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# A Retrospective Mortality Study Within Operating Segments of a Petroleum Company

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This retrospective mortality study was conducted among 34,597 oil industry workers in diverse operating segments. Employees were traced through Statistics Canada, and overall mortality (SMR = 0.85) was lower than general population rates and similar to other petrochemical cohorts. The most notable finding was a significant excess of malignant melanoma [observed deaths (N) = 16, SMR = 1.87, 95% CI = 1.07, 3.04], which concentrated among upstream workers (N = 6, SMR = 6.00, 95% CI = 2.19, 13.06), and was directly related to employment duration and latency. Specific substances or hydrocarbon (HC) streams could not be implicated, although possible explanations include dermal HC exposure, ultraviolet light exposure, or a synergistic effect between these two factors.

Marketing/transportation workers showed a non-significant excess of multiple myeloma (SMR = 1.81), which was also related to employment duration, latency, and commencement of employment before 1950. Lymphatic cancer, skin cancer, and kidney cancer mortality was not elevated in refinery workers, a finding at odds with some previous refinery worker studies.

Although the malignant melanoma and possibly the multiple myeloma mortality patterns are consistent with an occupational link, further studies are needed to investigate the relationship of these diseases with particular exposures. 

y words: hydrocarbon, oil, gasoline, malignant melanoma, multiple myeloma, aortic aneurysm, cancer, occupational exposures, refinery workers, female mortality

## INTRODUCTION

Most studies of the oil industry have been restricted to refinery workers [Delzell et al., 1988; Wong and Raabe, 1989] and have not examined other operating segments such as marketing/distribution, pipeline, or production workers. Exposures are clearly different for workers in different petroleum operating segments [Runion,

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1988]. Only Divine and Barron [1987] and Rushton and Alderson [1983] have studied these latter operating segments.

The International Agency for Research on Cancer (IARC) has recently classified refinery operations as probably carcinogenic to humans [IARC, 1989] based on mortality findings for leukemia and skin cancer. Kidney cancer is also an issue, due to findings for wholly vaporized unleaded gasoline in male rats [Kitchen, 1984], but this may be due to a species and sex-specific protein not present in humans [Health Effects Institute, 1988].

The major objective of this study was to examine the mortality experience of a large Canadian cohort of oil industry workers in the context of other similar studies. In particular, the risk of leukemia and other lymphatic cancers, malignant melanoma, and kidney cancer was focused upon. A second study objective was to describe mortality in relation to the different exposures prevalent in operating segments within the petroleum industry. A subsequent report will focus on hydrocarbon exposure.

## METHODS

### Population Definition

This study is a retrospective cohort mortality study. Some study subjects were previously studied by Hanis et al. [1979]. The study population consists of a) all active employees and living annuitants (i.e., retirees) on 1 January 1964 and b) all new regular employees hired between 1 January 1964 and 31 December 1983. One year of work was necessary before an employee, male or female, was included in the cohort. This study population differed from that of Hanis et al. [1979] in that all employees are included instead of males only, and a 1 year employment criterion is imposed in lieu of 5 years for terminated workers and 1 year for active workers.

### Work History Summarization

Work histories for each cohort member were obtained from computer records, generally available from 1960–1964[1] forward. For employees who worked before this time (55%), the first computerized job entry was extrapolated back to their employment date. For annuitants who terminated before 1960–1964, the last job was coded and is extrapolated back to the employment date. The work histories were used to derive operating segment (e.g., marine, marketing, etc.) and presumed exposure to hydrocarbons (HCs) for each employee.

