Exhibit 235

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An updated cohort mortality study of workers at a northeastern United States petroleum refinery

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Abstract An update of a cohort study of 4855 employees at a Paulsboro, New Jersey refinery was conducted to further examine mortality patterns. The earlier study investigated refinery workers employed for a minimum of 1 year between 1 January 1946 and 1 January 1979. The vital status of these workers was ascertained through 1979. The update extended enrollment in the study and vital status follow-up for an additional 8 years (1980–1987). As in the previous study, mortality from all causes [standardized mortality ratio (SMR) = 87; 95% confidence interval (95% CI): 83–91] was significantly lower than expected compared with the general population. Total cancer mortality was also lower than expected (SMR = 96; 95% CI: 86–106). A borderline significant mortality increase in prostatic cancer was found (SMR = 144; 95% CI: 106–190). This increase was similar to the nonsignificant increase reported in the original study (SMR = 135; 95% CI: 90–196). The excess was of comparable magnitude among white males and nonwhite males, although it was not significant for the latter. Detailed analysis indicated that the prostatic cancer was not likely to be related to employment at the refinery. Mortality from lymphatic and hematopoietic cancers was similar to the expected mortality. Mortality from overall leukemia was as expected and detailed analyses by specific cell type showed no increase. An increase in mortality occurred from non-Hodgkin's lymphoma among male workers (SMR = 132; 95% CI: 74–217). The increase was not statistically significant and unlikely to be associated with refinery employment. Mortality from multiple myeloma among male employees was lower than expected (SMR = 74; 95% CI: 20–190). Mortality from asbestos-related diseases (pulmonary fibrosis, lung cancer, malignant mesothelioma) was also lower than expected among male workers. No cause-specific mortality was found to be associated with duration of employment at the refinery, including several causes which have been reported to be elevated in previous studies. The findings of this updated study indicate, as in the previous report, the generally favorable mortality experience of Paulsboro refinery workers.

Key words Petroleum refinery workers · Cohort studies · Occupational mortality

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Introduction

This study is a further investigation of the mortality experience of workers at a refinery in Paulsboro, New Jersey. The refinery began operations in 1917 and was designed to manufacture quality lubricants. During the 1920s and 1930s the refinery pioneered innovative lube oil techniques including two-state fractionation and continuous solvent treatment. During the mid-1930s the Paulsboro refinery became the site of the first commercial catalytic cracking unit. Currently, the refinery produces gasoline, aviation fuel, diesel fuel, and a full line of lubrication oil products.

In 1985, a previous retrospective cohort mortality study of workers at a Paulsboro, New Jersey refinery was conducted on workers employed for a minimum of 1 year between 1 January 1946 and 1 January 1979 (Morgan and Wong 1985). The vital status of these workers was ascertained through 31 December 1979. Based on comparison with national mortality rates,

workers at the Paulsboro refinery experienced at 17% deficit in overall mortality and an 8% deficit in total cancer mortality, and no significant excess cause-specific mortality. Analyses of cause-specific mortality by length of employment and time since first employment showed a significant excess of prostatic cancer deaths among white males employed for 20 or more years. Additional analyses based on work history data did not identify occupational agent(s) or condition(s) responsible for the excess.

To continue surveillance of the mortality experience of the Paulsboro refinery workers, enrollment in the study was extended over an additional 8 years of employment eligibility (1980–1987). Vital status follow-up was extended from 31 December 1979 to 31 December 1987. The objective of the update was to expand the previous study, and to continue monitoring the mortality patterns of the Paulsboro refinery workers.

Materials and methods

The cohort consisted of all employees who worked for a year or more at the Paulsboro refinery between 1 January 1946 and 31 December 1987. Cohort members were identified through a combination of personnel records located on-site and computerized payroll files. Information abstracted from these records included social security number, name, sex, race, date of birth, date of employment, employment status at the closing date of the study, vital status at the closing date of the study, and date of retirement, separation, or death when applicable. Employment histories of cohort members were also obtained from personnel records. Work history data consisted of the beginning and ending dates for each job, job title, and department.

Several sources were used for vital status ascertainment, including the company's personnel database, Pension Benefits Information, Inc., the National Death Index, and the Social Security Administration. Death certificates were obtained from company personnel files or from state vital records offices. A qualified nosologist coded the underlying cause of death according to the Eighth Revision of the International Classification of Diseases (ICD) (World Health Organization 1967, 1968).

Overall and cause-specific mortality was analyzed with OCMAP, a modified life-table computer program that calculates cause-specific standardized mortality ratios (SMRs), the ratio of observed to expected deaths expressed as a percentage (Marsh and Preininger 1980). Expected deaths were derived from mortality rates for the U.S. population, which are specific for cause, sex, race, and 5-year intervals of age and calendar period (Monson 1987). OCMAP also computed cause-specific mortality relative to cumulative duration of employment and time since first employment.

Entry into vital status follow-up was 1 January 1947 for workers with at least 1 year of full-time employment prior to that date or, for all others, 1 day after completion of 1 year of employment. SMRs for specific death categories were examined relative to time since first employment (<20, 20–29, 30+ years) and by duration of employment (<10, 10–29, 30+ years). These cutoff points were those used in the previous investigation. The method proposed by Breslow et al. (1983) was used to test for linear trends in SMRs.

In addition, because of the a priori interest in lymphatic and hematopoietic cancers, SMRs for major cell type-specific leukemias (acute myelogenous, chronic myelogenous, acute lymphatic, and chronic lymphatic leukemia, or AML, CML, ALL, and CLL), non-Hodgkin's lymphoma (NHL) and multiple myeloma (MM), which were not calculated by the standard rate sets supplied to the OCMAP program, were also performed. U.S. age-specific mortality rates for cell type-specific leukemias and similar rates for non-Hodgkin's lymphoma and multiple myeloma compiled by the National Cancer Institute were used in computing the expected deaths from these causes (Selvin et al. 1983; Pickle et al. 1987). In addition, separate analyses for asbestos-related diseases were also performed. Expected deaths were based on U.S. rates for pulmonary fibrosis obtained from the National Center for Health Statistics (unpublished data by special request) and for malignant mesothelioma (Connelly et al. 1987).

