

Exhibit 262

Mortality Updates (1970–1997) of Two Refinery/Petrochemical Plant Cohorts at Baton Rouge, Louisiana, and Baytown, Texas

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Objective: The purpose of this retrospective cohort study is to update mortality experience at refinery/petrochemical plant facilities in Baton Rouge, Louisiana, and Baytown, Texas. **Methods:** Standardized mortality ratios (SMRs) were calculated for 1970–1997 based on death rates in the respective states. **Results:** SMRs are near or below unity for most causes of death. Among elevations, Baton Rouge has an SMR of 2.42 (95% CI = 1.16–4.45) for chronic lymphocytic leukemia, 1.58 (95% CI = 1.17–2.08) for hypertension with heart disease, and 1.47 (95% CI = 0.98–2.11) for non-Hodgkin's lymphoma. Baytown has an SMR of 2.13 (95% CI = 1.10–3.73) for acute nonlymphocytic leukemia (ANLL) and 3.11 (95% CI = 1.01–7.26) for unspecified brain/spinal cord neoplasms. The above findings pertain to pre-1950 hires and exhibit no apparent job-related patterns. Both cohorts have approximately twofold increases in mesothelioma deaths, similar to the last update, with most decedents having held maintenance jobs. **Conclusions:** Periodic examination of mortality patterns has an important role in assessing overall employee health status and identifying potential areas of increased risk. Mortality surveillance will continue to monitor these outcomes among more recent workers. (J Occup Environ Med. 2004;46:1229–1245)

The purpose of this study is to update the mortality experience of employees at two ExxonMobil refinery/petrochemical plant facilities located in Baton Rouge, Louisiana, and Baytown, Texas, and compare findings with previous updates and other literature on similar types of workers. These studies are part of an ongoing surveillance program that tracks mortality patterns of company employees. Such investigations help identify potential clusters of disease so that focused analyses or other actions can be initiated in a timely manner. The surveillance process also provides an overall assessment of the health status of the population studied.

Baton Rouge and Baytown employees have been part of several studies of a combined refinery/petrochemical plant cohort from three locations in Louisiana, Texas, and New Jersey.^{1–5} These previous studies have shown low rates relative to the general population for overall mortality, all malignant neoplasms combined, and most specific causes of death.

In the previous (1970–1992) update of employees active at least 1 month during 1970–1982,⁴ approximately twofold elevations were seen in mesothelioma deaths at both Baton Rouge and Baytown based on six and seven deaths, respectively. For Baton Rouge, a significant mortality increase was found for kidney cancer, with no pattern of increasing risk with longer employment. Analyses by time period suggested that kidney

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cancer mortality had slightly decreased since the early 1980s. A 3.5-fold increase of chronic lymphocytic leukemia (CLL) was found at Baton Rouge, with all 10 deaths among employees hired before 1950. A subsequent incidence study (1983–1994) did not find elevated CLL.⁶ Baton Rouge also had elevations for categories of “other” (non-central nervous system) benign and unspecified neoplasms and hypertension with heart disease, and Baytown had an elevation of neoplasms of “uncertain behavior/unspecified neoplasms of brain/spinal cord.”⁴

The current study provides an additional 5 years of follow up for a total of 28 years (1970–1997) and presents more detailed analyses by period of hire. The analysis focuses on previously observed increases and on other causes of death reported and reviewed in the literature on petroleum workers.^{7–10} In general, these studies have found lower mortality from all causes and all malignant neoplasms compared to the U.S. general population and/or individual states. These reviews have noted inconsistent reports of increases for kidney cancer, brain and central nervous system tumors, cancers of blood and blood-forming organs (also termed lymphohematopoietic [LH] malignancies), mesothelioma, and melanoma. Thus, these causes of death are of a priori interest in the present study.

Materials and Methods

Population Definition

Characteristics of the Baton Rouge and Baytown cohorts have been described previously.^{3,4} In brief, the two cohorts in the present study consist of all employees active on January 1, 1970, or hired in the period 1970–1982 with at least 1 month of employment at the facility. Employee information is primarily from a health status registry of computerized work history and demographic records derived from human re-

sources databases, and is supplemented by paper work records.

Mortality Information

Mortality was updated using both annuitant benefits records and vital status tracing through the National Death Index (NDI). For the period of 1970–1982 only, Social Security Administration (SSA) data were also used. A nosologist trained by the National Centers for Health Statistics (NCHS) coded underlying cause of death according to the International Classification of Diseases (ICD) revision in effect at time of death (ICD 8 or 9). Periodic quality control studies have shown an error rate of less than 1% for cancer underlying causes of death as determined by this nosologist.

Design/Analysis

The study uses a retrospective cohort design, which tracks mortality in a cohort over a specified time period. Subjects contributed person-years from January 1, 1970, or from 30 days after hire at that site, whichever was later. Follow up ended at date of death or end of study (December 31, 1997), whichever was earlier. For individuals lost to follow up, period of follow up ended at date of employee termination.

Mortality for the period 1970–1997 was examined with a traditional standardized mortality ratio (SMR) analysis (observed/expected deaths). Each cohort was analyzed separately. Expected deaths were computed using a modified life table approach with the Occupational Cohort Mortality Analysis Program (OCMAP) software package.¹¹ OCMAP calculates expected deaths by multiplying person-years by mortality rates for the Louisiana (Baton Rouge) or Texas (Baytown) general population, specific for gender, race (white/nonwhite), and 5-year categories of age and calendar time. U.S. rates were also run for comparison purposes. OCMAP computes 95% confidence intervals (CIs) for SMRs using an exact method.¹² Tables in

the report include SMRs for causes of death with at least three observed or three expected deaths; otherwise, only observed and expected numbers are reported.

There is no single code for mesothelioma in ICD-8 and ICD-9 coding. As a result, the condition may be coded to a specific site (eg, peritoneum 158.9, pulmonary 162.9, pleural 163.9) or to “site unspecified” codes (eg, 199.1). Accordingly, all death certificates with these underlying or secondary codes were manually reviewed for any mention of mesothelioma. In the absence of U.S. death rates and because mortality for this condition is similar to incidence, we calculated expected numbers from Surveillance, Epidemiology, and End Results (SEER) incidence rates for invasive mesothelioma.¹³ For the 1970–1974 period, we applied 1970 SEER rates.

SMR analyses were run for each cohort and for subcohorts stratified by gender and race. We also ran analyses by period of hire (<1950, 1950+), and for findings of a priori interest and other increases (and when sample size permitted), we performed analyses by duration of employment duration (<15, 15–29, 30+ years) and latency (<20, 20+ years between first employment and death). The year 1950 was selected as a cutpoint to approximate early versus modern work eras, which is consistent with dates used in other studies. For the most part, other cutpoints were chosen to conform with previous analyses of these and other cohorts. Although no formal analyses by job type were done, we checked job information in computerized and paperwork history records and on death certificates to aid in interpretation.

Quality Control and Human Subjects

Several quality control procedures were followed to ensure data accuracy and consistency, including double data entry of death certificate

information. Crosschecks of data accuracy from different record sources were conducted, along with audits of computer output and reports for accuracy and completeness.

All study data were collected from company records, NDI, and SSA. The study required no contact with study subjects. Strict confidentiality standards were upheld in all phases of the study according to requirements of the Good Epidemiology Practices¹⁴ and those of NDI. These safeguards result in minimal or no risk to study subjects. On the other hand, the studies offer potential benefits to subjects and other employees who stand to gain from knowledge of potential workplace risks and assurance of low risk.

Results

Study Population Characteristics

The study cohort includes a total of 7637 Baton Rouge employees and 7007 Baytown employees. Demographic and employment characteristics are presented in Table 1. The cohorts are similar, consisting of predominantly white males, with approximately half hired in the 1970s and later. The average age at hire is 27 years, and average duration of employment is 22 years, with approximately one third employed for 30 years or longer. As determined from a previous assessment of “main occupation” during the period 1970–1982,³ approximately 70% of subjects were classified as operators, mechanics, and laborers. Other cohort members were assigned as professionals and technicians, with smaller proportions of managers and office/clerical workers. Almost all job categories are predominantly male (between 85% and 99%), whereas office/clerical jobs contain approximately 68% females.

