

Update of a study of crude oil production workers 1946-94

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Abstract

Objective—To update information on workers in the petroleum industry engaged in the production of crude oil to determine whether the patterns of mortality have changed with 14 additional years of follow up.

Methods—All workers were employed at company production and pipeline locations sometime during 1946-94. The cohort now consists of 24 124 employees with an average of 22 years of follow up.

Results—The overall mortality, and most cause specific mortalities were lower than or similar to those for the general United States population. For white men (81% of the cohort), there were 4361 observed deaths and 5945 expected, resulting in a significantly lower standardised mortality ratio (SMR) of 73. There were significant deficits for all the leading causes of death in the United States including all cancers, cancer of the lung, stroke, heart disease, respiratory disease, and accidents. Slightly increased mortality was found for cancer of the prostate, cancer of the brain and central nervous system, and cancer of other lymphatic tissue. For benign and unspecified neoplasms, the SMR was 152 (95% confidence interval (95% CI) 95 to 230). There was a significant increase for acute myelogenous leukaemia that was restricted to people who were first employed before 1940 and who were employed in production and pipeline jobs for >30 years. Overall mortality patterns for non-white men and women were similar to those for white men. Mortality patterns for white men were also examined by duration of employment, time first employed, and by job group.

Conclusions—The results of the updated study showed a favourable mortality experience for crude oil production workers compared with the United States population.

(*Occup Environ Med* 2000;57:411-417)

Keywords: petroleum industry; occupational cancer; mortality; crude oil

Although numerous epidemiology studies have examined the patterns of mortality in refinery workers and petroleum distribution workers, only one epidemiological study has been carried out on production and pipeline workers whose primary exposure was to crude oil.¹ The report documented the known hazards associated with exposure to crude oil and employ-

ment in production. The cohort comprised more than 11 000 United States workers whose mortality experience was recorded from 1946-80. There were significant deficits in mortality from all causes of death combined, all malignant neoplasms, stroke, arteriosclerotic heart disease, respiratory disease, accidents, and lung cancer. Two causes of death had standardised mortality ratios (SMR) >100, thyroid cancer and benign and unspecified neoplasms; neither SMR was significant.

In this report, the period of observation was extended to the end of 1994, for a maximum follow up of 49 years. These additional 14 years of follow up allowed updated comparisons of additional causes and rates of death among crude oil production workers with those in the general United States population. The objective was to determine whether employment in production and pipeline operations with its potential exposure to crude oil and other possibly hazardous materials has had an effect on the mortality experience of these workers.

Materials and methods

Members of the original study cohort worked at production or pipeline facilities for at least 1 day between 1 January 1946 and 31 December 1980, were employed at one of these facilities for at least 6 months, and were employed by a participating facility either on their last day of employment or on the original study end date (31 December 1980). The study data collection and vital status follow up methods are detailed in Divine and Barron.¹

The current cohort consists of the members of the original cohort plus people who met the same eligibility criteria by 31 December 1994. New members of the cohort who had ever worked at the participating locations were identified with information from the company's computerised personnel system. The work histories for employed members of the original cohort were updated until 31 December 1994 with the computerised personnel system. Information on new cohort members was obtained primarily from the computerised personnel system and from original records if early work history information had not been computerised. Members whose records lacked information on sex or race were assumed for the analysis to be men and white.

Each employee's complete work history was coded. When more than one job was held concurrently, all were coded with the same start and end dates, and the time was divided evenly among the jobs during analysis. Because of the many different jobs, jobs with similar

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Accepted 18 January 2000

Table 1 Mortality study of crude oil workers: total cohort by vital status

Vital status at the end of 1994	White men		Non-white men		White women		Non-white women		Total	
	n	%	n	%	n	%	n	%	n	%
Alive	14520	74.1	1098	91.7	2453	81.5	305	92.4	18376	76.2
Dead	4361	22.3	53	4.4	195	6.5	3	0.9	4612	19.1
Unknown	707	3.6	47	3.9	361	12.0	21	6.7	1136	4.7
Total	19588	100	1198	100	3009	100	326	100	24124	100

responsibilities and potential exposures were grouped into larger categories chosen by a company industrial hygienist most familiar with producing and pipeline operations. These groups account for almost all of the jobs and are the same as those developed and used in the earlier report.¹

The vital status of the cohort was updated to 31 December 1994 from company files when available, and otherwise from the National Death Index (NDI), the Social Security Administration Master Beneficiary Record file, or the Health Care Financing Administration (HCFA). People who were known to be alive in 1979 were assumed to be alive if no death record was found in the NDI to the end of 1994. Copies of the death certificates for deceased employees were obtained from company files when available or from the health departments in the states where the deaths occurred. A trained nosologist coded causes of death listed on the death certificates to the eighth revision of the International Classification of Diseases (ICD-8).

