 (28.2%)	3677 (61.4%)	274 (59.6%)

TABLE 2
Mortality from Malignant Neoplasms for Female and Male Workers at a Cable Manufacturing Plant

Cause*	Women			Men			Chloracne Subcohort		
	Observed	SMR	95% Confidence Interval	Observed	SMR	95% Confidence Interval	Observed	SMR	95% Confidence Interval
MNs									
Buccal and pharynx	1	0.32	0.09–1.78	39	1.87	1.33–2.56	1	0.64	0.01–3.54
Lip	0	0.00	0.00–0.00	0	0.00	0.00–0.00	0	0.00	0.00–0.00
Tongue	0	0.00	0.00–0.00	10	1.98	0.95–3.65	0	0.00	0.00–0.00
Other parts of buccal cavity	0	0.00	0.00–0.00	15	2.70	1.51–4.45	0	0.00	0.00–0.00
Pharynx	1	0.75	0.02–4.17	14	1.47	0.80–2.47	1	1.35	0.03–7.47
Digestive organs	69	1.12	0.87–1.42	266	1.25	1.11–1.41	21	1.32	0.82–2.02
Esophagus	3	1.09	0.22–3.19	19	1.00	0.60–1.56	5	3.26	1.05–7.61
Stomach	12	1.41	0.73–2.47	64	1.42	1.09–1.81	6	2.04	0.74–4.45
Intestine (except rectum)	27	1.06	0.70–1.54	79	1.21	0.96–1.51	4	0.77	0.21–1.96
Rectum	12	2.06	1.06–3.61	46	2.04	1.49–2.72	2	1.33	0.16–4.82
Biliary passages, liver, gallbladder	7	1.45	0.58–2.99	16	1.24	0.71–2.01	1	1.01	0.02–5.58
Liver (not specified)	1	1.78	0.01–3.12	1	0.16	0.00–0.86	0	0.00	0.00–0.00
Pancreas	7	0.64	0.25–1.31	34	0.90	0.63–1.26	3	1.02	0.21–2.97
Peritoneum and unspecified digestive organs	0	0.00	0.00–0.00	7	1.95	0.78–4.02	0	0.00	0.00–0.00
Respiratory system	37	1.35	0.95–1.86	281	1.29	1.14–1.45	25	1.33	0.86–1.96
Larynx	2	2.77	0.33–10.0	12	1.16	0.60–2.02	0	0.00	0.00–0.00
Trachea, bronchus, and lung	35	1.34	0.93–1.86	268	1.31	1.15–1.47	25	1.40	0.90–2.06
Nasal and other parts of respiratory system	0	0.00	0.00–0.00	1	0.38	0.01–2.12	0	0.00	0.00–0.00
Breast	47	0.98	0.72–1.31	0	0.00	0.00–0.00	2	3.33	0.38–11.3
Female genital organs	41	1.00	0.72–1.35		NA		0	0.00	0.00–0.00
Cervix uteri	13	1.03	0.55–1.76		NA		0	0.00	0.00–0.00
Other and unspecified parts of uterus	13	1.12	0.59–1.91		NA		0	0.00	0.00–0.00
Ovary	11	0.71	0.35–1.26		NA		0	0.00	0.00–0.00
Other female organs	4	3.37	0.92–1.91		NA		0	0.00	0.00–0.00
Male genital organs			NA	61	0.91	0.70–1.17	6	1.17	0.43–2.54
Prostate			NA	58	0.90	0.69–1.17	6	1.22	0.45–2.66
Urinary organs	10	1.54	0.73–2.83	46	1.20	0.88–1.60	4	1.40	0.38–3.58
Kidney	5	1.53	0.50–3.58	19	1.23	0.74–1.92	1	0.80	0.02–4.43
Bladder and other urinary organs	5	1.54	0.50–3.60	27	1.18	0.77–1.71	3	1.88	0.39–5.48
Lymphatic and hematopoietic	9	0.50	0.23–0.95	47	0.82	0.60–1.09	1	0.21	0.00–1.16
Lymphosarcoma and reticulosarcoma	1	0.29	0.01–1.60	11	0.99	0.49–1.76	1	1.22	0.03–6.76
Hodgkin's disease	2	1.46	0.18–5.28	3	0.61	0.13–1.78	0	0.00	0.00–0.00
Leukemia and aleukemia	5	0.70	0.23–1.64	20	0.79	0.48–1.22	0	0.00	0.00–0.00
Other lymphatic or hematopoietic	1	0.17	0.00–0.92	13	0.83	0.44–1.42	0	0.00	0.00–0.00
Other sites	24	0.92	0.59–1.37	74	0.99	0.78–1.24	6	0.94	0.34–2.05
Connective tissue	1	0.99	0.02–5.48	4	1.78	0.48–4.55	1	4.82	0.12–26.8
Neoplasms of benign and unspecified nature	6	1.09	0.40–2.36	4	0.41	0.11–1.04	4	4.93	1.34–12.6
All other cancers	15	0.66	0.37–1.08	47	0.86	0.71–1.28	1	4.23	0.00–1.32
Certificates not obtained	24			179			4		
All cancers	238	1.03	0.90–1.17	814	1.18	1.10–1.26	66	1.17	0.90–1.49
All deaths	858	0.92	0.86–0.98	3677	1.04	1.00–1.07	274	1.03	0.91–1.16

* The International Classification of Diseases categories included in each "cause" are tabulated in Reference 24.