Male and female cohort members contributed 166,057 (Baton Rouge) and 152,213 (Baytown) person-years of observation during the 28-year period of follow up (January 1, 1970 to December 31, 1997). Average fol-

TABLE 1
Characteristics of Cohort Members

	Baton Rouge		Baytown	
	No.	Percentage	No.	Percentage
Total cohort	7637	100	7007	100
Sex				
Male	6941	91	6241	89
Female	696	9	766	11
Race				
White	6403	84	6223	89
Nonwhite	1234	16	784	11
Sex/race				
White males	5847	77	5559	79
Nonwhite males	1094	14	682	10
White females	556	7	664	9
Nonwhite females	140	2	102	1
Year of birth				
<1920	2091	27	1778	25
1920–1929	989	13	1055	15
1930–1939	398	5	498	7
1940–1949	2269	30	1808	26
1950+	1890	25	1868	27
Year of hire				
<1940	630	8	655	9
1940–1949	2041	27	1791	26
1950–1959	255	3	219	3
1960–1969	1109	15	766	11
1970–1979	2868	38	2770	40
1980–1982	734	10	806	12
Duration of employment (yrs)				
<15	2185	29	2196	3
15–29	2979	39	2753	39
30+	2473	32	2058	29
Status at end of study (12/31/97)				
Active	1811	24	1414	20
Deceased	2018	26	1667	24
Terminated	3808	50	3926	56
Year of death				
1970–1979	426	21	302	18
1980–1989	783	39	661	40
1990–1997	809	40	704	42

low-up time was 22 years and three fourths of cohort members were followed for 20 or more years. At the end of study follow up, approximately half of cohort members had separated or retired and a fourth each were actively employed or deceased. The average age at last observation (time of death or end of study period) and age at death are approximately 59 years and 69 years, respectively. Most (87%) deaths were ascertained from company records and the remainder from the NDI and SSA. Ninety-four percent of Baton Rouge cohort deaths occurred in Louisiana and 96% of Baytown cohort deaths

occurred in Texas. Fewer than 2% of subjects, whose vital status during 1970–1978 could not be confirmed by SSA, were considered lost to follow up (121 in Baton Rouge, 81 in Baytown). Death certificates were obtained for >99% of known Baton Rouge deaths and 100% of Baytown deaths.

Overall Findings for Males

Findings are presented in Table 2 for males of all races. In Baton Rouge, nonwhite males comprise 16% of all males and account for 16% (308) of deaths, and the corresponding figures in Baytown are

TABLE 2

Male Employees: Baton Rouge and Baytown Cohorts, 1970–1997

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
All causes (1–999)	1982	2606.07	0.76†	0.73–0.80	1617	2185.86	0.74†	0.70–0.78
Infectious and parasitic diseases (1–139)	21	53.36	0.39†	0.24–0.60	15	43.68	0.34†	0.19–0.57
Malignant neoplasms (MN) (140–208)	552	661.04	0.84†	0.77–0.91	438	544.10	0.80†	0.73–0.88
MN of buccal cavity and pharynx (140–149)	6	16.42	0.36†	0.13–0.80	7	12.77	0.55	0.22–1.13
MN of digestive organs and peritoneum (150–159)	126	145.96	0.86	0.72–1.03	109	123.51	0.88	0.72–1.06
MN of esophagus (150)	13	15.49	0.84	0.45–1.44	12	12.61	0.95	0.49–1.66
MN of stomach (151)	14	20.17	0.69	0.38–1.16	14	16.27	0.86	0.47–1.44
MN of large intestine (153)	48	49.54	0.97	0.71–1.28	40	42.30	0.95	0.68–1.29
MN of rectum (154)	5	9.24	0.54	0.18–1.26	9	7.37	1.22	0.56–2.32
MN of biliary passages (including gallbladder)/liver (155–156)	7	16.18	0.43*	0.17–0.89	16	15.72	1.02	0.58–1.65
MN of pancreas (157)	36	32.68	1.10	0.77–1.52	15	26.65	0.56*	0.32–0.93
MN of respiratory system (160–165)	199	267.54	0.74†	0.64–0.86	144	212.56	0.68†	0.57–0.80
MN of larynx (161)	6	8.93	0.67	0.25–1.46	2	6.70	0.30	0.04–1.08
MN of bronchus, trachea, lung (162)	193	256.71	0.75†	0.65–0.87	140	203.87	0.69†	0.58–0.81
MN of pleura (163)	0	0.60	—	—	1	0.73	—	—
Malignant melanoma (172)	4	6.93	0.58	0.16–1.48	6	7.90	0.76	0.28–1.65
MN of breast (174–175)	0	0.68	—	—	0	0.48	—	—
MN of prostate (185)	40	61.19	0.65†	0.47–0.89	52	52.06	1.00	0.75–1.31
MN of bladder and other urinary (188; 189.3–189.4; 189.8–189.9)	15	14.24	1.05	0.59–1.74	9	11.95	0.75	0.34–1.43
MN of kidney (189.0; 189.1; 189.2)	21	14.43	1.46	0.90–2.22	15	13.30	1.13	0.63–1.86
MN of central nervous system, including brain (191–192)	17	12.98	1.31	0.76–2.10	12	12.75	0.94	0.49–1.64
MN of brain (191)	17	11.98	1.42	0.83–2.27	12	11.81	1.02	0.52–1.78
MN of other/ill-defined sites/secondary neoplasms (195–199)	34	55.71	0.61†	0.42–0.85	27	37.04	0.73	0.48–1.06
MN of lymphatic and hematopoietic tissue (200–208)	81	55.17	1.47†	1.17–1.82	54	49.32	1.10	0.82–1.43
Hodgkin's disease (201)	0	2.70	—	—	1	2.25	—	—
Non-Hodgkin's lymphoma (200.0–200.2; 200.8; 202.0–202.2; 202.8–202.9)	29	19.77	1.47	0.98–2.11	15	17.85	0.84	0.47–1.39
Multiple myeloma (203.0)	13	10.00	1.30	0.69–2.22	11	9.03	1.22	0.61–2.18
Leukemia (204–208)	37	21.92	1.69†	1.19–2.33	27	19.56	1.38	0.91–2.01
Acute nonlymphocytic (ANLL) (205.0; 206.0; 207.0; 207.2)	11	7.23	1.52	0.76–2.72	12	5.62	2.13*	1.10–3.73
Chronic myelocytic (CML) (205.1)	5	2.75	1.82	0.59–4.25	4	2.61	1.54	0.42–3.93
Acute lymphocytic (ALL) (204.0)	2	0.89	—	—	2	1	—	—
Chronic lymphocytic (CLL) (204.1)	10	4.13	2.42*	1.16–4.45	5	4.07	1.23	0.40–2.87
Other and unspecified leukemia (204.2–204.9; 205.2–205.9; 206.1–206.9; 207.1; 207.8; 208.0–208.9)	9	6.90	1.30	0.60–2.48	4	6.22	0.64	0.18–1.65
Benign/CIS/uncertain behavior/unspecified neoplasms (210–239)	16	8.61	1.86*	1.06–3.02	9	5.16	1.74	0.80–3.31
Benign CNS (including brain) (225.0–225.9)	0	0.76	—	—	0	0.70	—	—
Uncertain behavior/unspecified neoplasms of brain/spinal cord (237.5; 239.6)	7	3.73	1.88	0.76–3.87	5	1.61	3.11*	1.01–7.26
Other benign/in situ/uncertain behavior/unspecified (210–223; 226–236; 237.0–237.4; 238.0–239.5; 239.8–239.9)	8	3.76	2.13	0.92–4.19	2	2.70	—	—
Endocrine/nutritional/metabolic diseases (240–279)	48	69.05	0.70†	0.51–0.92	44	55.37	0.80	0.58–1.07
Diabetes mellitus (250)	39	55.57	0.70*	0.50–0.96	30	43.49	0.69*	0.46–0.98
All diseases of blood and blood-forming organs (280–289)	11	8.89	1.24	0.62–2.21	7	7.63	0.92	0.37–1.89
Aplastic anemia (284)	4	1.60	2.50	0.68–6.41	2	1.24	—	—
Nervous system/sense organ disease (320–389)	36	35.87	1.00	0.70–1.39	43	33.66	1.28	0.92–1.72
Amyotrophic lateral sclerosis (335.2)	8	4.05	1.98	0.85–3.89	7	3.73	1.87	0.75–3.86
Circulatory disease (390–459)	943	1174.96	0.80†	0.75–0.86	695	946.02	0.74†	0.68–0.79
All heart disease (390–398; 402; 404; 410–429)	788	957.74	0.82†	0.77–0.88	584	773.71	0.76†	0.70–0.82
Hypertension with heart disease (402; 404)	49	31.09	1.58†	1.17–2.08	5	19.43	0.26†	0.08–0.60
Ischemic heart disease (410–414)	563	711.70	0.79†	0.73–0.86	456	542.69	0.84†	0.76–0.92
Cerebrovascular disease (430–438)	102	147.12	0.69†	0.56–0.84	80	118.56	0.68†	0.54–0.84
Diseases of arteries/veins/other circulatory (440–459)	45	62.75	0.72*	0.52–0.96	27	46.46	0.58†	0.38–0.85