The last date of observation for a cohort member was defined as the date last employed if lost to follow up, the date of death if known, the date of the NDI search if known to be alive in 1979 or later, the date of the HCFA search if

HCFA indicated the person was still alive, the date of the personnel system search for people still receiving pension payments, or the current study end date (31 December 1994) for those known to be alive as of 1 January 1995.

Patterns of mortality were analysed with the computer program developed by Monson.²

The program uses the observed and expected numbers of deaths for specific causes to calculate SMRs with the United States population as the comparison group and 95% confidence intervals (95% CIs) assuming a Poisson distribution for the observed frequency in the numerator of the SMR. Person-years of observation were counted from the date the employee accumulated 6 months of employment or the study start date, whichever came last, until the date last observed.

Total mortality was examined by race and sex. Because of the few non-white women (1.4% of the total cohort), patterns of mortality for all women were examined with the United States mortalities for white women as the comparison. Also, for white men, patterns of mortality were examined by duration of employment, by time first employed, and by job group. The small numbers in the other race-sex groups preclude making similar subgroup analyses.

Table 2 Study of crude oil workers: SMRs for selected causes of death, by sex and race, 1946-94

Cause of death (ICDA-8)	White men n=19588 p-y=434397			Non-white men n=1198, p-y=17413			All women n=3338, p-y=51763		
	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI
All causes	4361	73	71 to 76	53	46	35 to 61	198	89	77 to 102
All cancers (140-209)	1080	83	78 to 88	15	83	47 to 138	74	103	81 to 129
Digestive system (150-159)	252	73	64 to 83	2	40	5 to 145	19	130	78 to 202
Oesophagus (150)	18	59	35 to 93	0	0	0 to 319	0	0	0 to 649
Stomach (151)	37	66	47 to 91	1	115	2 to 641	2	129	14 to 464
Large intestine (153)	95	77	62 to 94	0	0	0 to 319	13	197	105 to 337
Liver (155-156)	22	94	59 to 142	0	0	0 to 691	0	0	0 to 304
Pancreas (157)	62	94	72 to 120	0	0	0 to 471	4	133	36 to 341
Larynx (161)	6	35	13 to 77	0	0	0 to 945	1	437	6 to 2430
Lung (162)	347	80	72 to 89	8	132	57 to 259	12	93	48 to 162
Skin (172-173)	22	86	54 to 131	0	0	0 to 1733	1	79	1 to 439
Breast (174)							15	90	50 to 149
Cervix (180)							4	157	42 to 402
Uterus (181)							2	109	12 to 92
Prostate (185)	135	119	100 to 141	1	131	2 to 731			
Bladder (188)	30	78	52 to 111	1	669	9 to 3723	0	0	0 to 575
Kidney (189)	16	51	29 to 82	1	279	4 to 1553	2	178	20 to 643
Brain and CNS (191-192)	37	110	77 to 152	0	0	0 to 1025	2	98	11 to 352
Thyroid (193)	4	175	47 to 449	0	0	0 to 15268	0	0	0 to 2004
Lymphatic and haematopoietic (200-209)	116	93	77 to 112	1	60	1 to 332	3	48	10 to 140
Lymphosarcoma and reticulosarcoma (200)	10	56	27 to 104	0	0	0 to 2580	0	0	0 to 471
Hodgkin's disease (201)	5	50	16 to 118	0	0	0 to 2369	1	201	3 to 1120
Leukaemia (204-207)	49	97	72 to 129	0	0	0 to 575	0	0	0 to 154
Other lymphatic tissue (202,203,208)	45	103	75 to 138	1	138	2 to 770	2	81	9 to 291
Benign neoplasms (210-239)	22	152	95 to 230	0	0	0 to 1655	1	101	1 to 564
Diabetes mellitus (250)	36	41	29 to 57	0	0	0 to 201	2	38	4 to 138
Arteriosclerotic heart disease (410-413)	1489	74	70 to 77	6	40	15 to 88	35	72	50 to 100
Vascular lesions of CNS (430-438)	282	74	66 to 84	3	59	12 to 173	14	88	48 to 147
Non-malignant respiratory disease (460-519)	299	68	60 to 76	2	38	4 to 137	8	59	25 to 116
Pneumonia (480-486)	100	63	51 to 77	0	0	0 to 129	3	59	12 to 173
Cirrhosis of liver (571)	38	32	23 to 44	1	20	0 to 113	4	84	23 to 215
All external causes (800-998)	283	59	53 to 67	15	49	27 to 80	23	139	88 to 209

p-y = Person years.