### Operating Segment Definition

Three experienced industrial hygienists reviewed all unique location/department combinations from the work histories. They assigned each entry to one of the following segments: a) refinery, b) agricultural chemicals, c) petrochemical, d) marketing-transportation, e) marine, f) exploration/drilling/production, g) pipeline, h) coal and minerals, i) office, j) fabricated building products subsidiary, and k) other/unknown. These were chosen to exploit the differences in exposure patterns particular to each segment. For example, pipeline workers and drilling workers can be dermally

[1]1960–1964 are approximate dates for the start of work histories. A small percentage of employees had computerized work history before this date, while another very small percentage started after 1964.

exposed to crude oil and drilling muds. Most marketing terminal workers are not exposed to crude oil but can be exposed to finished petroleum products on an intermittent basis, most likely through inhalation. Refinery workers can be exposed to a wider range of hydrocarbons including crude oil, intermediate streams associated with processes such as alkylation or reforming, and/or finished product [see Runion, 1988 for more detail].

### Hydrocarbon Exposure Definition

Eight industrial hygienists supplied a score for exposure to HCs for each unique work history entry in their region of responsibility. Scores were defined as exposed to HC, not exposed to HC, and unknown. This resulted in a matrix of scores for each location/job/department which was extrapolated to each cohort member. HC exposure represents dermal and inhalation exposure to many different streams and/or products, and includes combustion products. For this paper, analyses were conducted on an ever/never exposed basis. If an employee was ever assigned to a job/location/department combination deemed exposed, all subsequent person-years are counted as exposed. For most analyses, the HC-exposed subcohort shows mortality patterns similar to that of all workers. For brevity, we will not routinely present results for the HC-exposed subcohort, but will note where mortality patterns in this group are different from the total cohort.

### Mortality Tracing

Mortality information was obtained through three sources: a) the Statistics Canada (SC) Mortality Data Base; b) internal data sources (including provincial registrars used in the previous study); and c) the National Death Index. Records on all employees not actively employed on 31 December 1983, nor receiving company benefits were sent to Statistics Canada (SC).

SC identified 3,764 total deaths (96.3% of the total), and the U.S. National Death Index (NDI) also identified 19 deaths (0.5%). Another 128 decedents were identified through company sources or the first study.

Company records for five employees indicated that they were alive and receiving benefits, yet they were identified as decedents by SC. Two of these potential decedents with relatively low SC matching scores [see Smith and Newcombe, 1980] were counted as withdrawals; their date last employed was counted as the date last observed alive. Thus, of the 3,764 deaths identified by SC, 3,762 were counted as decedents.

Employee records not matched to SC or NDI files were assumed alive until the end of the follow-up period. The validity of this critical assumption was assessed previously [Schnatter et al., 1990]. This test showed that SC blindly ascertained 97.6% of the known Canadian deaths from this cohort. Importantly, there were no strong determinants of death ascertainment such as province or age of death, indicating a uniform underascertainment of 2.4%. This number provides the best estimate of negative bias on the SMRs due to death under ascertainment.

### Determination of Underlying Cause of Death

The revision of the International Classification of Diseases (ICD) coding scheme in effect at the time of death was used to assign the underlying cause of death. For the 3,762 decedent records in which an SC death code was available, it was used.

TABLE I. Total Cohort of All Operating Segments in a Petroleum Company by Gender and Vital Status on 12/31/83

| | Vital status | | | |
|---|---|---|---|---|
| Gender | Alive | Dead | Withdrawn | Total |
| Males | 22,644 | 3,702 | 2 | 26,348 |
| Females | 8,042 | 207 | 0 | 8,249 |
| Total | 30,686 | 3,909 | 2 | 34,597 |

For the 128 records in which only a code from the first study was available, it was used. For the 19 death certificates obtained through NDI, a certified nosologist determined the underlying cause of death. Records for 39 (1.0%) decedents with no underlying cause of death are included as deaths for total mortality but are not assigned to a cause specific category. Owing to the presence of asbestos and vinyl chloride monomer at particular study locations, an SC-appointed nosologist reviewed all death certificates with codes in the Appendix for any mention of mesothelioma and/or angiosarcoma.