TABLE 2
Continued

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
Nonmalignant respiratory disease (460–519)	109	192.49	0.57†	0.46–0.68	123	180.35	0.68†	0.57–0.81
Pneumonia (480–486)	29	54.62	0.53†	0.36–0.76	33	50.49	0.65*	0.45–0.92
Bronchitis, emphysema, and asthma (490–493)	25	36.83	0.68	0.44–1.00	14	34.51	0.41†	0.22–0.68
Emphysema (492)	21	28.64	0.73	0.45–1.12	12	26.99	0.44†	0.23–0.78
Chronic obstructive pulmonary disease (496)	28	69.49	0.40†	0.27–0.58	51	68.95	0.74*	0.55–0.97
Pneumoconiosis/other lung disease—external agents (500–508)	8	7.61	1.05	0.45–2.07	11	7.43	1.48	0.74–2.65
Asbestosis (501)	2	0.63	—	—	4	0.64	6.25†	1.70–16.01
Digestive disease (520–579)	51	91.20	0.56†	0.42–0.74	55	85.17	0.65†	0.49–0.84
Cirrhosis of liver (571)	21	38.74	0.54†	0.34–0.83	20	40.78	0.49†	0.30–0.76
Genitourinary disease (580–629)	29	45.05	0.64*	0.43–0.92	27	28.76	0.94	0.62–1.37
Nephritis and nephrosis (580–589)	17	26.66	0.64	0.37–1.02	17	14.91	1.14	0.66–1.83
All external causes of death (800–999)	133	215.93	0.62†	0.52–0.73	118	178.73	0.66†	0.55–0.79
Accidents (800–949)	88	130.85	0.67†	0.54–0.83	74	103.87	0.71†	0.56–0.89
Motor vehicle accidents (810–825)	47	57.84	0.81	0.60–1.08	33	51.01	0.65†	0.44–0.91
Suicides (950–959)	37	46.94	0.79	0.56–1.09	30	40.24	0.75	0.50–1.06
Homicides and legal intervention (960–978)	8	36.12	0.22†	0.10–0.44	14	32.63	0.43†	0.24–0.72
All other causes of death (290–319, 680–799)	33	49.57	0.66†	0.46–0.93	43	77.19	0.55†	0.40–0.75

*Statistically significant at $P < 0.05$.

†Statistically significant at $P < 0.01$.

Observed (Obs) and Expected (Exp) Deaths, Standardized Mortality Ratios (SMRs), and 95% Confidence Intervals (CIs) Expected deaths based on state (Louisiana and Texas) male mortality rates (all races combined).

Baton Rouge: $n = 6941$, person-yr = 152,056.

Baytown: $n = 6241$, person-yr = 136,938.

11% and 7% (118). In general, mortality patterns in nonwhite males are similar to white males (not shown), although interpretation is hindered by small numbers for many causes of death. Similar to previous studies, males from both cohorts have substantial deficits for all causes, all malignant neoplasms combined, respiratory cancer, and many categories of nonmalignant disease, including heart, respiratory, digestive, and external causes of death.

As seen in Table 2, in the broad category of lymphohematopoietic (LH) malignancies, Baton Rouge males have a statistically significant SMR of 1.47. For leukemia, males have a statistically significant SMR of 1.69, based on 37 deaths, of which 34 are among whites. Leukemia findings include a statistically significant increase in CLL based on 10 deaths (SMR = 2.42) and not significant elevations of acute nonlymphocytic leukemia (ANLL) (SMR = 1.52) and chronic myelocytic leukemia (CML) (SMR = 1.82), based on 11

and 5 deaths, respectively. There is also a near-significant SMR of 1.47 for the subcategory of non-Hodgkin lymphoma (NHL), with a higher SMR for nonwhite males (3.16; 95% CI = 1.03–7.38, five deaths) compared with white males (1.32; 95% CI = 0.84–1.96, 24 deaths). Other nonsignificant elevations among Baton Rouge males include cancers of the kidney (SMR = 1.46, 21 deaths) and brain (SMR = 1.42, 17 deaths). For Baytown males, the overall leukemia SMR is a nonsignificant 1.38, with a statistically significant SMR of 2.13 for ANLL (12 deaths) and a nonsignificant SMR of 1.54 for CML (four deaths).

Among deaths resulting from neoplasms not classified as malignant, seven Baton Rouge deaths are in a category of “uncertain behavior/ unspecified neoplasms of the brain/spinal cord” (SMR = 1.88) and eight others are in a combined category of uncertain behavior and unspecified tumors *not* in the nervous system (SMR = 2.13) (both not significant).

In Baytown, five deaths resulting from “uncertain behavior/unspecified neoplasms of the brain/spinal cord” yield a borderline significant SMR of 3.11.

Mesothelioma was analyzed separately using a comparison of SEER incidence rates for invasive mesothelioma (see “Methods”). In Baton Rouge, eight deaths are reported versus 4.02 expected, for a nonsignificant SMR of 1.99 (95% CI = 0.86–3.92). Seven of the eight deaths occurred among men hired before 1950 (SMR = 2.10; 95% CI = 0.84–4.33). In Baytown, there were nine mesothelioma deaths compared with 3.90 expected, for a statistically significant SMR of 2.31 (95% CI = 1.06–4.38). All deaths occurred among workers hired in the early to mid-1940s, and the SMR for pre-1950 hires is 2.78 (95% CI = 1.27–5.28). All mesothelioma decedents from Baton Rouge and Baytown are white.

For nonneoplastic causes of death, substantial and statistically signifi-

cant deficits are found for almost all broad categories among both cohorts. For disease subgroups, the only statistically significant elevations are an SMR of 1.58 for hypertension with heart disease based on 49 deaths in Baton Rouge and an SMR of 6.25 for asbestosis based on four deaths in Baytown. Both cohorts have approximately twofold, nonsignificant increases for “motor neuron disease” based on eight and seven deaths in Baton Rouge and Baytown, respectively. Most of these deaths are classified as amyotrophic lateral sclerosis (ALS), also called Lou Gehrig’s disease.

Overall Findings for Females

In the Baton Rouge cohort, 36 deaths are reported among the 696 female members. SMRs are substantially below expected for overall mortality (SMR = 0.51; 95% CI = 0.36–0.71) and for all malignant neoplasms (SMR = 0.45; 95% CI = 0.20–0.85). Among nine cancer deaths, NHL has three versus 0.76 expected (not significant); all three decedents were hired before 1950 and had secretarial/administrative jobs. Baton Rouge females have four deaths versus 1.01 expected in the category of homicides and legal intervention (statistically significant), and nonsignificant elevations are found for cirrhosis of the liver and motor vehicle accidents (MVAs) based on three deaths each. Only four deaths occurred among nonwhite females. In the Baytown cohort, 50 deaths are reported among 766 female members. The SMR is 0.84 for overall mortality (95% CI, 0.62–1.10) and 1.20 for all malignant neoplasms (95% CI = 0.75–1.82). Only one death occurred among nonwhite females. White females have a statistically significant elevation of ovarian cancer (SMR = 4.96; 95% CI = 1.61–11.58), based on five deaths, and a nonsignificant elevation of cancer of the large intestine based on four deaths versus 1.39 expected.

Because the small numbers of female deaths preclude further analyses or meaningful interpretation, the remainder of this report is confined to males.

Subgroup Analyses—Males

Period of Hire. Male employees hired *before 1950* account for 85% of male deaths in both cohorts. SMRs for pre-1950 hires tend to be similar or slightly higher compared with the all-male analysis (Table 3). In Baton Rouge, statistically significant elevations are seen in the pre-1950 hire group for all leukemia and CLL subtypes and for “uncertain behavior/unspecified neoplasms of brain/spinal cord,” other benign/in situ/uncertain behavior/unspecified neoplasms, and hypertension with heart disease. In Baytown, SMRs are significantly elevated for asbestosis among all males and for multiple myeloma among nonwhite males (not shown) based on four deaths each.

In general, the low number of deaths (283 in Baton Rouge and 237 in Baytown) among males hired in *1950 or later* prevents meaningful analysis of all but the major causes of death (Table 4). This group has substantial and statistically significant mortality deficits for all causes and all malignant neoplasms (Baton Rouge SMRs = 0.50 and 0.56, respectively; Baytown SMRs = 0.52 and 0.67, respectively), with deficits for both whites and nonwhites. SMRs at or below unity are generally seen for broad categories of cancer, noncancer diseases, and external causes of death. Bearing in mind the small numbers, no elevations of note are seen for specific causes of death among 1950+ hires in Baton Rouge. In Baytown, 1950+ hires have a borderline nonsignificant SMR of 2.17 for leukemia (95% CI = 0.99–4.12) based on nine deaths, not confined to any single cell type. Finally, a nonsignificant SMR of 1.42 from prostate cancer based on six deaths is seen in Baytown males hired after 1950.

Latency/Duration. Analyses of trends by latency and duration of employment are limited by small numbers of employees in the shorter categories. For example, only 22 of 552 of Baton Rouge cancer decedents, and 24 of 438 Baytown cancer decedents, had <20 years latency. Analyses by duration of employment (<15, 15–29, 30+ years) were therefore confined to males with 20 or more years latency. As shown in Table 5, no trends are seen for either cohort for all causes of death or all malignant neoplasms. For disease subcategories of a priori interest, numbers in the <15-year category are too small to assess overall trends, and differences between the other two duration categories are generally unremarkable. The exception, perhaps, is the confinement of ANLL deaths (and increased risk) in both cohorts to the 30+-year latency category). Formal tests for trend¹⁵ were conducted on all results in Table 5; the only statistically significant finding was a downward trend for CLL, based on small numbers.