Table 3 Study of crude oil workers: SMRs for selected causes of death, by duration of employment, 1946-94

Cause of death (ICDA-8)	White men employed <5 y (n=5415) p-y=125958			White men employed 5-9 y (n=4217) p-y=99432			White men employed 10-19 y (n=3438) p-y=80978			White men employed ≥20 y (n=6518) p-y=128029		
	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI
All causes	412	71	64 to 78	304	63	56 to 71	718	72	66 to 77	2927	75	72 to 78
All cancers (140-209)	95	76	61 to 93	56	54	41 to 71	175	83	71 to 97	754	87	81 to 93
Digestive system (150-159)	27	93	61 to 135	13	51	27 to 87	42	74	53 to 101	170	73	62 to 85
Oesophagus (150)	4	133	35 to 341	0	0	0 to 150	1	20	0 to 113	13	65	34 to 110
Stomach (151)	3	69	14 to 203	3	71	14 to 208	4	39	10 to 101	27	72	48 to 105
Large intestine (153)	7	67	27 to 140	5	56	18 to 131	15	79	44 to 130	68	79	62 to 100
Pancreas (157)	5	87	28 to 204	4	81	21 to 208	11	102	51 to 183	42	94	68 to 127
Larynx (161)	0	0	0 to 280	0	0	0 to 286	1	35	0 to 198	5	44	14 to 102
Lung (162)	29	68	45 to 98	13	38	20 to 65	62	91	70 to 117	243	85	74 to 96
Skin (172-173)	5	121	39 to 284	4	129	34 to 330	5	104	33 to 243	8	59	25 to 117
Prostate (185)	5	87	28 to 204	3	54	10 to 159	13	91	48 to 156	114	129	107 to 155
Bladder (188)	2	86	9 to 313	2	95	10 to 343	1	17	0 to 99	25	87	56 to 129
Kidney (189)	1	30	0 to 168	1	38	0 to 213	3	55	11 to 162	11	54	27 to 97
Brain and CNS (191-192)	4	76	20 to 196	4	100	26 to 256	10	147	70 to 270	19	108	65 to 168
Lymphatic and haematopoietic (200-209)	8	54	23 to 107	10	86	41 to 159	19	89	53 to 139	79	102	81 to 128
Lymphosarcoma and reticulosarcoma (200)	2	111	12 to 401	0	0	0 to 235	3	83	16 to 243	5	46	15 to 107
Hodgkin's disease (201)	1	50	0 to 278	0	0	0 to 249	0	0	0 to 159	4	97	26 to 248
Leukaemia (204-207)	3	52	10 to 153	3	66	13 to 194	8	94	40 to 185	35	111	77 to 154
Other lymphatic tissue (202, 203, 208)	2	40	4 to 147	7	184	73 to 379	5	75	24 to 176	31	109	74 to 155
Benign neoplasms (210-239)	2	117	13 to 424	1	71	0 to 397	8	296	127 to 583	11	127	63 to 227
Diabetes mellitus (250)	2	24	2 to 88	2	28	3 to 104	5	34	11 to 81	27	47	31 to 68
Arteriosclerotic heart disease (410-413)	96	68	55 to 83	103	77	63 to 93	262	78	69 to 88	1028	73	68 to 77
Vascular lesions of CNS (430-438)	10	49	23 to 91	10	48	23 to 88	43	73	53 to 98	219	78	68 to 89
Non-malignant respiratory disease (460-519)	18	61	36 to 97	21	79	49 to 121	33	53	36 to 74	227	70	61 to 80
Pneumonia (480-486)	6	59	21 to 129	2	21	2 to 78	10	43	20 to 79	82	70	56 to 87
Cirrhosis of liver (571)	8	48	20 to 95	7	51	20 to 106	6	23	8 to 50	17	27	16 to 43
All external causes (800-998)	93	71	58 to 88	35	44	30 to 61	63	61	47 to 79	92	56	45 to 68

p-y = Person years.