Results

Descriptive statistics

A total of 4855 workers were eligible for inclusion in the updated study (Table 1). This represented an increase of 475 whites (81% male), 97 nonwhites (74% male), and 20 of unknown race (95% male) over the original cohort. Consistent with the previous report, those of unknown race (approximately 3.5% of the total cohort) were treated as white. This resulted in 91% of cohort members being male and white (including Hispanic).

Selected employment and demographic characteristics of the cohort are also provided in Table 1. Seventy-four percent were first employed in their twenties or thirties, with only 8% hired at age 40 or older. Slightly more than half (56%) were hired prior to 1950, and 35% were employed for 30 or more years.

The maximum length of potential follow-up was 41 years (1 January 1947 to 31 December 1987). The

Table 1 Characteristics of the Paulsboro, New Jersey Cohort

	No.	% Total
Total employees	4 855	100.0
Total person-years	120 718	100.0
Race		
White	4 265	87.8
Nonwhite	421	8.7
Unknown	169	3.5
Sex		
Male	4 433	91.3
Female	422	8.7
Year of hire		
Before 1950	2 706	55.7
1950+	2 149	44.3
Duration of employment (yr)		
<10	1 591	32.8
10–29	1 550	31.9
30+	1 714	35.3
Age at hire		
<20	858	17.7
20–39	3 587	73.9
40+	410	8.4
Age at death		
<50	129	7.7
50–59	260	15.5
60–69	518	30.8
70+	774	46.0
Total deaths	1 681	100.0

average duration of follow-up was 25 years. Approximately 60% of cohort members had a follow-up of 20 years or more. The increase in person-years from the previous study was 27 284, or 29% to a total of 120 718.

By the end of follow-up (31 December 1987), 21% of cohort members were still employed, 44% had separated or retired, and 35% were known to be dead. Information on vital status was 98% complete, with 81 (2%) separated employees lost to follow-up. Of the 1681 cohort members identified as having died between 1946 and 1987, death certificates were obtained for 98%. Sixty-eight percent of all deaths occurred among retirees. The average age at death was 68 years, with the highest percentage of deaths (30%) occurring since 1980.

Overall and cause-specific mortality

The number of observed deaths, SMRs, and 95% confidence interval (95% CI) for selected causes of death for all workers are presented in Table 2. The total number

Table 2 Observed and expected deaths, SMRs and 95% confidence limits by cause for the Paulsboro refinery cohort. Number of persons: 4855; person-years: 120 718

Cause of death (8th ICD)	Observed deaths	Expected deaths	SMR	95% confidence limits	
				Lower	Upper
All causes (001–999)	1681	1941.6	87**	83	91
Infective and parasitic diseases (001–139)	15	29.8	50**	28	83
All cancers (140–209)	382	399.7	96	86	106
Cancer of buccal cavity and pharynx (140–149)	8	11.5	69	30	137
Cancer of digestive system (150–159)	117	114.7	102	84	122
Cancer of esophagus (150)	10	10.3	97	47	178
Cancer of stomach (151)	16	22.1	72	41	118
Cancer of large intestine (153)	45	37.3	121	88	162
Cancer of rectum (154)	14	11.8	118	65	199
Cancer of liver (155–156)	3	9.2	33*	7	95
Cancer of pancreas (157)	26	21.2	123	80	180
Cancer of respiratory system (160–163)	105	129.1	81*	67	99
Cancer of larynx (161)	5	5.6	89	29	207
Cancer of lung (162–163)	99	122.2	81*	66	99
Cancer of bone (170)	1	1.6	62	2	348
Cancer of skin (172–173)	5	6.3	80	26	186
Cancer of breast (174)	3	3.1	96	20	282
Cancer of prostate (185)	49	34.1	144*	106	190
Cancer of testis (186–187)	1	1.6	64	2	358
Cancer of bladder (188)	10	12.4	81	39	148
Cancer of kidney (189)	4	9.1	44	12	112
Cancer of brain and CNS (191–192)	9	9.4	96	44	183
Lymphatic and hematopoietic cancer (200–209)	38	36.0	106	75	145
Lymphosarcoma and reticulosarcoma (200)	3	6.6	46	9	134
Hodgkin's disease (201)	2	3.4	59	7	214
Leukemia and aleukemia (204–207)	15	14.9	101	56	166
Other lymphatic tissue cancer (202, 203, 208)	18	10.9	165	98	260
Benign neoplasms (210–239)	6	5.0	119	44	259
Diabetes mellitus (250)	31	28.6	108	74	154
Diseases of blood (280–289)	9	4.8	187	85	354
Diseases of nervous system (320–389)	6	17.6	34**	13	74
Diseases of circulatory system (390–458)	941	1021.8	92*	86	98
Chronic rheumatic heart disease (393–398)	11	15.1	73	36	131
Arteriosclerotic heart disease (410–413)	672	677.9	99	92	107
Vascular lesions of CNS (430–438)	115	148.4	78**	64	93
Nonmalignant respiratory disease (460–519)	69	129.1	53**	42	68
Pneumonia (480–486)	23	48.9	47**	30	71
Emphysema (492)	10	25.2	40**	19	73
Diseases of digestive system (520–577)	64	81.3	79	61	101
Cirrhosis of liver (571)	29	38.2	76	51	109
Diseases of genitourinary system (580–629)	17	32.7	52**	30	83
Senility and ill-defined conditions (780–799)	13	22.4	58**	31	99
Accidents, Poisonings, and violence (800–998)	74	141.1	52**	41	66
Accidents (800–949)	49	94.3	52**	39	69
Motor vehicle accidents (810–827)	28	41.2	68*	45	98
Suicide (950–959)	23	31.3	73	47	110

* Statistically significant at 0.05 level
** Statistically significant at 0.01 level

of observed deaths was 1681 compared with 1941.6 expected (SMR = 87; 95% CI: 83–91), indicating a statistically significant deficit of 13% in overall mortality. Mortality from all cancers was slightly lower than expected. The total number of observed cancer deaths was 382, versus 399.7 expected deaths (SMR = 96; 95% CI: 86–106), and the deficit was not statistically significant.