Subsets of Pre-1950 Hire Period. Because observed mortality increases in the Baton Rouge cohort were largely confined to employees hired before 1950, we examined finer cuts of the pre-1950 hire period. Table 6 shows findings for Baton Rouge males for the periods before, during, and after World War II (<1940, 1940–1944, and 1945–1949, respectively) for all causes, all circulatory diseases, all malignant neoplasms, and specific causes of death of a priori interest. For categories of all causes, circulatory diseases, and to a lesser extent, malignant neoplasms, those hired during the war (1940–1944) appear to have slightly higher mortality risk compared with those hired before and after the war. For specific causes, based on smaller numbers, Table 6 depicts some outcomes with more substantial risk among pre-1940 hires (eg, NHL, ANLL, other benign/uncertain behavior/unspecified neoplasms) or among those hired during

TABLE 3
Male Employees Hired Before 1950: Baton Rouge and Baytown Cohorts, 1970–1997

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
All causes (1–999)	1699	2043.89	0.83†	0.79–0.87	1380	1730.92	0.80†	0.76–0.84
Infectious and parasitic diseases (1–139)	18	28.73	0.63*	0.37–0.99	14	21.46	0.65	0.36–1.10
Malignant neoplasms (MN) (140–208)	483	538.09	0.90*	0.82–0.98	371	444.43	0.84†	0.75–0.92
MN of buccal cavity and pharynx (140–149)	4	12.72	0.31†	0.09–0.80	6	10.04	0.60	0.22–1.30
MN of digestive organs and peritoneum (150–159)	111	118.65	0.94	0.77–1.13	91	100.33	0.91	0.73–1.11
MN of esophagus (150)	10	11.98	0.84	0.40–1.54	10	9.99	1.00	0.48–1.84
MN of stomach (151)	12	16.73	0.72	0.37–1.25	11	13.13	0.84	0.42–1.50
MN of large intestine (153)	44	40.72	1.08	0.78–1.45	32	34.90	0.92	0.63–1.29
MN of rectum (154)	5	7.60	0.66	0.21–1.54	9	5.87	1.53	0.70–2.91
MN of biliary passages (including gallbladder)/liver (155–156)	5	12.75	0.39*	0.13–0.92	15	12.40	1.21	0.68–2.00
MN of pancreas (157)	32	26.76	1.20	0.82–1.69	12	21.94	0.55*	0.28–0.96
MN of respiratory system (160–165)	176	218.66	0.80†	0.69–0.93	125	174.81	0.72†	0.60–0.85
MN of larynx (161)	6	7.10	0.84	0.31–1.84	1	5.47	0.18	0.00–1.02
MN of bronchus, trachea, lung (162)	170	210.16	0.81†	0.69–0.94	122	167.78	0.73†	0.60–0.87
MN of pleura (163)	0	0.49	—	—	1	0.59	—	—
Malignant melanoma (172)	3	4.33	0.69	0.14–2.02	4	5.28	0.76	0.21–1.94
MN of breast (174–175)	0	0.52	—	—	0	0.40	—	—
MN of prostate (185)	37	56.41	0.66†	0.46–0.90	46	47.83	0.96	0.70–1.28
MN of bladder and other urinary (188; 189.3–189.4; 189.8–189.9)	14	12.68	1.10	0.60–1.85	9	10.58	0.85	0.39–1.62
MN of kidney (189.0; 189.1; 189.2)	17	10.99	1.55	0.90–2.48	13	10.26	1.27	0.68–2.17
MN of central nervous system, including brain (191–192)	14	8.66	1.62	0.88–2.71	9	8.92	1.01	0.46–1.92
MN of brain (191)	14	7.86	1.78	0.97–2.99	9	8.18	1.10	0.50–2.09
MN of other/ill-defined sites/secondary neoplasms (195–199)	32	45.24	0.71*	0.48–1.00	23	30.21	0.76	0.48–1.14
MN of lymphatic and hematopoietic tissue (200–208)	70	42.55	1.64†	1.28–2.08	42	38.43	1.09	0.79–1.48
Hodgkin's disease (201)	0	1.53	—	—	1	1.41	—	—
Non-Hodgkin's lymphoma (200.0–200.2; 200.8; 202.0–202.2; 202.8–202.9)	23	14.65	1.57	1.00–2.36	13	13.55	0.96	0.51–1.64
Multiple myeloma (203.0)	12	8.26	1.45	0.75–2.54	10	7.52	1.33	0.64–2.45
Leukemia (204–208)	34	17.42	1.95†	1.35–2.73	18	15.42	1.17	0.69–1.84
Acute nonlymphocytic (ANLL) (205.0; 206.0; 207.0; 207.2)	11	5.69	1.93	0.96–3.46	9	4.35	2.07	0.95–3.93
Chronic myelocytic (CML) (205.1)	4	1.93	2.08	0.57–5.32	2	1.82	—	—
Acute lymphocytic (ALL) (204.0)	1	0.55	—	—	1	0.61	—	—
Chronic lymphocytic (CLL) (204.1)	10	3.56	2.81†	1.35–5.17	3	3.52	0.85	0.18–2.49
Other and unspecified leukemia (204.2–204.9; 205.2–205.9; 206.1–206.9; 207.1; 207.8; 208.0–208.9)	8	5.67	1.41	0.61–2.78	3	5.07	0.59	0.12–1.73
Benign/CIS/uncertain behavior/unspecified neoplasms (210–239)	16	6.69	2.39†	1.37–3.89	8	4.15	1.93	0.83–3.80
Benign CNS (including brain) (225.0–225.9)	0	0.60	—	—	0	0.58	—	—
Uncertain behavior/unspecified neoplasms of brain/spinal cord (237.5; 239.6)	7	2.75	2.54*	1.02–5.24	4	1.22	3.28	0.89–8.39
Other benign/in situ/uncertain behavior/unspecified (210–223; 226–236; 237.0–237.4; 238.0–239.5; 239.8–239.9)	8	3.08	2.60*	1.12–5.12	2	2.25	—	—
Endocrine/nutritional/metabolic diseases (240–279)	38	53.27	0.71*	0.50–0.98	35	42.70	0.82	0.57–1.14
Diabetes mellitus (250)	34	44.04	0.77	0.54–1.08	27	34.15	0.79	0.52–1.15
All diseases of blood and blood-forming organs (280–289)	10	6.86	1.46	0.70–2.68	7	6.20	1.13	0.45–2.33
Aplastic anemia (284)	4	1.33	3.01	0.82–7.72	2	1.04	—	—
Nervous system/sense organ disease (320–389)	34	28.35	1.20	0.83–1.68	39	27.78	1.40	1.00–1.92
Amyotrophic lateral sclerosis (335.2)	7	3.00	2.33	0.94–4.81	6	2.88	2.08	0.77–4.54
Circulatory disease (390–459)	852	990.55	0.86†	0.80–0.92	625	801.78	0.78†	0.72–0.84
All heart disease (390–398; 402; 404; 410–429)	707	801.91	0.88†	0.82–0.95	522	651.34	0.80†	0.73–0.87
Hypertension with heart disease (402; 404)	44	24.80	1.77†	1.29–2.38	5	14.76	0.34†	0.11–0.79
Ischemic heart disease (410–414)	510	599.93	0.85†	0.78–0.93	403	461.26	0.87†	0.79–0.96

TABLE 3
Continued

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
Cerebrovascular disease (430–438)	95	127.73	0.74†	0.60–0.91	74	103.65	0.71†	0.56–0.90
Diseases of arteries/veins/other circulatory (440–459)	43	54.82	0.78	0.57–1.06	25	40.72	0.61*	0.40–0.91
Nonmalignant respiratory disease (460–519)	99	167.50	0.59†	0.48–0.72	112	158.58	0.71†	0.58–0.85
Pneumonia (480–486)	27	47.48	0.57†	0.38–0.83	29	43.76	0.66*	0.44–0.95
Bronchitis, emphysema, and asthma (490–493)	21	32.25	0.65*	0.40–1.00	13	30.67	0.42†	0.23–0.72
Emphysema (492)	19	25.63	0.74	0.45–1.16	11	24.32	0.45†	0.23–0.81
Chronic obstructive pulmonary disease (496)	26	61.88	0.42†	0.27–0.62	45	61.69	0.73*	0.53–0.98
Pneumoconiosis/other lung disease—external agents (500–508)	7	6.67	1.05	0.42–2.16	11	6.61	1.66	0.83–2.98
Asbestosis (501)	2	0.53	—	—	4	0.57	7.05†	1.92–18.05
Digestive disease (520–579)	41	66.29	0.62†	0.44–0.84	45	61.81	0.73*	0.53–0.97
Cirrhosis of liver (571)	17	25.18	0.68	0.39–1.08	14	26.04	0.54*	0.29–0.90
Genitourinary disease (580–629)	27	38.70	0.70	0.46–1.02	25	25.10	1.00	0.64–1.47
Nephritis and nephrosis (580–589)	16	22.73	0.70	0.40–1.14	16	12.87	1.24	0.71–2.02
All external causes of death (800–999)	53	82.48	0.64†	0.48–0.84	62	75.07	0.83	0.63–1.06
Accidents (800–949)	34	54.14	0.63†	0.44–0.88	44	48.33	0.91	0.66–1.22
Motor vehicle accidents (810–825)	12	19.73	0.61	0.31–1.06	18	20.10	0.90	0.53–1.42
Suicides (950–959)	15	20.91	0.72	0.40–1.18	15	19.06	0.79	0.44–1.30
Homicides and legal intervention (960–978)	4	6.89	0.58	0.16–1.49	3	6.96	0.43	0.09–1.26
All other causes of death (290–319, 680–799)	28	36.38	0.77	0.51–1.12	37	61.85	0.60	0.42–0.82

*Statistically significant at $P < 0.05$.