The SMRs for white men for mesothelioma, non-Hodgkin's lymphoma, leukaemia by cell type, multiple myeloma, and all brain tumours, which are not available in Monson's program, were calculated in the same manner as in an earlier study of refinery, petrochemical, and research workers using the same mortalities.³

Results

There were 24 124 people included in the study cohort (table 1) with 503 573 person-years of observation. Eighty six per cent of the cohort members were men, and 94% of the men were white. Ninety per cent of the women were white. Seventy six per cent of the total cohort were still alive, only 4.7% were lost to follow up, and death certificates were obtained for all but 1.9% of the known deaths. Because the cohort overwhelmingly consisted of white men (81%), the following descriptive statistics refer only to that group. Twenty two per cent of the cohort was still employed on 31 December 1994, and 36% were retired (33.7%) or on permanent disability (2.2%). Thirty three per cent were employed by the company at study locations for ≥20 years. Thirty five per cent of the cohort was born before 1930, and more than 23% was first employed before 1950. Sixty eight per cent of the employees who had died were ≥65 years of age at the time of death, and over half the deaths occurred since the previous study end date of 31 December 1980.

Table 2 shows the patterns of mortality by race and sex. For white men, 5945 deaths were expected, and only 4361 were observed giving a significant all causes SMR of 73. There were also significant deficits for all cancers combined (SMR 83), digestive system cancers (SMR 73), lung cancer (SMR 80), arterioscle-

rotic heart disease (SMR 74), stroke (SMR 74), non-malignant respiratory disease (SMR 68), and all external causes (SMR 59). For cancer of the prostate, cancer of the brain and central nervous system (CNS), and cancer of other lymphatic tissue, the observed number of deaths was similar to the expected number, but the SMRs were slightly increased, and SMRs >100 were also found for benign and unspecified neoplasms (SMR 152) and thyroid cancer (SMR 175).

For non-white men, a total of 114 deaths were expected and 53 were observed (SMR 44). There were deficits for many major causes of death such as all cancers combined (SMR 83), cancer of the digestive system (SMR 40), lymphohaematopoietic cancer (SMR 60), arteriosclerotic heart disease (SMR 40), stroke (SMR 59), non-malignant respiratory disease (SMR 38), and all external causes (SMR 49). There were non-significantly increased SMRs for cancer of the lung (SMR 132) and motor vehicle accidents (SMR 126). For all women, there were 198 deaths and 223 were expected giving a significant deficit for the all causes SMR of 89. There were deficits in mortality from arteriosclerotic heart disease (SMR 72), vascular lesions of CNS (SMR 88), and respiratory disease (SMR 59). For all cancers combined, the observed and expected numbers of deaths were similar. The SMR for cancer of the colon was significantly increased (SMR 197), and there were non-significantly increased SMRs for cancers of the digestive system (SMR 130), cancer of the pancreas (SMR 133), cancer of the cervix (SMR 157), cancer of the ovary (SMR 179), motor vehicle accidents (SMR 138), and suicide (SMR 206).

All further results apply only to the sub-cohort of white men. Table 3 shows the patterns of mortality by duration of employment, and

Table 4 Study of crude oil workers: SMRs for selected causes of death, by time first employed, 1946–94