Significant deficits in cause-specific mortality occurred for infective and parasitic diseases, liver cancer, lung cancer, diseases of the nervous system, circulatory system diseases, genitourinary system diseases, nonmalignant respiratory diseases, senility and ill-defined conditions, and accidents, poisonings, and violence. The only statistically significant excess in cause-specific mortality occurred for prostatic cancer (SMR = 144; 95% CI: 106–190).

The results for white males (Table 3), comprising 83% of the cohort, showed few differences from those for the total cohort. The only statistically significant

Table 3 Observed and expected deaths, SMRs and 95% confidence limits by cause for white male employees in the Paulsboro refinery cohort. Number of persons: 4049; person-years: 103 048

Cause of death (8th ICD)	Observed deaths	Expected deaths	SMR	95% confidence limits	
				Lower	Upper
All causes (001–999)	1499	1720.5	87**	83	92
Infective and parasitic diseases (001–139)	14	23.5	60*	33	100
All cancers (140–209)	335	352.5	95	85	106
Cancer of buccal cavity and pharynx (140–149)	6	10.2	59	22	128
Cancer of digestive system (150–159)	101	100.9	100	82	122
Cancer of esophagus (150)	9	8.3	109	50	207
Cancer of stomach (151)	14	19.0	74	40	124
Cancer of large intestine (153)	39	33.6	116	83	159
Cancer of rectum (154)	12	10.8	111	57	194
Cancer of liver (155–156)	3	7.9	38	8	111
Cancer of pancreas (157)	21	18.9	111	69	170
Cancer of respiratory system (160–163)	95	116.5	82*	66	100
Cancer of larynx (161)	5	5.0	100	32	232
Cancer of lung (162–163)	89	110.4	81*	65	99
Cancer of bone (170)	1	1.4	70	2	387
Cancer of skin (172–173)	5	5.9	85	28	198
Cancer of prostate (185)	43	30.0	144*	104	193
Cancer of testis (186–187)	1	1.4	70	2	391
Cancer of bladder (188)	9	11.6	78	36	148
Cancer of kidney (189)	4	8.4	48	13	122
Cancer of brain and CNS (191–192)	9	8.6	104	48	198
Lymphatic and hematopoietic cancer (200–209)	33	32.6	101	70	142
Lymphosarcoma and reticulosarcoma (200)	2	6.0	33	4	120
Hodgkin's disease (201)	2	3.1	65	8	235
Leukemia and aleukemia (204–207)	14	13.6	103	56	172
Other lymphatic tissue cancer (202, 203, 208)	15	9.6	156	87	257
Benign neoplasms (210–239)	6	4.4	137	50	298
Diabetes mellitus (250)	28	24.7	114	76	164
Diseases of blood (280–289)	7	4.2	166	67	343
Diseases of nervous system (320–389)	5	15.4	33**	11	76
Diseases of circulatory system (390–458)	850	920.0	92*	86	99
Chronic rheumatic heart disease (393–398)	10	13.4	75	36	137
Arteriosclerotic heart disease (410–413)	616	626.6	98	91	106
Vascular lesions of CNS (430–438)	103	126.9	81*	66	99
Nonmalignant respiratory disease (460–519)	65	116.3	56**	43	71
Pneumonia (480–486)	22	42.4	52**	33	79
Emphysema (492)	10	23.9	42**	20	77
Diseases of digestive system (520–577)	56	71.9	78*	59	101
Cirrhosis of liver (571)	26	33.8	77	50	113
Diseases of genitourinary system (580–629)	12	26.7	45**	23	79
Senility and ill-defined conditions (780–799)	11	17.1	64	32	115
Accidents, Poisonings, and violence (800–998)	64	121.0	53**	41	68
Accidents (800–949)	42	82.3	51**	37	69
Motor vehicle accidents (810–827)	22	36.2	61*	38	92
Suicide (950–959)	21	29.5	71	44	109

* Statistically significant at 0.05 level
** Statistically significant at 0.01 level

excess was found for prostatic cancer (SMR = 144). Unlike the total cohort, the liver cancer deficit among white males was not significant.

A total of 36 deaths were observed among 422 female cohort members (white and nonwhite), compared with 48.0 expected (not shown). The overall SMR of 75 was not significant and was mainly attributable to fewer than expected deaths from circulatory system diseases (SMR = 67). Mortality from all cancers was slightly lower than expected (SMR = 87) based on only 12 observed deaths. There were no statistically significant findings for any cause of death among females.

A total of 146 deaths were observed among 384 nonwhite males compared with 173.1 expected (not shown). The corresponding SMR of 84 was significant and reflected deficits for respiratory system cancers and diseases of the respiratory and digestive systems, and accidents, poisonings, and violence. The deficits for respiratory system diseases and for accidents, poisonings, and violence were significant, as was found among white males. Also, mortality from motor vehicle accidents was slightly elevated (SMR = 126), in contrast to the significant deficit found for white males. No statistically significant excess was found, although nonsignificant elevations based on small numbers of deaths were shown for several other cancer sites, including prostate, pancreas, and other lymphatic tissue.