†Statistically significant at $P < 0.01$

Expected deaths based on state (Louisiana and Texas) male mortality rates (all races combined).

Observed (Obs) and Expected (Exp) Deaths, Standardized Mortality Ratios (SMRs), and 95% Confidence Intervals (CIs)

Baton Rouge: $n = 2598$, person-yr = 52,931.

Baytown: $n = 2377$, person-yr = 51,690.

1940–1944 (eg, CLL, “uncertain behavior/unspecified neoplasms of brain/spinal cord”); tests for trend¹⁵ were not statistically significant.

Discussion

Strengths and Limitations

This study design offers an efficient way to monitor mortality patterns and trends in large worker populations. The cohorts have undergone thorough mortality tracking, and have sufficient follow-up time and periods of employment to detect potentially work-related effects. Both the percentage of those lost to follow up and the number of deaths lacking death certificates are low and unlikely to impact results. The SMR analysis takes into consideration the effects of age, gender, race, and calendar time of death. An important limitation of the study is lack of data on possible confounding effects of factors such as lifestyle,

socioeconomic status, and employment elsewhere. To some extent, however, the use of state mortality rates mitigated effects of regional differences in mortality as a result of factors related to socioeconomic status. Interpretation of findings is also limited by reliance on relatively crude measures of job experience to evaluate relationships with employment. Analyses by period of hire have been useful in discerning increases in earlier hire groups, but assessment of trends in employment duration and latency are substantially limited by small numbers in the shorter categories. Information on job type from paper records and from “usual occupation” on death certificates was often helpful but not consistent.

Other uncertainties associated with the retrospective cohort design need to be considered in interpretation. The best known is the “healthy worker effort” (HWE), or the gener-

ally lower mortality in groups that can gain employment and receive health advantages of maintaining employment (eg, better access to medical care, health benefits of higher socioeconomic status). Another aspect of the HWE is that approximately half of the cohort members were hired before 1970 and to some degree may be “healthy survivors” compared with others who died or separated before the beginning of follow up in 1970. In general, risks may also be *underestimated* because of dilution of SMRs from heterogeneity of exposure. On the other side, risks may be *overestimated* as a result of diagnostic and detection biases of conditions among employed persons, and results from multiple comparisons need to be considered cautiously to avoid over-interpretation of findings that may be the result of chance. On balance, although the uncertainties discussed here preclude our ability to make

TABLE 4
Male Employees Hired in 1950 or Later: Baton Rouge and Baytown Cohorts, 1970–1997

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
All causes (1–999)	283	562.17	0.50†	0.45–0.57	237	454.94	0.52†	0.46–0.59
Infectious and parasitic diseases (1–139)	3	24.63	0.12†	0.02–0.36	1	22.23	0.04†	0.01–0.25
Malignant neoplasms (MN) (140–208)	69	122.96	0.56†	0.44–0.71	67	99.68	0.67†	0.52–0.85
MN of buccal cavity and pharynx (140–149)	2	3.70	0.54	0.06–1.95	1	2.73	—	—
MN of digestive organs and peritoneum (150–159)	15	27.31	0.55*	0.31–0.91	18	23.18	0.78	0.46–1.23
MN of esophagus (150)	3	3.51	0.86	0.18–2.50	2	2.62	—	—
MN of stomach (151)	2	3.44	0.58	0.07–2.10	3	3.14	0.96	0.20–2.79
MN of large intestine (153)	4	8.82	0.45	0.12–1.16	8	7.39	1.08	0.47–2.13
MN of rectum (154)	0	1.65	—	—	0	1.49	—	—
MN of biliary passages (including gallbladder)/liver (155–156)	2	3.42	0.58	0.07–2.11	1	3.32	0.30	0.01–1.68
MN of pancreas (157)	4	5.91	0.68	0.18–1.73	3	4.71	0.64	0.13–1.86
MN of respiratory system (160–165)	23	48.88	0.47†	0.30–0.71	19	37.75	0.50†	0.30–0.79
MN of larynx (161)	0	1.83	—	—	1	1.22	—	—
MN of bronchus, trachea, lung (162)	23	46.56	0.49†	0.31–0.74	18	36.09	0.50†	0.30–0.79
MN of pleura (163)	0	0.11	—	—	0	0.14	—	—
Malignant melanoma (172)	1	2.60	0.38	0.01–2.14	2	2.62	0.76	0.09–2.76
MN of breast (174–175)	0	0.16	—	—	0	0.09	—	—
MN of prostate (185)	3	4.78	0.63	0.13–1.83	6	4.23	1.42	0.52–3.09
MN of bladder and other urinary (188; 189.3–189.4; 189.8–189.9)	1	1.56	—	—	0	1.37	—	—
MN of kidney (189.0; 189.1; 189.2)	4	3.44	1.16	0.32–2.98	2	3.04	0.66	0.08–2.38
MN of central nervous system, including brain (191–192)	3	4.32	0.69	0.14–2.03	3	3.84	0.78	0.16–2.29
MN of brain (191)	3	4.12	0.73	0.15–2.13	3	3.63	0.83	0.17–2.41
MN of other/ill-defined sites/secondary neoplasms (195–199)	2	10.47	0.19†	0.02–0.69	4	6.83	0.59	0.16–1.50
MN of lymphatic and hematopoietic tissue (200–208)	11	12.62	0.87	0.44–1.56	12	10.89	1.10	0.57–1.92
Hodgkin's disease (201)	0	1.17	—	—	0	0.84	—	—
Non Hodgkin's lymphoma (200.0–200.2; 200.8; 202.0–202.2; 202.8–202.9)	6	5.13	1.17	0.43–2.55	2	4.30	0.46	0.06–1.68
Multiple myeloma (203.0)	1	1.74	—	—	1	1.51	—	—
Leukemia (204–208)	3	4.50	0.67	1.14–1.95	9	4.14	2.17	0.99–4.12
Acute nonlymphocytic (ANLL) (205.0; 206.0; 207.0; 207.2)	0	1.54	—	—	3	1.27	2.36	0.49–6.88
Chronic myelocytic (CML) (205.1)	1	0.82	—	—	2	0.78	2.55	0.31–9.21
Acute lymphocytic (ALL) (204.0)	1	0.33	—	—	1	0.39	—	—
Chronic lymphocytic (CLL) (204.1)	0	0.57	—	—	2	0.55	—	—
Other and unspecified leukemia (204.2–204.9; 205.2–205.9; 206.1–206.9; 207.1; 207.8; 208.0–208.9)	1	1.23	—	—	1	1.14	—	—
Benign/CIS/uncertain behavior/unspecified neoplasms (210–239)	0	1.92	—	—	1	1.01	—	—
Benign CNS (including brain) (225.0–225.9)	0	0.16	—	—	0	0.12	—	—
Uncertain behavior/unspecified neoplasms of brain/spinal cord (237.5; 239.6)	0	0.97	—	—	1	0.39	—	—
Other benign/in situ/uncertain behavior/unspecified (210–223; 226–236; 237.0–237.4; 238.0–239.5; 239.8–239.9)	0	0.68	—	—	0	0.45	—	—
Endocrine/nutritional/metabolic diseases (240–279)	10	15.78	0.63	0.30–1.17	9	12.66	0.71	0.32–1.35
Diabetes mellitus (250)	5	11.53	0.43	0.14–1.01	3	9.34	0.32*	0.07–0.94
All diseases of blood and blood-forming organs (280–289)	1	2.03	—	—	0	1.43	—	—
Aplastic anemia (284)	0	0.27	—	—	0	0.20	—	—
Nervous system/sense organ disease (320–389)	2	7.53	0.27*	0.03–0.96	4	5.88	0.68	0.18–1.74
Amyotrophic lateral sclerosis (335.2)	1	1.05	—	—	1	0.86	—	—
Circulatory disease (390–459)	91	184.42	0.49†	0.40–0.61	70	144.25	0.48†	0.38–0.61
All heart disease (390–398; 402; 404; 410–429)	81	155.83	0.52†	0.41–0.65	62	122.38	0.51†	0.39–0.65
Hypertension with heart disease (402; 404)	5	6.29	0.80	0.26–1.86	0	4.67	—	—
Ischemic heart disease (410–414)	53	111.77	0.47†	0.36–0.62	53	81.43	0.65†	0.49–0.85