Cause of death (ICD-8)	White men first employed <1940 (n=2162) p-y=74085			White men first employed 1940– (n=2352) p-y=93985			White men first employed ≥1950 (n=15074) p-y=266345		
	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI
All causes	1817	73	70 to 77	1345	75	71 to 80	1199	71	67 to 75
All cancers (140–209)	407	84	76 to 92	351	86	77 to 95	322	79	70 to 88
Digestive system (150–159)	89	63	50 to 77	82	77	61 to 95	81	85	68 to 106
Oesophagus (150)	5	47	15 to 110	4	42	11 to 107	9	87	40 to 166
Stomach (151)	16	62	35 to 100	11	66	33 to 117	10	76	36 to 139
Large intestine (153)	31	62	42 to 88	36	92	65 to 128	28	80	53 to 116
Pancreas (157)	23	89	56 to 134	20	96	59 to 148	19	98	59 to 153
Larynx (161)	3	47	9 to 136	3	55	11 to 162	0	0	0 to 71
Lung (162)	111	78	64 to 94	123	88	73 to 105	113	76	62 to 91
Skin (172–173)	3	44	9 to 129	6	90	33 to 196	13	108	58 to 185
Prostate (185)	72	128	100 to 161	43	119	86 to 160	20	96	58 to 148
Bladder (188)	18	96	57 to 152	6	49	18 to 108	6	77	28 to 168
Kidney (189)	4	37	10 to 94	6	61	22 to 134	6	55	20 to 120
Brain and CNS (191–192)	12	134	69 to 235	9	91	42 to 173	16	108	61 to 175
Lymphatic and haematopoietic (200–209)	48	109	80 to 145	36	95	67 to 132	32	75	51 to 105
Lymphosarcoma and reticulosarcoma (200)	4	56	15 to 143	3	54	11 to 157	3	60	12 to 175
Hodgkin's disease (201)	2	67	8 to 243	2	71	8 to 258	1	24	0 to 135
Leukaemia (204–207)	24	126	81 to 187	13	86	46 to 148	12	74	38 to 129
Other lymphatic tissue (202,203,208)	16	116	66 to 189	14	105	57 to 175	15	90	51 to 149
Benign neoplasms (210–239)	8	144	62 to 284	6	134	49 to 291	8	179	77 to 353
Diabetes mellitus (250)	21	59	36 to 90	10	38	18 to 70	5	20	6 to 47
Arteriosclerotic heart disease (410–413)	645	70	65 to 75	499	80	73 to 87	345	73	65 to 81
Vascular lesions of CNS (430–438)	162	78	67 to 91	89	81	65 to 100	31	50	34 to 70
Non-malignant respiratory disease (460–519)	163	80	68 to 93	83	59	47 to 74	53	55	41 to 72
Pneumonia (480–486)	63	77	59 to 99	25	53	34 to 78	12	40	21 to 70
Cirrhosis of liver (571)	7	21	9 to 44	10	29	14 to 53	21	41	25 to 63
All external causes (800–998)	49	46	34 to 60	64	59	46 to 76	170	65	56 to 76

p-y = Person years.

table 4 shows them by time first employed: before 1940, 1940–1949, and after 1949. The patterns were similar for all of the groups regardless of duration or time of employment with significant deficits for most major causes of death. The SMR for cancer of the prostate for those employed ≥20 years was significantly increased (SMR 129) as was the SMR for benign and unspecified neoplasms for those employed 10–20 years (SMR 296). For the groups by time first employed, there were no significant increases; however, mortality from cancer of the prostate and cancer of other lymphatic tissue was increased in the two earliest employment groups, mortality from leukaemia was increased in the earliest employment group, and mortality from benign and unspecified neoplasms was increased in all three groups. Trends by duration of employment and by time first employed were examined. The only significant trends were for external causes of death and for cancers of the digestive system by time first employed. There was no reason to think that these increases with later time of employment are likely to be related to employment.

Tables 5 shows the patterns of cancer mortality for those employed in the largest job groups for at least 5 years. These groups all had patterns of mortality similar to those seen for the overall cohort with deficits for the leading causes of death. For those employed in jobs with little or no potential for exposure such as managers, supervisors, and office workers, there was a significantly increased SMR for cancer of the brain and CNS (SMR 183). This group also showed non-significantly increased SMRs for liver cancer, prostate cancer, cancer of other lymphatic tissue, and benign and unspecified neoplasms. For those employed as pumpers in production, there were increased

SMRs for cancer of the prostate, liver cancer, and benign and unspecified neoplasms. For those employed in maintenance, there were significantly increased SMRs for cancer of the prostate and benign and unspecified neoplasms. There were non-significant increases for leukaemia and cancer of other lymphatic tissue. For roustabouts (semi-skilled oil field labourers), there were increased SMRs for cancer of the prostate, liver cancer, benign and unspecified neoplasms, and cancer of other lymphatic tissue. For people employed in pipeline field jobs, there were non-significantly increased SMRs for cancer of the skin, cancer of the prostate, leukaemia, cancer of other lymphatic tissue, and benign and unspecified neoplasms.

There were only three deaths from mesothelioma in the cohort of white men and 8.2 expected for an SMR of 37. These decedents were first employed before 1 January 1960. Thirteen of the deaths from benign and unspecified brain neoplasms were brain tumours and 8.3 deaths were expected for an SMR of 135 (95% CI 84 to 207). When these were combined with the deaths from cancer of the brain and CNS, there were 50 observed deaths and 41.8 expected from all brain tumours for an SMR of 119 (95% CI 88 to 157).