Mortality by duration of employment

Table 4 shows cause-specific SMRs by duration of employment among white male employees. Only two intervals of duration of employment (<20 and 20+ years) were used in the original report. In the present update, since there were more deaths, three intervals were used (<20, 20–29, 30+ years). The results indicated that the SMRs for overall mortality increased slightly with increasing employment duration, ranging from 74 among workers with <20 years of employment to 93 for those with 30+ years. Although a formal test indicated that the trend was statistically significant ($\chi^2_{1\,df} = 10.09$), all three intervals showed a deficit in overall mortality. The upward trend in overall mortality was due largely to the significant upward trend in mortality from circulatory diseases (SMRs rising from 77 to 100, $\chi^2_{1\,df} = 7.89$). On the other hand, SMRs for brain cancer showed a negative trend by duration of employment, which was statistically significant ($\chi^2_{1\,df} = 4.42$). No significant trends were detected for any other cause of death among white males. In particular, no upward trend in mortality was detected for prostatic cancer ($\chi^2_{1\,df} = 0.82$), all lymphopoietic cancers ($\chi^2_{1\,df} = 3.37$), leukemia ($\chi^2_{1\,df} = 1.42$), or other lymphatic tissue cancer ($\chi^2_{1\,df} = 1.84$). Elevated SMRs for prostatic cancer (SMR = 154; 95% CI: 102–222) and other lymphatic tissue cancer (SMR = 208; 95% CI: 104–371) were found among white males with 30+ years of employment. Both elevations were borderline significant.

For nonwhite males (not shown), overall mortality rose with employment duration, and the trend was statistically significant ($\chi^2_{1\,df} = 10.83$). This upward trend was largely due to a nonsignificant upward trend for diseases of the circulatory system. No other significant trends were detected for nonwhite males. For prostatic cancer, there appeared to be a negative trend by duration of employment among nonwhite males (SMR = 300, 141, and 125), but the trend was not statistically significant ($\chi^2_{1\,df} = 0.41$).

Mortality analysis by duration of employment for both white and nonwhite males was similar to that reported for white males in Table 3. No analysis by duration of employment was performed for white female employees, since the number of deaths was small.

Mortality by interval since first employment

Mortality analysis for white males by interval since first employment (<20, 20–29, 30+ years) is shown in Table 5. Mortality from all causes was marked by increasing SMRs with increasing interval since first employment ($\chi^2_{1\,df} = 27.24$). The upward trend in overall mortality was primarily due to the significant upward trend in diseases of the circulatory system ($\chi^2_{1\,df} = 5.94$). In addition, there were significant upward trends for rectal cancer, pancreatic cancer, and diabetes. However, the upward trends were created primarily by the absence or small number of deaths in the <20 or 20–29 year intervals. In Table 5, two SMRs for the 30+ year interval were borderline significant; for prostatic cancer SMR = 148 (95% CI: 105–202), and for other lymphatic tissue cancers SMR = 185 (95% CI: 101–311). On the other hand, a number of causes of death in Table 5 showed significant mortality deficits (e.g., cancer of the digestive system, diseases of the circulatory system, diseases of the respiratory system, and external causes).

For nonwhite males (not shown), mortality in relation to interval since first employment showed a pattern similar to that among white males. However, because of much smaller numbers, only overall mortality showed a statistically significant upward trend. Mortality excess from arteriosclerotic heart disease was borderline significant among nonwhite males in the category of 30 or more years since hire (SMR = 141; 95% CI: 101–193). No other causes of death showed significantly elevated SMRs.

Mortality analysis by interval since first employment for both white and nonwhite males was similar to that reported for white males in Table 5. In particular, mortality increases from prostatic cancer (SMR = 150; 95% CI: 109–200) and other lymphatic tissue cancer (SMR = 207; 95% CI: 120–331) were statistically

Table 4 Observed deaths (*Obs.*) SMRs, and 95% confidence limits for selected causes of death among white male employees in the Paulsboro refinery cohort by length of employment

Cause of death (8th ICD)	Length of employment											
	<20 years				20–29 years				30+ years			
	Obs.	SMR	Lower	Upper	Obs.	SMR	Lower	Upper	Obs.	SMR	Lower	Upper
All causes of death	307	74**	66	83	390	89*	80	98	802	93*	86	99
All malignant neoplasms	68	82	64	105	83	97	77	120	184	100	86	115
Cancer of digestive organs and peritoneum	16	73	42	118	30	114	77	163	55	104	79	136
Cancer of esophagus	5	264	86	617	1	48	1	269	3	70	14	204
Cancer of stomach	1	25	1	138	6	105	39	231	7	75	30	155
Cancer of large intestine	7	97	39	201	8	98	42	193	24	132	84	196
Cancer of rectum	0	0	–	–	7	232	93	478	5	91	30	212
All cancer of liver	0	0	–	–	0	0	–	–	3	73	15	213
Cancer of pancreas	3	71	15	208	5	107	35	250	13	131	70	223
Cancer of respiratory system	26	93	61	137	22	82	52	125	47	76	56	101
Cancer of larynx	2	174	21	629	1	78	2	432	2	78	9	280
All cancer of lung – primary and secondary	24	91	58	135	21	84	52	128	44	75	54	100
Cancer of skin	1	51	1	286	0	0	–	–	4	156	43	400
Cancer of prostate	4	88	24	224	11	153	77	274	28	154*	102	222
Cancer of bladder	2	97	12	352	1	34	1	192	6	91	33	198
Cancer of kidney	0	0	–	–	3	145	30	423	1	24	1	132
Cancer of brain and other CNS	6	197	72	430	2	96	12	347	1	29	1	159
Lymphosarcoma and reticulosarcoma	0	0	–	–	0	0	–	–	2	71	9	257
Hodgkin's disease	2	148	18	534	0	0	–	–	0	0	–	–
Leukemia and aleukemia	2	56	7	201	3	92	19	269	9	133	61	252
Cancer of other lymphatic tissue	2	86	10	309	2	100	12	361	11	208*	104	371
All lymphopoietic cancer	6	67	24	145	5	66	21	154	22	138	86	209
Benign neoplasms	1	76	2	425	0	0	–	–	5	253	82	589
Diabetes mellitus	4	70	19	180	7	112	45	230	17	134	78	215
All diseases of blood and blood-forming organs	1	97	2	543	3	266	55	778	3	146	30	427
All diseases of circulatory system	147	77**	65	91	219	90	78	102	484	100	91	109
Arteriosclerotic heart disease, including CHD	107	81*	66	98	162	100	85	117	347	105	94	116
All vascular lesions of CNS	16	71	41	116	31	88	60	125	56	81	61	105
All respiratory diseases	8	35**	15	70	26	95	62	139	31	47**	32	66
All pneumonia	2	24*	3	86	9	84	38	159	11	47**	24	85
Emphysema	2	48	6	173	5	91	29	211	3	21**	4	62
All diseases of digestive system	17	82	48	131	14	71	39	119	25	80	52	118
Cirrhosis of liver	8	70	30	139	4	44	12	112	14	106	58	178
All external causes of death	31	52**	35	74	15	55*	31	90	18	53**	31	84
All accidents	18	45**	26	70	10	54*	26	99	14	60	33	101
Motor vehicle accidents	13	64	34	110	3	40	8	118	6	71	26	154
Suicide	12	89	46	115	5	71	23	165	4	45	12	115
Number of persons at risk	3 420				2 075				1 544			
Number of person-years	57 119				21 469				24 461			