TABLE 4
Continued

Cause of Death (9th revision ICD codes)	Baton Rouge				Baytown			
	Obs	Exp	SMR	95% CI	Obs	Exp	SMR	95% CI
Cerebrovascular disease (430–438)	7	19.39	0.36†	0.14–0.74	6	14.91	0.40*	0.15–0.88
Diseases of arteries/veins/other circulatory (440–459)	2	7.93	0.25*	0.03–0.91	2	5.74	0.35	0.04–1.26
Nonmalignant respiratory disease (460–519)	10	25.00	0.40†	0.19–0.74	11	21.77	0.51*	0.25–0.90
Pneumonia (480–486)	2	7.13	0.28	0.03–1.01	4	6.73	0.59	0.16–1.52
Bronchitis, emphysema, and asthma (490–493)	4	4.58	0.87	0.24–2.24	1	3.84	0.26	0.01–1.45
Emphysema (492)	2	3.00	0.67	0.08–2.41	1	2.67	0.38	0.01–2.09
Chronic obstructive pulmonary disease (496)	2	7.61	0.26*	0.03–0.95	6	7.26	0.83	0.30–1.80
Pneumoconiosis/other lung disease—external agents (500–508)	1	0.94	—	—	0	0.82	—	—
Asbestosis (501)	0	0.10	—	—	0	0.07	—	—
Digestive disease (520–579)	10	24.91	0.40†	0.19–0.74	10	23.36	0.43†	0.21–0.79
Cirrhosis of liver (571)	4	13.56	0.30†	0.08–0.76	6	14.74	0.41*	0.15–0.89
Genitourinary disease (580–629)	2	6.35	0.32	0.04–1.14	2	3.66	0.55	0.07–1.98
Nephritis and nephrosis (580–589)	1	3.93	0.26	0.01–1.42	1	2.04	—	—
All external causes of death (800–999)	80	133.46	0.60†	0.48–0.75	56	103.66	0.54†	0.41–0.70
Accidents (800–949)	54	76.71	0.70†	0.53–0.92	30	55.54	0.54†	0.36–0.77
Motor vehicle accidents (810–825)	35	38.10	0.92	0.64–1.28	15	30.91	0.49†	0.27–0.80
Suicides (950–959)	22	26.03	0.84	0.53–1.28	15	21.18	0.71	0.40–1.17
Homicides and legal intervention (960–978)	4	29.24	0.14†	0.04–0.35	11	25.67	0.43†	0.21–0.77
All other causes of death (290–319, 680–799)	5	13.18	0.38	0.12–0.88	6	15.34	0.39	0.14–0.85

*Statistically significant at $P < 0.05$.

†Statistically significant at $P < 0.01$.

Expected deaths based on state (Louisiana and Texas) male mortality rates (all races combined).

Observed (Obs) and Expected (Exp) Deaths, Standard Mortality Ratios (SMRs), and 95% Confidence Intervals (CIs)

Baton Rouge: $n = 4343$, person-yr = 99,125.

Baytown: $n = 3864$, person-yr = 85,248.

definitive conclusions about risk, these studies are valuable for broad surveillance and identification of potential problem areas that may need to be addressed further.

Discussion of Results

Overall, findings from this study are similar to the last update⁴ and have shown generally lower mortality compared with the Louisiana and Texas general populations. This held true when we used U.S. population rates for comparisons (not shown), although SMRs were generally slightly higher than when using Louisiana and Texas rates. This difference was expected because mortality rates are generally lower in the United States than in Louisiana and Texas and highlights the importance of using a source population closer to that from which the workers arose to obtain a less biased comparison.

Analyses by period of hire were the most informative. The Baton Rouge cohort, in particular, had several elevations among workers hired before 1950 and some apparent differences in risk depending on when they were hired during the 1940s (Table 6) (an effect not seen in the Baytown cohort). Others have observed higher mortality patterns among men hired during World War II, suggesting that wartime hires may experience differences in exposures and processes in wartime versus other periods and/or may have poorer overall health compared with those in active military service.^{16,17} The Baton Rouge facility expanded and increased production as part of the war effort (Baton Rouge as well as Baytown produced raw materials for synthetic rubber and large volumes of high octane aviation gasoline). Although hiring patterns and wartime activities should be consid-

ered in interpretation, it is not possible to characterize exactly how these factors may have affected patterns of mortality.

Chronic Lymphocytic Leukemia

The previous update (1970–1992) found a 3.5-fold excess of CLL in Baton Rouge employees based on 10 deaths.⁴ All decedents were hired in the period 1937–1947, worked from 27 to 38 years, and had latencies of 31 to 50 years. All spent at least some time in operator or maintenance jobs. However, detailed assessment of paper job history records by an industrial hygienist found no obvious patterns in work assignments or potential exposures (Armstrong TW, Huebner WW, internal report). A subsequent study of LH malignancy *incidence* was conducted in collaboration with the Louisiana Tumor Registry (LTR),⁶ which examined a somewhat younger cohort

TABLE 5
Analyses By Duration of Employment for 20+ Years Latency for Selected Causes of Death. Male Employees: Baton Rouge and Baytown Cohorts—1970–1997

Cause of death	Baton Rouge			Baytown		
	<15 yr duration	15–29 yr duration	30+ yr duration	<15 yr duration	15–29 yr duration	30+ yr duration
All causes	44/56.70 0.78 (0.56–1.04)	501/609.23 0.82† (0.75–0.90)	1308/1647.28 0.79† (0.75–0.84)	38/56.83 0.67* (0.47–0.92)	435/556.64 0.78† (0.71–0.86)	1031/1307.71 0.79† (0.74–0.84)
All malignant neoplasms (MNs)	12/15.38 0.78 (0.40–1.36)	132/153.12 0.86 (0.72–1.02)	386/445.94 0.87† (0.78–0.96)	14/15.83 0.88 (0.48–1.48)	116/138.07 0.84 (0.69–1.01)	284/344.58 0.82† (0.73–0.93)
Malignant melanoma	0/0.31	2/1.63	1/3.61	0/0.31	2/1.92	3/4.07
MN of kidney	0/0.47	7/3.51	(0.01–1.54) 13/9.12	0/0.50	3/3.43	(0.15–2.15) 11/7.98
All LH malignancies	1/1.47	(0.80–4.11) 24/12.55 1.91†	(0.76–2.44) 51/35.17 1.45*	2/1.55	(0.18–2.56) 13/12.20 1.07	(0.69–2.47) 32/29.71 1.08
NHL	0/0.64	9/4.46	18/12.35	0/0.66	7/4.40	7/10.56
All leukemia	1/0.49	(0.92–3.83) 9/4.99	(0.86–2.30) 25/14.26 1.75*	1/0.56	(0.64–3.28) 4/4.78	(0.27–1.36) 16/11.92
ANLL	0/0.16	(0.82–3.42) 1/1.63	(1.13–2.59) 10/4.66 2.15*	0/0.17	(0.23–2.14) 1/1.38	(0.77–2.18) 9/3.37
CLL	0/0.08	3/0.93	(1.03–3.95) 7/2.96	1/0.11	2/1.04	(1.22–5.07) 2/2.74
Other/unspecified leukemia (besides ANLL, CML, ALL, CLL)	0/0.13	(0.66–9.40) 4/1.61	(0.95–4.87) 4/4.58 0.87	0/0.16	1/1.52	2/3.92
MN of brain	1/0.49	(0.68–6.36) 3/2.77	(0.24–2.23) 12/6.62 1.81	1/0.51	5/2.91	(0.06–1.84) 5/6.36
Uncertain behavior/unspecified neoplasms of brain/spinal cord	0/0.09	(0.22–3.16) 2/0.86	(0.94–3.16) 5/2.23 2.24	0/0.04	(0.56–4.01) 2/0.41	(0.26–1.83) 3/0.91
Other benign/in situ/uncertain behavior/unspecified neoplasms	0/0.08	1/0.82	(0.73–5.22) 7/2.56	0/0.07	1/0.68	(0.68–9.60) 1/1.72
Hypertension with heart disease	1/0.90	11/6.70	(1.10–5.63) 36/21.02 1.71†	0/0.73	2/4.71	3/11.59
		(0.82–2.94) 1.64	(1.20–2.37) 1.71†		(0.05–1.54) 0.42	(0.05–0.76) 0.26†

*Statistically significant at $P < 0.05$.