Table 6 shows the results of the analyses of specific lymphohaematopoietic cancers. The number of observed deaths from non-Hodgkin's lymphoma (lymphosarcoma, reticulum cell sarcoma, and lymphoma combined) was significantly less than expected with an SMR of 69. There were slightly more deaths from multiple myeloma than expected with an SMR of 122. The SMRs were calculated for the cell type specific leukaemias for categories where at least three deaths occurred. Mortality

Table 5 Study of crude oil workers: SMRs for selected causes of death, by job, 1946–94

Cause of death (ICDA-8)	Unexposed category, >5 y White men (n=5042) p-y=98931			Producing, pumper, >5 y White men (n=3456) p-y=79128			Craft, maintenance, >5 y White men (n=2342) p-y=43798			Producing, roustabout, >5 y White men (n=3820) p-y=96183			Pipeline, field, >5 y White men (n=1456) p-y=39478		
	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI	Observed deaths	SMR	95% CI
All causes	1046	67	63 to 71	1379	77	73 to 81	684	76	70 to 81	1260	76	72 to 80	711	72	67 to 78
All cancers (140–209)	293	80	71 to 90	322	84	75 to 93	173	86	74 to 100	310	84	75 to 94	175	84	72 to 98
Digestive system (150–159)	73	78	61 to 98	67	64	49 to 81	30	56	38 to 80	61	62	48 to 80	38	67	47 to 92
Stomach (151)	13	91	48 to 155	9	50	23 to 96	3	34	7 to 101	7	44	18 to 91	5	52	17 to 122
Large intestine (153)	29	84	56 to 121	22	58	37 to 88	13	67	35 to 114	22	62	39 to 94	20	97	59 to 150
Liver (155–156)	7	110	44 to 226	10	136	65 to 250	1	27	0 to 150	9	133	61 to 252	0	0	0 to 92
Pancreas (157)	15	82	46 to 135	19	95	57 to 149	6	58	21 to 127	17	90	53 to 145	8	74	32 to 146
Lung (162)	85	67	54 to 83	109	88	73 to 107	55	83	62 to 108	118	96	80 to 115	50	76	57 to 101
Skin (172–173)	6	85	31 to 185	5	78	25 to 182	0	3	45 to 9 to 132	4	127	34 to 324	4	127	34 to 324
Prostate (185)	34	111	77 to 155	46	122	89 to 162	29	154	103 to 221	38	114	81 to 156	28	130	86 to 188
Bladder (188)	10	97	47 to 179	12	95	49 to 165	5	79	25 to 184	9	80	36 to 152	5	70	22 to 163
Kidney (189)	5	56	18 to 130	5	55	18 to 129	6	125	46 to 273	4	45	12 to 116	1	21	0 to 116
Brain and CNS (191–192)	17	183	107 to 293	9	107	49 to 203	3	65	13 to 190	6	67	24 to 146	4	92	25 to 234
Lymphatic and haematopoietic (200–209)	38	112	79 to 154	27	77	51 to 112	24	130	84 to 194	33	96	66 to 135	25	131	85 to 194
Lymphosarcoma and reticulosarcoma (200)	2	44	5 to 159	1	19	0 to 106	2	75	8 to 270	2	40	4 to 144	2	68	8 to 246
Hodgkin's disease (201)	2	88	10 to 318	1	43	1 to 239	1	83	1 to 462	2	80	9 to 290	2	155	17 to 561
Leukaemia (204–207)	12	90	46 to 156	13	90	48 to 154	11	147	73 to 263	17	123	71 to 196	10	127	61 to 233
Other lymphatic tissue (202,203,208)	20	156	95 to 241	11	91	45 to 163	9	137	62 to 260	12	99	51 to 173	9	140	64 to 265
Benign neoplasms (210 to 239)	5	133	43 to 309	5	122	39 to 284	6	280	102 to 610	3	75	15 to 218	6	259	95 to 564

p-y = Person years.

from acute lymphocytic leukaemia was the same as expected (SMR 104), and there was a deficit for chronic lymphocytic leukaemia (SMR 52). Mortality from chronic myelogenous leukaemia was essentially the same as expected (SMR 94). For acute leukaemia and for unspecified leukaemia, mortality was non-significantly increased (SMRs 150 and 175, respectively).