* Statistically significant at 0.05 level
** Statistically significant at 0.01 level

significant in white and nonwhite males in the category of 30 or more years since hire. On the other hand, in the same group significant deficits were reported for a number of causes of death, including lung cancer, respiratory diseases, and external causes. As stated above, the number of deaths among white female workers was too small for any subcohort analysis. No analysis by interval since first employment was performed for white female employees.

Mortality from lymphatic and hematopoietic cancers

Analysis of mortality from lymphatic and hematopoietic cancers by year of first employment has been found to be informative by other studies of petroleum workers. In 1947, the recommended standard for benzene was reduced from 100 ppm to 50 ppm, and it was further reduced to 35 ppm in 1948. In general, benzene exposure levels in the petroleum industry after

Table 5 Observed deaths (*Obs.*), SMRs, and 95% confidence limits for selected causes of death among white male employees in the Paulsboro refinery cohort by time since first employment

Cause of death (8th ICD)	Time since first employment											
	<20 years				20–29 years				30+ years			
			95% confidence limits				95% confidence limits				95% confidence limits	
	Obs.	SMR	Lower	Upper	Obs.	SMR	Lower	Upper	Obs.	SMR	Lower	Upper
All causes of death	122	64**	53	76	204	71**	61	81	1 173	95	89	100
All malignant neoplasms	24	75	48	111	40	69*	49	94	271	103	91	116
Cancer of digestive organs and peritoneum	5	58	19	134	8	47*	20	92	88	117	94	144
Cancer of esophagus	2	294	36	1063	2	136	17	493	5	82	27	191
Cancer of stomach	0	0	–	–	1	27	1	150	13	97	52	166
Cancer of large intestine	3	117	24	343	4	80	22	204	32	123	84	174
Cancer of rectum	0	0	–	–	0	0	–	–	12	154	79	268
All cancer of liver	0	0	–	–	0	0	–	–	3	51	11	150
Cancer of pancreas	0	0	–	–	1	31	1	175	20	142	87	219
Cancer of respiratory system	8	84	36	165	18	91	54	144	69	79	62	100
Cancer of larynx	1	227	6	1265	1	105	3	584	3	83	17	242
All cancer of lung – primary and secondary	7	78	31	161	17	92	53	147	65	79	61	100
Cancer of skin	0	0	–	–	0	0	–	–	5	133	43	311
Cancer of prostate (males only)	1	112	3	625	3	112	23	328	39	148*	105	202
Cancer of bladder	0	0	–	–	0	0	–	–	9	96	44	183
Cancer of kidney	0	0	–	–	2	127	15	459	2	33	4	120
Cancer of brain and other CNS	3	176	36	514	3	152	31	444	3	61	13	178
Lymphosarcoma and reticulosarcoma	0	0	–	–	0	0	–	–	2	51	6	185
Hodgkin's disease	2	198	24	716	0	0	–	–	0	0	–	–
Leukemia and aleukemia	0	0	–	–	2	92	11	331	12	124	64	217
Cancer of other lymphatic tissue	1	131	3	732	0	0	–	–	14	185*	101	311
All lymphopoietic cancer	3	67	14	195	2	37	4	133	28	124	82	179
Benign neoplasms	0	0	–	–	1	119	3	661	5	179	58	417
Diabetes mellitus	0	0	–	–	3	75	15	218	25	138	89	204
All diseases of blood and blood-forming organs	1	190	5	1058	1	138	4	770	5	169	55	395
All diseases of circulatory system	60	79	61	102	112	76**	63	91	678	97	90	105
Arteriosclerotic heart disease, including CHD	45	87	63	116	89	87	70	107	482	102	93	112
All vascular lesions of CNS	5	62	20	145	13	76	41	130	85	84	67	103
All respiratory diseases	2	26*	3	93	5	34**	11	80	58	62**	47	80
All pneumonia	0	0	–	–	1	19	1	104	21	62*	39	95
Emphysema	1	75	2	415	1	32	1	176	8	41**	18	81
All diseases of digestive system	3	28*	6	81	13	79	42	135	40	90	64	122
Cirrhosis of liver	0	0**	–	–	6	67	24	145	20	106	65	164
All external causes of death	23	52**	33	78	11	41**	20	73	30	61**	41	87
All accidents	14	45**	25	76	7	39**	16	81	21	62*	39	95
Motor vehicle accidents	10	61	29	113	4	52	14	134	8	65	28	129
Suicide	8	84	36	166	4	56	15	143	9	70	32	133
Number of persons at risk	3 317				2 854				2 593			
Number of person-years	43 391				23 896				35 762			

* Statistically significant at 0.05 level
** Statistically significant at 0.01 level