TABLE 3
Comparison of Standardized Mortality Ratios for Selected Causes for Male and Female Workers at a Cable Manufacturing Plant Based on Westchester County, New York, and US Referent Rates, 1960–1985*

Cause of Death	Women			Men		
	Observed	SMR (US Referent Rates)	SMR (Westchester County Referent Rates)	Observed	SMR (US Referent Rates)	SMR (Westchester County Referent Rates)
MNs of connective and soft tissue	1	1.09	0.79	4	1.90	1.94
MNs of liver	6	1.53	1.41	12	1.28	1.15
MNs of lymphatic and hematopoietic tissue	6	0.39†	0.36†	35	0.77	0.76
MNs of rectum	10	2.19†	1.49	29	1.86‡	1.29
MNs of stomach	10	1.51	1.14	43	1.51†	1.25
MNs of lung	35	1.41	1.26	230	1.30‡	1.34‡
MNs of buccal cavity and pharynx	1	0.36	0.29	30	1.90‡	1.59†
MNs of the breast	46	1.17	0.96	0	0.00	0.00
Pneumoconiosis and other respiratory diseases	11	0.97	1.10	56	0.93	1.27
All cancers	219	1.13	1.01	661	1.20‡	1.11‡

* Analyses were limited to 1960–1985, since county rates were not available in the National Institute for Occupational Safety and Health life table before then. To directly compare US and county-based analyses, this table presents standardized mortality ratios based on US rates for this restricted time period as well.
† $P < .05$.
‡ $P < .01$.

Table 4 summarizes the relationship between duration of employment and latency (time since first employment during the time interval when chlorinated naphthalenes were used). One notable pattern was seen for MN of the connective tissue, in which four of the five deaths were concentrated in workers with over 1 year of exposure and over 25 years of latency (SMR = 3.54; 95% CI = 0.97 to 9.07).

Table 5 compares temporal trends in cancer mortality between women and men. For women, the highest SMRs for all cancer mortality were seen during the 1970s and 1980s; for men, the highest SMRs were seen in the 1950s and 1960s. One death from mesothelioma occurred in the entire cohort (based both on the results of the SMR analysis and a review of all cancer-related death certificates) and no deaths were from asbestosis.

Discussion

This study involved a large and unique cohort in which a well-documented chloracne outbreak had occurred.[25] Several limitations in this study influence interpretation of the cancer mortality data. First, chlorinated naphthalenes were used at the plant for only a short time, so the maximum duration of exposure was approximately 5 years; moreover, 80% of the cohort was exposed for less than 2 years. A second limitation is that workers were exposed concomitantly to both asbestos and chlorinated naphthalenes, making it difficult to interpret the significant excesses in cancer sites that have been associated with asbestos in prior studies (primarily lung, but also gastrointestinal tract, cancer). Smoking may also be associated with the excesses in lung and buccal cavity cancer; however, there are no data to compare smoking habits in the cohort with the US population.

The hypothesis of strongest a priori interest was that chlorinated naphthalene exposure might be related to increased risk of soft tissue sarcoma. The SMR for MN of the connective tissue in the overall cohort was nonsignificantly elevated; four of the five deaths in this category occurred among workers with over 1 year of exposure and over 25 years of latency (SMR = 3.54; 95% CI = 0.97 to 9.07). In addition, four of the seven deaths coded under "MN of the peritoneum and retroperitoneum," in which there was an SMR of 1.95 for men, were identified as soft tissue sarcomas on the death certificates. Because of problems with identifying soft tissue sarcomas using the International Classification of Diseases system, as well as problems in the agreement rate of death certificates and hospital diagnoses for this cause of death,[9,26,27] the association between soft tissue sarcomas and chlorinated naphthalene exposure is difficult to test in a mortality study. The results of this study suggest an association, but do not conclusively demonstrate it.