However, there was a significant excess of deaths from acute myelogenous leukaemia (SMR 192) which was based on 16 deaths. The increase was restricted to those who were first employed before 1940 (SMR 374), and who were employed in production and pipeline jobs for at least 20 years (SMR 265). An examination of the work histories for these workers showed that most were employed as roustabouts (SMR 276), pumpers (all were also employed as roustabouts, SMR 280), or pipeline field personnel (SMR 247). These were the most common jobs for most of this cohort.

Table 6 Study of crude oil workers: SMRs for lymphohaematopoietic cancer, white men, 1946–94

	Observed	Expected	SMR	95% CI
Non-Hodgkin's lymphoma	32	45.9	69	47 to 98
Multiple myeloma	23	18.7	122	77 to 184
Leukaemia	49	50.3	97	72 to 129
Acute lymphocytic leukaemia (ALL)	3	2.9	104	20 to 305
Chronic lymphocytic leukaemia (CLL)	5	9.6	52	16 to 121
Acute myelogenous leukaemia (AML)	16	8.3	192	110 to 313
Ever employed as roustabout	12	4.3	276	142 to 482
Ever employed as pumper	9	3.2	280	127 to 531
Ever employed in maintenance	4	2.7	148	39 to 380
Ever employed in pipeline field	4	1.6	247	66 to 632
First employed <1940	11	2.9	374	186 to 670
First employed 1940 to 9	2	2.6	77	8 to 280
First employed ≥1950	3	2.9	105	21 to 307
Employed <20 y	3	3.4	88	17 to 257
Employed ≥20 y	13	4.9	265	141 to 553
Chronic myelogenous leukaemia (CML)	6	6.3	94	34 to 205
Acute unspecified leukaemia (AUL)	4	2.7	150	40 to 384
Unspecified leukaemia (UL)	6	3.4	175	64 to 382

Discussion

The patterns of mortality found in this updated study of workers engaged in the production of crude oil were similar to those found in the first analysis of this cohort.¹ There was a more favourable mortality experience for the cohort compared with the United States population, and there were deficits for all of the leading causes of death in the United States including all cancers, heart disease, stroke, lung cancer, respiratory disease, and external causes of death. The only increased causes of death found in the general analysis of the cohort of white men were for thyroid cancer and for benign and unspecified neoplasms; these increases were not significant. The increase in thyroid cancer was found in the earlier analysis of this cohort,¹ based on only four deaths. No additional deaths from thyroid cancer have occurred since 1980, and the SMR has decreased from 342 to 175. Over half of the deaths from benign and unspecified neoplasms were from benign and unspecified tumours of the brain. When these brain tumours were examined together with the brain cancers, the overall brain tumour SMR was only slightly increased. There was a significantly increased SMR for cancer of the brain and CNS but only for the unexposed group and this might be due to more complete diagnosis in this group.

The results of this analysis are consistent with those found in other studies of petroleum industry workers. In general, these studies have all shown a strong healthy worker effect with numerous significant deficits for most leading causes of death and few significant increases. The study results are also consistent with the conclusions of a meta-analysis of specific cancers in workers employed in various segments of the petroleum industry.⁴ Wong and Raabe found that petroleum industry

workers had a significantly low cancer mortality for all cancer sites combined and for cancers of the digestive system, stomach, and lung. Similar deficits were found in this study. They also found mortality to be similar to that for the United States population for cancers of the skin, brain, pancreas, prostate, and kidney. Small increases for these causes occurred among various subgroups in this study, but the increases were not consistent nor were they linked with increasing duration of employment in the job subgroups.

The slight excess of brain tumours is not uncommon in studies in the petroleum industry. A similar small increase in brain tumours has been reported by Divine and Hartman³ in a large study of refinery, petrochemical, and research workers and by Satin *et al*⁵ in a large cohort refinery study. Satin *et al* also noted that the brain is often a metastatic cancer site. A previous report on brain tumours in this same refinery cohort⁶ discussed the problems associated with diagnostic errors of brain tumours that may lead to increases in employed populations.

The meta-analysis of studies of petroleum industry workers by Wong and Raabe⁴ suggested that some petroleum workers, especially those first employed before 1940, might have an increased risk of leukaemia. A subsequent meta-analysis of leukaemia cell type specific results in petroleum industry cohorts⁷ did not show increased SMRs for acute myelogenous leukaemia or any of the other leukaemia cell types. However, a case-control study of leukaemia among petroleum workers at the Unocal Corporation⁸ found myelogenous leukaemia to be associated with work related to production in the oil and gas division of the company (odds ratio (OR) 2.0). The association was even stronger for those employed for >30 years in activities related to production (OR 3.9) and also for those with acute myelogenous leukaemia (OR 2.8). The researchers also found a consistent trend of increasing ORs with increasing duration of employment.