1948–1950 were much lower than before. Table 6 shows mortality from lymphatic and hematopoietic cancers by year of first employment (<1950, 1950+). The SMR for other lymphatic tissue cancer among all male workers was elevated (SMR = 171; 95% CI: 101–270) with borderline significance. This increase was confined to workers hired before 1950 (SMR = 198; 95% CI: 117–313). In the same group of male workers, mortality from leukemia was slightly elevated (SMR = 118; 95% CI: 66–195), whereas a nonsignificant deficit was observed for lymphosarcoma and reticulosarcoma (SMR = 36; 95% CI: 4–129) based on only 2 observed deaths. There were 1834 male workers hired in or after 1950, and only 144 deaths occurred among them. No

Table 6 Observed and expected deaths, SMRs, and 95% CIs for lymphatic and hematopoietic cancers among all males by year of first employment (<1950, 1950+)

Cause of death (8th ICD)	Year of first employment											
	<1950				1950+				Total			
	Obs.	Expected	SMR	(95% CI)	Obs.	Expected	SMR	(95% CI)	Obs.	Expected	SMR	(95% CI)
All lymphopoietic cancer	**37**	**30.3**	**122**	**(86–168)**	**0**	**4.5**	**0***	**(–)**	**37**	**34.8**	**106**	**(75–147)**
Lymphosarcoma and reticulosarcoma	2	5.6	36	(4–129)	0	0.8	0	(–)	2	6.3	32	(4–114)
Hodgkin's disease	2	2.7	74	(9–268)	0	0.6	0	(–)	2	3.3	61	(7–221)
Leukemia	15	12.7	118	(66–195)	0	1.7	0	(–)	15	14.4	104	(58–171)
Other lymphatic tissue	18	9.1	198*	(117–313)	0	1.5	0	(–)	18	10.5	171*	(101–270)

* Statistically significant at 0.05 level
** Statistically significant at 0.01 level

deaths from lymphopoietic cancers were reported, whereas 4.5 were expected (SMR = 0; 95% CI: 0–82), and the deficit was statistically significant.

Detailed work histories of leukemia and other lymphatic tissue cancer decedents were examined to determine whether there were common work assignments. A review of these records showed no evidence of any clustering of work assignments or locations.

The subcategories of lymphatic and hematopoietic cancers in the OCMAP program do not permit a specific analysis for major cell type-specific leukemias, NHL, or MM. Therefore, analyses for these specific subcategories of lymphatic and hematopoietic cancers among all male employees were carried out separately. Female cohort members were not included in these analyses since they contributed fewer than one expected death to these subcategories each. Table 7 shows that there were four observed AML deaths, compared with 4.2 expected (SMR = 95; 95% CI: 26–244). Nonsignificant deficits were observed for both CML and CLL. The SMRs were 53 (95% CI: 1–293) and 71 (95% CI: 9–258) for CML and CLL, respectively. For ALL, one death was observed, whereas 0.8 was expected (SMR = 125; 95% CI: 3–696).

For NHL among all males, the observed mortality was higher than expected (15 observed versus 11.4 expected deaths; SMR = 132; 95% CI: 74–217), but the increase was not statistically significant. For MM among all males, SMR was less than expected (4 observed versus 5.4 expected deaths; SMR = 74; 95% CI: 20–190), but the deficit was not statistically significant.

Table 7 Observed and expected deaths, SMRs, and 95% CIs for leukemia cell types, non-Hodgkin's lymphoma, and multiple myeloma among male employees (*ALL* acute lymphatic leukemia, *CLL* chronic lymphatic leukemia, *AML* acute myelogenous leukemia, *CML* chronic myelogenous leukemia, *NHL* non-Hodgkin's lymphoma, *MM* multiple myeloma)

Cause of death (ICD-8)	Observed deaths	Expected deaths	SMR	(95% CI)
ALL (204.0)	1	0.8	125	(3–696)
CLL (204.1)	2	2.8	71	(9–258)
AML (205.0)	4	4.2	95	(26–244)
CML (205.1)	1	1.9	53	(1–293)
NHL (200, 202)	15	11.4	132	(74–217)
MM (203)	4	5.4	74	(20–190)

Mortality from asbestos-related disease

Because of the use of asbestos at the refinery in the past, mortality from asbestos-related diseases (asbestosis, lung cancer, and malignant mesothelioma) among cohort members was examined. Although the OCMAP program provided an analysis for lung cancer, it did not analyze mortality data specifically for asbestosis or mesothelioma. Analyses for asbestosis (pulmonary fibrosis) and mesothelioma were performed separately.

The term asbestosis refers to pulmonary fibrosis caused by exposure to asbestos, or pulmonary fibrosis in persons with a documented asbestos exposure history. The ICD code (8th revision) for asbestosis is 515.2, and it is, therefore, part of the broad category "nonmalignant respiratory disease" (ICD 460–519). All death certificates within this broad category among Paulsboro refinery workers were reviewed, and no asbestosis death was found. However, since certifying physicians might not be aware of the decedents' asbestos exposure (if any), such deaths could have been coded simply as pulmonary fibrosis. Therefore, the category "pulmonary fibrosis" was analyzed. For pulmonary fibrosis (ICD 515–517), the number of expected deaths was 8.5, compared to 0 observed (Table 8). The corresponding SMR for pulmonary fibrosis was 0, which was statistically significant.

As stated above, lung cancer results were provided by the OCMAP program. The result for the overall cohort is presented in Table 2. Table 8 shows that the lung cancer SMR for all males was 81 (95% CI: 65–98), which was statistically significant.