Mortality patterns in the chloracne subcohort did not differ from mortality patterns in the cohort as a whole, with the exception of two relatively rare causes of death (MNs of the esophagus and "benign and unspecified neoplasms"), which were significantly elevated in the chloracne subcohort but not the cohort as a whole. In interpreting the findings in the chloracne subcohort, it is important to note that chloracne is not necessarily a marker of higher exposure.[28]

Despite the substantially lower success rate in vital status follow-up, and the much smaller total number of deaths for female compared with male workers, site-specific cancer mortality was generally concordant between

TABLE 4
Standardized Mortality Ratios for Selected Causes for Male and Female Workers, and the Chloracne Subcohort, at a Cable Manufacturing Plant by Duration of Exposure and Latency (Time Since First Exposure)

Cause of Death	Latency (years)	Duration of Exposure: Women <1 Year n	Women <1 Year SMR	Women ≥1 Year n	Women ≥1 Year SMR	Men <1 Year n	Men <1 Year SMR	Men ≥1 Year n	Men ≥1 Year SMR	Chloracne Subcohort <1 Year n	Chloracne Subcohort <1 Year SMR	Chloracne Subcohort ≥1 Year n	Chloracne Subcohort ≥1 Year SMR
MNs of connective and soft tissue	≤25	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00
	>25	0	0.00	1	3.28	1	1.58	3	3.66	1	32.0	0	0.00
MNs of liver	≤25	0	0.00	2	2.58	4	1.51	2	0.61	0	0.00	1	4.45
	>25	4	2.31	1	0.76	3	0.98	7	1.79	0	0.00	0	0.00
MNs of lymphatic and hematopoietic tissue	≤25	1	0.30	3	1.25	7	0.68	14	1.10	0	0.00	1	0.94
	>25	4	0.58	1	0.19	10	0.68	16	0.82	0	0.00	0	0.00
MNs of rectum	≤25	1	0.71	1	0.97	8	1.59	14	2.13*	0	0.00	2	4.66
	>25	5	2.62	5	3.44*	9	1.95	15	2.38†	0	0.00	0	0.00
MNs of stomach	≤25	1	0.46	2	1.34	13	1.13	24	1.65*	0	0.00	2	2.11
	>25	4	1.41	5	2.50	11	1.32	16	1.48	1	2.86	3	2.14
MNs of lung	≤25	1	0.42	1	0.62	46	1.58†	48	1.37*	1	1.56	5	1.89
	>25	1	1.61	12	1.32	77	1.24	97	1.23	4	1.37	15	1.29
MNs of other parts of buccal cavity	≤25	0	0.00	0	0.00	4	3.76*	6	4.55	0	0.00	0	0.00
	>25	0	0.00	0	0.00	2	1.44	3	1.68	0	0.00	0	0.00
MNs of breast	≤25	4	0.38	3	0.40	0	0.00	0	0.00	0	0.00	0	0.00
	>25	25	1.45	13	1.21	0	0.00	0	0.00	1	12.5	1	2.61
Pneumoconiosis and other respiratory diseases	≤25	1	0.94	0	0.00	8	1.37	11	1.55	0	0.00	8	1.68
	>25	3	0.50	8	1.78	24	1.02	26	0.85	1	1.92	0	0.00
All cancers	≤25	23	0.49	23	0.70	137	1.16†	184	1.26†	1	0.38	16	1.48
	>25	121	1.38*	71	1.12	201	1.09	292	1.22	9	1.06	40	1.16

*$P < .05$.
†$P < .01$.

TABLE 5
Standardized Mortality Ratios for Mortality from All Cancers by Calendar Time for Female and Male Workers at a Cable Manufacturing Plant

Time Interval of Follow-up	Women Observed	Women SMRs	Men Observed	Men SMRs
1940–1949	6	0.61	24	0.66
1950–1959	13	0.46	99	1.27
1960–1969	31	0.60	223	1.29
1970–1979	104	1.31	173	1.20
1980–1989	84	1.36	165	1.10

men and women (cancer of the buccal cavity and pharynx being the only substantial exception). The difficulty in follow-up of female workers was largely due to problems with their Social Security numbers, which may be related to the cohort's restriction to the time period 1940 through 1945, when the system was in its infancy.

Both data on percentage of workers who are deceased and temporal trends in SMRs for all cancers suggest that termination of mortality follow-up in 1985, when over 50% of the overall cohort was deceased, was premature for the female workers. A much smaller proportion of women than men had died at the end of follow-up because female workers were younger at the time of hire and have lower age-specific death rates. While cancer mortality peaked for men in the 1950s and 1960s, the highest SMRs for women are seen in the 1970s and 1980s. In summary, it is difficult to draw definitive conclusions about the carcinogenicity of chlorinated naphthalenes based on this study because of several limitations, most importantly, the concomitant exposure of workers to asbestos and the relatively short duration of exposure to chlorinated naphthalenes of most of the cohort. The importance of the nonsignificant excess in soft tissue sarcomas, which has been linked with other chloracnegenic compounds but not with asbestos, may be clarified during continued follow-up of this cohort.

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