The analysis of leukaemia by cell type for the present cohort showed an increased SMR for acute myelogenous leukaemia among white men which was restricted to those people first employed before 1940 with at least 30 years of employment in production and pipeline jobs. These results are, therefore, similar to those found in the Unocal study. No information was available to describe oil field exposures in the early 1900s, but others have reported⁸ that personal hygiene was poor and that some oil field workers had heavy exposure to crude oil. In particular, there is no information about the benzene content of the crude oil over time or oil fields. Furthermore, it is not known whether benzene was used as a solvent or cleaning agent in oil field work in the past. Increased risk of leukaemia (for all cell types except chronic lymphocytic leukaemia) has also been associated with exposure to large doses of ionising radiation. Although there is potential for exposure to naturally occurring radioactive

material in the production fields, there is no consistent evidence that the low levels of natural background radiation increase the risk of leukaemia.⁹

This cohort study of crude oil production workers has many strengths. The cohort includes >24 000 people whose mortality experience has been studied for 49 years (1946–94). Vital status is known for >95% of the entire cohort, and for >88% of the women. Only 89 of 4612 death certificates could not be found (1.9%). Also, the study includes information about the cohort members' complete work histories, and numerous analyses by job were performed.

The study does have its limitations. As with other mortality studies, it has the problems inherent with using the death certificate diagnosis for the cause of death—for example, diagnostic accuracy and specificity, and comparability of ICD codes over time. Mortalities vary over time and geographical location. Because this study covered many locations across the United States, the United States mortalities were used for comparison. Although the cohort was large, many of the analysis subgroups were small. Information about the specific chemicals associated with the jobs is not available, nor is there any industrial hygiene sampling data covering the first 35 years of the study. Jobs and units with similar responsibilities were grouped together for the analyses as a surrogate for exposures, but provide little information to link exposure and outcome.

Conclusion

The results of the update of workers engaged in the production of crude oil show a favourable mortality experience for these employees compared with the general United States population. Most causes of death in the study show either similar mortality or a significant deficit when compared with the general population. The overall 27% deficit in total deaths translates into an increased life expectancy of 3.7 years¹⁰ for these workers compared with white men in the general population of the United States. The few increases in various causes of death were not consistently found across jobs with similar exposures, nor did most rise with increasing duration of employment.

There was an apparent excess of death for some of the leukaemia cell type specific causes of death, including acute myelogenous leukaemia. The increased mortality from acute myelogenous leukaemia was found only in those people who were first employed before 1940 and who were employed in the production of crude oil for >30 years.

1 Divine BJ, Barron V. Texaco mortality study III. A cohort study of producing and pipeline workers. *Am J Ind Med* 1987;11:189–202.

2 Monson RR. Analysis of relative survival and proportionate mortality. *Computer Biomed Res* 1974;7:325–32.

- 3 Divine BJ, Hartman CM, Wendt JK. An update of the Texaco mortality study 1947–1993: part II. Analysis of specific causes of death for white males employed in refining, research, and petrochemicals. *Occup Environ Med* 1999;**56**:174–80.
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- 10 Tsai SP, Hardy RJ, Wen CP. The standardized mortality ratio and life expectancy. *Am J Epidemiol* 1992;**135**:824–31.

Vancouver style

All manuscripts submitted to *Occup Environ Med* should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style.)

Occup Environ Med, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style. The style (described in full in the *JAMA*[1]) is intended to standardise requirements for authors, and is the same as in this issue.

References should be numbered consecutively in the order in which they are first mentioned in the text by Arabic numerals on the line in square brackets on each occasion the reference is cited (Manson[1] confirmed other reports[2][3][4][5]). In future references to papers submitted to *Occup Environ Med* should include: the names of all

authors if there are three or less or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter. Titles not in *Index Medicus* should be given in full.

Examples of common forms of references are:

- 1 International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomed journals. *JAMA* 1993;**269**:2282–6.
- 2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687–90.
- 3 Weinstein L, Swartz MN. Pathogenic properties of invading micro-organisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology, mechanisms of disease*. Philadelphia: W B Saunders, 1974:457–72.