Table 8 Observed and expected deaths, SMRs, and 95% CIs for asbestos-related diseases (pulmonary fibrosis, lung cancer, and malignant mesothelioma) among male employees

Cause of death (ICD-8)	Observed deaths	Expected deaths	SMR (95% CI)
Pulmonary fibrosis (515–517)	0	8.5	0** (–)
Lung cancer (162)	97	120.3	81* (65–98)
Malignant mesothelioma (for ICD codes, see text)	2	2.3	87 (11–314)

* Significant at 0.05 level
** Significant at 0.01 level

In the 8th revision of ICD, mesothelioma could be coded either as a malignant neoplasm of the peritoneum and retroperitoneum (ICD 158), a respiratory cancer (ICD 162 or 163), a malignant neoplasm of connective and other soft tissue (ICD 171), a malignant neoplasm with unspecified site (ICD 199), or a benign digestive system (ICD 211) or respiratory tumor (ICD 212, 215, or 228). Death certificates with the above ICD codes among Paulsboro refinery workers were reviewed to identify all malignant or benign mesotheliomas. Two deaths listed as "malignant mesothelioma" (ICD 162.1 and 199.1) were identified among male workers. Based on national mortality rates (Connelly et al. 1987), 2.3 deaths from malignant mesothelioma were expected (Table 8). The corresponding SMR for malignant mesothelioma among males was 87 (95% CI: 11–314).

Discussion

This study's results are consistent with the earlier investigation and other studies of refinery workers that have demonstrated a favorable overall mortality experience (Morgan and Wong 1985; Wong and Raabe 1989). Based on national rates, the updated SMR of 87 for all causes showed a significant deficit similar to that previously observed (SMR = 83; 95% CI: 79–88). The favorable mortality experience of these refinery workers might be attributed to the "healthy worker effect", whereby working populations exhibit decreased mortality due to initial selection into the workplace and maintenance of this healthier status through benefits derived from employment.

The updated SMR for all cancers of 96 for the total cohort was similar to that in the previous investigation (SMR = 92; 95% CI: 80–104); neither was statistically significant. Three cancer sites showed significant results in the update, compared to none in the original report. Two involved deficits: cancer of the respiratory system (SMR = 81), principally due to lung cancer (SMR = 81), and liver cancer (SMR = 33). The other significant cancer finding involved excess mortality from prostate cancer (SMR = 144).

The SMRs for all cancers among white males remained essentially unchanged (92 in the previous study versus 95 in the update). No cancer sites showed significant mortality deficits in the previous investigation, whereas the updated results showed a significant deficit for respiratory system cancer (SMR = 82), which was attributable to lung cancer (SMR = 81). The SMRs for all cancers for nonwhite males showed less stability due to small numbers of deaths (SMR = 94 in the previous report versus 105 in the update). Earlier findings for nonwhite males suggested elevated site-specific cancer mortality for buccal cavity and pharynx, rectum, pancreas, prostate, bladder, leukemia, and other lymphatic tissue, based on three or fewer observed deaths in each category. Results from the update showed that elevations in each of these categories remained, and the number of observed deaths involved in each category remained small as well.

Among females of both races (8828 person-years), the overall SMR and the SMR for all cancers increased about 20%. The total number of deaths was small (36 total in the update). Few specific causes of death had more than three observed deaths. Little interpretation could be made based on the statistical analyses among the females.

Epidemiologic studies of refinery workers have focused attention on a few suspect cancers: skin, brain or central nervous system, stomach, pancreas, kidney, bone, lung, colon, prostate, and lymphopoietic cancers (including leukemia). Updated results for Paulsboro refinery workers indicated that their mortality from stomach, skin, brain, pancreas, kidney, bone, colon, and lymphopoietic cancers (including leukemia) was similar to that in the general population. In addition, there was no relationship or pattern between mortality from these cancers and duration of employment or interval since hire. These findings from the updated Paulsboro refinery study were consistent with the conclusion regarding these cancer sites in a review by Wong and Raabe (1989).

With regard to prostatic cancer, a statistically significant increase was reported among white males (SMR = 144; 95% CI: 104–193), and a nonsignificant increase of the same magnitude was reported among nonwhite males (SMR = 144; 95% CI: 53–313). However, a formal trend analysis indicated there was no relationship between prostatic cancer and duration of employment among white males ($\chi^2_{1\,df} = 0.817$) or nonwhite males ($\chi^2_{1\,df} = 0.408$). This lack of relationship between prostatic cancer mortality and duration of employment argued against an interpretation that the statistical excess was related to employment at the refinery. Furthermore, a review of the detailed work histories of all male prostatic cancer deaths showed no evidence of common job assignments. Our interpretation that the prostatic cancer excess was not related to

refinery employment was consistent with the conclusion in the review by Wong and Raabe (1989), who reported a meta-SMR for prostatic cancer of 96 (95% CI: 87–105) based on 445 observed deaths from 12 studies. It is also consistent with findings of no excess (SMR = 91; 95% CI: 39–179) reported by Collingwood et al. (1991) among a subgroup of blending and packaging workers at this refinery.

A small amount of the prostate cancer excess may be explained by New Jersey state rates that average 4% and 8% higher than national rates for white and nonwhite males respectively. Besides regional differences in mortality, a number of risk factors for prostatic cancer have been suggested by previous epidemiologic studies, including occupation, diet, hormones, and sexual habits. Elevations in mortality for prostatic cancer have been found among cadmium workers studied in Britain, the United States, and Sweden (Workshop conference on the role of metals in carcinogenesis, 1981). An association between fat intake and prostatic cancer has been reported in several investigations (Heshmat et al. 1985; Kolonel et al. 1988; Mettlin et al. 1989): high consumption of both total and saturated fats has been linked to an increased risk of prostatic cancer. Hormonal stimulation (estrogen and testosterone) has been associated with prostatic cancer (Hill et al. 1982; Ross et al. 1987), and a history of multiple sexual partners and/or venereal disease has also been suggested to be related to prostatic cancer risk (Steele et al. 1971; Krain 1974). Unfortunately, no information on these potential risk factors was available in the present investigation.

Although the increase reported for the heterogeneous category of other lymphatic tissue cancer in the total cohort was almost statistically significant (SMR = 165; 95% CI: 98–260), the absence of any trend in SMRs with increasing duration of employment ($\chi^2 = 1.84$ and 0.60, in white and nonwhite males, respectively) was inconsistent with the hypothesis that the excess was related to exposures at the refinery. Furthermore, a review of the work histories of the 18 decedents showed no indication of common job assignments. It should be noted that state rates for other lymphatic tissue cancer are similar to U.S. rates for white males and approximately 7% higher among nonwhite males. Therefore only a small portion of the excess can be attributed to regional variation.