†Statistically significant at $P < 0.01$.

Expected deaths based on state (Louisiana and Texas) male mortality rates (all races combined).
Observed / Expected Deaths, Standardized Mortality Ratios (bold) and (95% Confidence Intervals)
Baton Rouge: $n = 5806$, person-yr = 76,454.
Baytown: $n = 4966$, person-yr = 68,935.

TABLE 6

Analyses of Periods Before, During, and After World War II for Selected Causes of Death. Males Employees Hired Before 1950: Baton Rouge, 1970–1997

Cause of Death	Baton Rouge—Pre-1950 Hire Group		
	<1940	1940–1944	1945–1949
	Average age at hire 23 yr	Average age at hire 27 yr	Average age at hire 26 yr
All causes	477/602.66 0.79* (0.72–0.87)	910/1024.07 0.89† (0.83–0.95)	312/417.16 0.75* (0.67–0.84)
All circulatory diseases	238/300.74 0.79† (0.69–0.90)	467/496.17 0.94 (0.86–1.03)	147/193.63 0.76† (0.64–0.89)
All malignant neoplasms (MN)	134/149.92 0.89 (0.75–1.06)	255/270.26 0.94 (0.83–1.07)	94/117.91 0.80 (0.64–0.98)
MN of kidney	2/2.86 —	9/5.45 1.65 (0.76–3.14)	6/2.68 2.23 (0.82–4.86)
All LH malignancies	22/11.77 1.87† (1.17–2.83)	35/21.21 1.65† (1.15–2.30)	13/9.57 1.36 (0.72–2.32)
NHL (non-Hodgkin's lymphoma)	8/3.80 2.11 (0.91–4.15)	10/7.24 1.38 (0.66–2.54)	5/3.61 1.39 (0.45–3.24)
All leukemia	12/4.98 2.41* (1.24–4.21)	17/8.68 1.96* (1.14–3.14)	5/3.76 1.33 (0.43–3.10)
ANLL (acute nonlymphocytic leukemia)	5/1.60 3.12* (1.01–7.28)	4/2.85 1.41 (0.38–3.60)	2/1.24 —
CLL (chronic lymphocytic leukemia)	3/1.03 2.91 (0.60–8.50)	6/1.77 3.39* (1.24–7.38)	1/0.76 —
MN of brain	4/1.75 2.29 (0.63–5.85)	6/3.89 1.54 (0.57–3.36)	4/2.22 1.81 (0.49–4.62)
Uncertain behavior/unspecified neoplasms of brain/spinal cord	2/0.74 —	5/1.37 3.64* (1.18–8.49)	0/0.64 —
Other benign/in situ/uncertain behavior/unspecified neoplasms	4/0.93 4.30* (1.17–11.00)	3/1.53 1.96 (0.40–5.74)	1/0.62 —
Hypertension with heart disease	14/7.99 1.75 (0.96–2.94)	26/12.70 2.05† (1.34–3.00)	4/4.11 0.97 (0.26–2.49)

*Statistically significant at $P < 0.05$.

†Statistically significant at $P < 0.01$.

Expected deaths based on Louisiana male mortality rates (all races combined).

Observed / Expected Deaths, Standardized Mortality Ratios (bold) and (95% Confidence Intervals)

Baton Rouge: $n = 2598$, person-yr = 52,931.

of Baton Rouge employees active between 1970 and 1994 and alive as of January 1, 1983. This study calculated standardized incidence ratios (SIR) for overall LH malignancies and subtypes. Using LTR records from 1983 to 1994, four incident cases of CLL were found versus 3.27 expected among pre-1950 hires and one case versus 0.83 expected among 1950+ hires.

Regarding the present mortality update, Baytown continues to show no CLL elevation. Baton Rouge has no new CLL deaths in the update period (SMR has decreased to 2.42). Baton Rouge men hired between 1940 and 1944 appear the most impacted by CLL, and analysis by du-

ration of employment does not indicate work-related association with CLL mortality.

Studies of other petroleum industry cohorts, including refining/chemical plant groups, have reported a lack of excess CLL.^{9,18,19} CLL and benzene have been examined in nested case-control studies of petroleum industry cohorts. An Institute of Petroleum study (marketing and distribution) performed many analyses of leukemia subtypes and various benzene exposure metrics, and the authors concluded that the data did not support a benzene/CLL association.²⁰ The Australian Health Watch study found positive associations between CLL and cumulative lifetime exposures to ben-

zene of 4 ppm-years and higher based on 11 cases.²¹ However, risk estimates were unstable.

To conclude, the recent incidence study⁶ and the present mortality update are reassuring, whereas reasons for the elevation among early employees remains unclear. Possible explanations include work or nonwork factors associated with earlier work eras, work conditions particular to wartime, or selection of relatively less healthy workers who were ineligible for active military service. However, the elevation is not seen in Baytown, which is likely to have undergone similar time-related conditions. Another possible expla-

nation is more complete diagnoses and cause of death determinations among employed persons

Acute Nonlymphocytic Leukemia

The ANLL category combined acute myelocytic (AML, the most common) with acute monocytic, megakaryocytic, and acute erythremia/erythroleukemia. In Baton Rouge, all 11 deaths from ANLL were diagnosed as AML. The update added three ANLL deaths and the SMR is the same (1.52, not significant). No ANLL deaths occurred among men hired in 1950 or later (vs. 1.54 expected). The highest SMR is for men hired *before 1940* who have a statistically significant, threefold elevation based on five deaths (Table 6). All 11 decedents were long-term employees (29–41 years) working primarily in maintenance and a variety of other plant jobs with an average latency of 47 years (range, 36–57 years).

In Baytown, the statistically significant SMR of 2.13 for ANLL (based on 12 deaths) was not evident in the last update⁴; the previous SMR for AML was 1.17 based on five deaths. In the present update, 10 of the 12 ANLL deaths are in the AML category, one is acute erythroleukemia and the other is acute monocytic leukemia. Nine of the 12 ANLL decedents began working before 1950 in various mechanical, process, and professional jobs averaging 35 years duration and latencies ranging from 36 to 59 years. The three ANLL deaths among 1950+ hires compare with 1.27 expected; decedents had a variety of jobs and two of the three had <5 years of company employment.

ANLL is causally linked to occupational exposure to high levels of benzene,²² although presumably at far higher levels than experienced in early refinery settings. Because the pre-1950 hire group is obviously heterogeneous regarding potential “exposures,” the SMR for the more ex-

posed workers in the group could be higher than we report here. On the other hand, the present data do not show patterns suggestive of a workplace etiology; specific jobs among decedents are varied and do not suggest common exposures, and the extremely long latency periods are uncharacteristic of chemically induced ANLL (some studies suggest 15 or fewer years)^{23,24}

The literature shows no evidence of substantial ANLL risk in refinery/chemical plant workers. Three other recent studies reported slight elevations for ANLL among pre-1950 hired workers.^{19,25,26} A combined analysis of 15 petroleum refinery cohort studies in the United States and United Kingdom found an overall SMR of 0.93 for AML.⁹ An earlier study at Shell’s Woodriver (Illinois) refinery found a 2.2-fold excess of AML for the years 1970–1984²⁷; a nested case–control study was not able to identify any responsible job or work location.²⁸ Another earlier case–control study of leukemia among petroleum workers at Union Oil Company did not have a sufficient sample size to contribute to knowledge about associations between refinery work and AML.²⁹ Inconsistent results for ANLL have been found among three more recent case–control studies that examined associations with lower levels of benzene exposure among petroleum distribution workers and petroleum workers as a whole.^{20,21,30}

Non-Hodgkin’s Lymphoma

Previous studies of this cohort did not examine NHL per se, but parts of NHL were analyzed in the category of lymph/reticulosarcoma (ICD-9 200) and in “malignancies of other lymphohematopoietic tissue,” which contains “other lymphomas,” (202) but also multiple myeloma (203), which is not NHL. The last update found SMRs of 1.00 or slightly above for both cohorts.⁴

The current analysis used the NHL definition from the International Classifications of Diseases for On-

cology, 2nd edition,³¹ expressed as ICD-9 codes³² as follows: reticulosarcoma (200.0), lymphosarcoma (200.1), Burkitt’s lymphoma (200.2), other named variants (200.8), nodular/follicular lymphoma (202.0), T-cell lymphoid variety (202.1–202.2), a general category of other lymphomas (202.8), and other and unspecified malignant neoplasms of lymphoid and histiocytic tissue (202.9).

As defined here, NHL has a borderline not significant SMR of 1.57 among Baton Rouge employees hired before 1950, based on 23 deaths. Most decedents were hired before 1945, and the highest SMR is seen for those hired before 1940 (SMR = 2.11, based on eight deaths). Among the 23 decedents hired before 1950, there are no consistent patterns of job type. Analysis by duration of employment gives no indication of an upward trend with increasing length of service. No excess NHL mortality was found at Baytown.