To further analyze mortality data for lymphatic and hematopoietic cancers, analyses for major cell type-specific leukemias (AML, CML, ALL, and CLL), NHL, and MM were performed. Mortality from cell type-specific leukemias was similar to expected (SMRs being 95, 53, 125, and 71 for AML, CML, ALL, and CLL, respectively). It must be pointed out that the number of observed deaths for each cell type was small. Nevertheless, these cell type-specific leukemia results are consistent with a meta-analysis based on more than 208 000 petroleum workers in the United States and the United Kingdom (Wong and Raabe 1995).

An increase in mortality was found for NHL among male employees (SMR = 132; 95% CI: 74–217), but the increase was not statistically significant. Other studies of refinery workers, which reported NHL as a separate disease category, did not find any association between NHL and refinery employment. In a recently completed study of workers at the company's Torrance, California refinery, Milcarek et al. (1994) reported an SMR of 120 (4 observed versus 3.3 expected; 95% CI: 33–307). Raabe et al. (1995) reported in a concurrent study at the company's Beaumont, Texas refinery that no significant increase in NHL was detected (24 observed versus 15.7 expected; SMR = 153; 95% CI: 98–226). Similarly, a recently completed large-scale study of refinery workers in Port Arthur, Texas, reported an SMR for NHL of 71 based on 30 observed NHL deaths (Satin et al. 1994). Thus, results from these studies supported the interpretation that the nonsignificant increase in NHL among the Paulsboro refinery workers was not occupationally related.

Epidemiologic studies have identified several potential risk factors for NHL, including autoimmune diseases, viruses, chemotherapy and radiation therapy, ionizing radiation, a history of diabetes, tuberculosis, or malaria, working or living on a farm, a family history of cancer, and cigarette smoking (Wilson et al. 1991; Franceschi et al. 1989; Cartwright et al. 1988; Vianna and Polan 1979; Schumacher 1985; Schumacher and Delzell 1988; McDonald 1986; Hoar et al. 1986; Zahm et al. 1990; Woods et al. 1987; Pearce et al. 1987). Unfortunately, no information on these potential risk factors was available for the NHL cases in this study.

Mortality from MM among male workers at the Paulsboro refinery was lower than expected (4 observed versus 5.4 expected; SMR = 74; 95% CI: 20–190). Similarly, other studies of refinery workers, in which MM was analyzed as a separate disease category, did not find any association between MM and refinery employment. The SMRs for MM were 128 (95% CI: 15–462) at Torrance, 121 (95% CI: 55–230) at Beaumont, and 98 (95% CI: 59–153) at Port Arthur refineries (Milcarek et al. 1994; Raabe et al 1995; Satin et al. 1994).

As at other industrial facilities, asbestos had been used at the Paulsboro refinery in the past. Although specific exposure measurements were not available, data from the industry as a whole have indicated that asbestos concentrations at refineries were generally extremely low. Among the Paulsboro refinery employees, no asbestosis death was reported, and no death was attributed to pulmonary fibrosis. Lung cancer mortality was significantly less than expected (SMR = 81). Furthermore, analysis for lung cancer by duration of employment did not show any trend. Two deaths were attributed to malignant mesothelioma, whereas 2.3

deaths were expected (SMR = 87). In addition, two deaths were coded as benign mesothelioma (ICD 212.3 and 228). Benign or localized mesothelioma is a distinct clinicopathologic entity from diffuse or malignant mesothelioma, and is not associated with exposure to asbestos (Antman and Corson 1985; Briselli et al. 1981; Weilbeacher and Sarma 1984). No mortality rates for benign mesothelioma for the general population were available for comparison. However, for the category "benign neoplasms", the observed mortality in the cohort was similar to the expected (6 observed versus 5.0 expected). Even if one assumed that these two benign mesotheliomas were misdiagnosed and were in fact malignant, the resulting SMR would be 174 (95% CI: 47–445), which would not be statistically significant.

In closing, it should be pointed out that there are some potential limitations in this study. Most are typical of a historical mortality study of industrial populations. First, although both the percentages of individuals with unknown vital status (2%) and the proportion of outstanding death certificates (2%) were low, it was possible, but unlikely, that some deaths from causes of interest might have been missed. We do not believe that such low percentages would have any significant impact on our results.

Second, the health endpoint in our study was mortality, which is a reasonable surrogate for incidence of most cancers. However, being a mortality study, the investigation inherited the problems associated with deaths certificates (diagnostic accuracy, for example). No detailed clinical information was available on the deaths in our study. However, it must be pointed out that although detailed information derived from medical records or pathology reports may be more accurate than that based on death certificates, it would be inappropriate to use such information in a historical cohort mortality study. In our study we compared diagnoses based on death certificates with national statistics which were derived from death certificates as well. Furthermore, our analysis was adjusted for calendar time, thus at least partially controlling for changes in survival and diagnostic practices.

Finally, although several additional detailed analyses were performed for both white and non-white male employees, the number of deaths among female employees remained small and it was not meaningful to perform similar detailed statistical analyses for the latter in this update.

Conclusion

This updated study has confirmed earlier findings of an overall favourable mortality experience of employees at the Paulsboro, New Jersey refinery compared to the U.S. general population. For the entire cohort, no significant increase was detected in any cause-specific mortality, except for prostatic cancer. Detailed analysis indicated that the prostatic cancer was not likely to be related to employment at the refinery. Mortality from leukemia (all cell types combined) was as expected. Detailed analyses by cell type-specific leukemias uncovered no increase. A nonsignificant increase in other lymphatic tissue cancers was also observed. However, no upward trend with duration of employment was detected for other lymphatic tissue cancer. Mortality from multiple myeloma was less than expected. No cause-specific mortality was found to be associated with duration of employment at the refinery, including several causes which have been reported to be elevated in some previous refinery studies.

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