Overall, NHL risk in refinery and other petroleum workers studies is not well characterized because of incomplete and inconsistent case definitions over time and across studies. With that caveat in mind, a recent combined analysis of NHL reports an overall SMR of 0.96 among 226,000 refinery workers from 14 U.S. studies, with individual studies ranging from 0.75 to 1.38³³ (the Baton Rouge and Baytown cohorts were not included in this paper). Updates from two of these studies also suggest a lack of NHL excess.^{25,34} When the authors³³ stratified on year of hire (pre-1950–1950+), they found no difference (SMRs of 0.99 and 0.98, respectively), which is inconsistent with the higher NHL SMR among pre-1950 hires at Baton Rouge (Table 3).

Nervous System Neoplasms

For Baton Rouge, pre-1950 hires have a borderline statistically significant 2.5-fold elevation of “uncertain behavior/unspecified neoplasms of brain/spinal cord” based on seven

deaths (two more since the last update). According to employment histories and “usual occupation” information on death certificates, most were refinery-based and held a variety of maintenance and other jobs. Risk from this disease category was confined to workers hired in the 1940–1944 period, based on five deaths (SMR = 3.64, statistically significant). Baton Rouge also has a borderline nonsignificant, 1.8-fold elevation of brain cancer among pre-1950 hires (14 deaths). Of the 14, occupational information from work histories and death certificates did not reveal patterns in refinery or chemical plant jobs; primary work included five mechanical, six professional/technician, and three process jobs. Among 1950 hires, there were three brain cancer deaths versus 4.12 expected.

In light of recent concern about a brain cancer cluster reported at an Amoco research facility, it should be noted that employment characteristics for the Baton Rouge decedents do not resemble the Amoco cases.³⁵ The brain cancer cases at Amoco were chemical researchers who worked on similar projects in a specific building, from 1970–1976 to the mid-1990s.

In Baytown, males have a borderline significant threefold increase in mortality from “uncertain behavior/ unspecified neoplasms of the brain/spinal cord,” with five observed deaths compared with 1.61 expected. Four of the five decedents were hired before 1950 with an average of 34 years of employment. Work patterns are not suggested, with three of the five decedents having worked in mechanical jobs and the other two as professionals. Brain cancer mortality at Baytown is similar to expected.

Some petroleum worker studies have reported elevated mortality from malignant and nonmalignant brain neoplasms,^{18,19,36–39} but overall there is little support for brain cancer excess,^{10,35} and benign and unspecified brain neoplasms have not been looked at regularly. It has

been suggested that brain tumor elevations may be partially explained by detection bias if employed persons have better access to medical care compared with the general population.⁴⁰

Kidney Cancer

An earlier mortality surveillance study raised sufficient concern about kidney cancer in the Baton Rouge cohort³ to prompt a case–control study. The subsequent investigation did not find a relationship between petroleum-related exposures and deaths from kidney cancer.⁴¹ The study suggested that body mass index was an important determinant, but available data could not fully explain the roles of work versus non-work factors. Subsequent cohort updates show the SMR declining to 1.78 (95% CI = 1.07–2.78) based on 14 deaths⁴ and now to 1.46 (95% CI = 0.90–2.22) based on 21 deaths in Baton Rouge. This downward trend suggests that the elevation, confined to earlier workers, is not continuing. Also, analyses by duration of employment do not show patterns suggestive of work-relatedness.

Mesothelioma and Asbestosis

Twofold elevations in mesothelioma are similar to the previous update, with two and three additional deaths in Baton Rouge and Baytown, respectively. For the eight deaths currently in the Baton Rouge cohort, the underlying cause of death for seven of them is ICD 199.1 (site unspecified); the other is ICD 162.9 (pulmonary). No benign mesotheliomas were found. There were 10 mesothelioma deaths in the Baytown cohort; one death was not counted in the SMR calculation because it was coded as benign (ICD 212.3). The ICD codes for the nine malignant mesothelioma deaths include five 199.1, and one each of 158.9 (peritoneal), 162.9 (pulmonary), 163.9 (pleural), and 195.1 (thorax). The latter case had mesothelioma as a contributory cause of death.

Seven of the eight Baton Rouge decedents were employed between 1937 and 1947, worked 29 to 39 years, had 40- to 54-year latencies, and two of the death certificates indicated prior military service. Work history information on job type and “usual occupation” on death certificates indicates that six of the eight decedents held maintenance jobs, with indications that some spent time as pipefitters and insulators.

Eight of the 10 Baytown decedents were hired in 1945, at ages ranging from 21–33 years. Durations of employment for the 10 decedents range from 26 to 42 years and latencies from 31 to 54 years. Five have military service stated on their death certificates. Work histories and “usual occupation” on death certificates suggests that eight of the 10 had maintenance jobs, with indications that several were pipefitters and insulators.

Regarding asbestosis, Baton Rouge has two deaths from asbestosis, versus 0.63 expected, and Baytown has four deaths from asbestosis versus 0.64 expected (statistically significant). For both locations, decedents were hired before 1950, had primarily maintenance jobs of 25 years or more, with no information on precompany employment.

Certain maintenance jobs such as insulator and pipefitter are recognized as having potentially higher asbestos exposure compared with other plant jobs. Asbestos was used in refineries for thermal insulation and as gasket material and protective screening around welding operations, and most refineries began to replace asbestos with other materials in the mid to late 1960s.⁴² Limited data reported by IARC⁴² suggest that asbestos concentrations in refineries were generally very low compared with industries such as asbestos mining and manufacturing and shipbuilding.

Mesothelioma excesses have been reported in other petroleum worker studies^{19,39,43–47} Two- to fivefold elevations are usually seen, and subco-

horts of maintenance workers have shown greater mesothelioma risk.^{19,48}

Price⁴⁹ examined trends in mesothelioma and estimated that the maximum lifetime risk is experienced by men born in the period 1925–1929. This appears to be the case among Baton Rouge decedents hired before 1950; two deaths have occurred among employees in the 1925–1929 birth cohort (rate is 0.7%), and five deaths have occurred among men born between 1910 and 1924 (rate is 0.2%). In contrast, the Baytown cohort has no mesothelioma deaths among those in the 1925–1929 birth cohort; all deaths are among employees born between 1910 and 1924 (rate is 0.5%).

Price also estimated that peak mesothelioma counts in the United States will occur before the year 2000 based on assumptions of maximum exposure to asbestos in the 1930s to 1960s with highest levels during World War II. According to U.S. incidence rates from SEER through 2001 (<http://seer.cancer.gov/FastStats/>), age-adjusted mesothelioma rates for males appear to be in a plateau and perhaps slightly declining in recent years. However, it is too soon to tell if this is contributing to a long-term trend or is the result of year-to-year fluctuations.

Hypertension With Heart Disease

The statistically significant, 1.58-fold excess of hypertension with heart disease in Baton Rouge employees has no obvious relationship with employment. The majority of decedents were hired before 1950. This condition makes up just 49 of 788 deaths in the larger category of “all heart disease” whose overall SMR is 0.82. Other outcomes related to hypertension are not elevated (eg, ischemic heart disease, strokes, renal disease). In contrast, hypertension with heart disease is significantly low in the Baytown cohort (SMR = 0.26 based on five deaths). It is possible that the elevation at Baton

Rouge is a diagnostic peculiarity. Few other studies have reported findings for this condition, although Satin et al. report a similar increase among pre-1950 hires based on 54 deaths in a refinery worker cohort and the same elevation for hypertension without heart disease.¹⁸

Motor Neuron Disease

The nonsignificant, twofold elevations of motor neuron disease in Baton Rouge and Baytown are based on eight and seven deaths, respectively. All but one of the death certificates indicate ALS, a fatal neurodegenerative disease whose causes are unknown. All but one decedent in each cohort was hired before 1950, with average job durations of more than 30 years, and occupations consisting of a mix of laboratory technicians, mechanical workers, supervisors, and professionals. This diversity of jobs argues against occupational origins of ALS.

ALS findings are not generally reported in petroleum industry studies. A few suspected occupational risks have appeared in the literature (eg, pesticides, electromagnetic fields, solvents, heavy metals), but none of the evidence is conclusive.^{50–54} A recent review of ALS⁵⁵ indicates that up to 10% of ALS cases are familial and the others are sporadic, and that the only established risk factors are age and family history.

Conclusions

The broad surveillance approach has provided reassurance about the overall health status of the Baton Rouge and Baytown cohorts. For a few observed elevations, we conclude: 1) this update and previous focused studies have reduced concern about kidney cancer and CLL at Baton Rouge; 2) mesothelioma should continue to be monitored to see if trends start to decrease; 3) we will increase scrutiny of ALS in subsequent cohort updates and monitor the literature on possible occupational etiologies; and 4) LH malignancy subtypes and brain tumors should continue to be examined carefully in future employee surveillance.

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