### Mortality Analyses

Employees contributed person-years from 1 January 1964 or date of employment plus 1 year, whichever was later. Person-years were enumerated via the Monson program [Monson, 1974] to the earliest of the following dates: date of death, the end of the follow-up period (31 December 1983), or date of last employment for employees designated as withdrawals. Canada's Laboratory Centre for Disease Control (LCDC) supplied mortality rates and ICD/LCDC rate translation tables. If an employee *ever* worked in one of the operating segments, subsequent person-years and mortality are counted in that segment. Person-years can be counted in more than one segment. This is generally preferred to other strategies which examine most frequent, first, or last assignment [Checkoway et al., 1989], since it maximizes the person-years in each segment. However, this strategy can lead to misclassification for short-term employment in an operating segment.

Expected deaths were calculated by multiplying Canadian population mortality rates by the study population person-year distributions specific for gender, quinquennia of age, and calendar period. SMRs were computed provided that either the observed or expected number of deaths was at least 5.0. Ninety-five percent confidence intervals were calculated for the SMR according to the formula of Byar, referenced in Rothman and Boice [1979]. For some analyses, we computed SMRs and exact confidence intervals according to Fisher [Rothman and Boice, 1979], when observed and expected deaths were less than 5.0.

## RESULTS

### Overall Cohort

A total of 34,597 employees contributed 428,190 person-years from 1964 through 1983. The distribution of persons by gender and vital status is displayed in Table I. For the total cohort, 20,928 or 60% of employees entered after 1964, indicating that this is a relatively young cohort. There are 19,565 additional employees and 346,133 additional person-years at risk (PYAR) in this cohort, compared to the previous study.

A total of 3,909 deaths occurred compared to 4,604 expected deaths corresponding to an all-cause SMR of 0.85 [95% confidence interval (CI) = 0.82, 0.88; Table II]. For the overall cohort, there are significant deficits for most broad non-malignant disease categories, as well as all cancers. Significant excesses are present for malignant melanoma [observed deaths (N) = 16, SMR = 1.87, 95% CI = 1.07, 3.04] and aortic aneurysm (N = 69, SMR = 1.43, 95% CI = 1.11, 1.81). A larger, less precise SMR is present for benign neoplasms (N = 8, SMR = 1.96, 95% CI = 0.84, 3.86).

For female employees (Table III), all-cause mortality is significantly low (N = 207, SMR = 0.73). No cause of death category is significantly elevated but there are slight excesses of lung cancer, cirrhosis, and diseases of the arteries and capillaries. There are deficits in most major disease categories, while cancer mortality is about as expected (N = 80, SMR = 0.96). The balance of this paper focuses on male employees.

No angiosarcomas and seven mesotheliomas (six pleural, one unspecified) were identified. Based on Canadian rates available from the literature [McDonald and McDonald, 1980] the expected number of deaths in this cohort is 1.24. The resulting SMR of 5.65 is significant (95% CI = 2.27, 11.63).

The average starting employment year for these seven decedents is 1933 (range 1927--1943), the average duration of employment is 35 years (range 29–42), and all decedents died between 1970 and 1980. Examination of the job histories suggests many (e.g., pipefitter and mechanic) in which asbestos exposure would be likely, although we did not estimate past asbestos exposure. Five of these seven cases worked at a single refinery/petrochemical plant complex, and six of the seven worked in refineries at some point in their careers.

Mortality patterns by employment duration are shown in Table IV for selected causes of death. Table IV presents mortality patterns by employment duration for selected causes of death. Latency periods of 10 years (shown) and 20 years (not shown) were considered. For malignant melanoma, SMRs are higher for longer employment duration subgroups, especially the 15–24 year subgroup. SMRs are very imprecise for the 35 + employment group, however. The patterns are similar when a 20 year latent period is considered. For leukemia, kidney cancer, and non-Hodgkin's lymphoma, however, there are no apparent trends with employment duration, and considerable overlaps in the 95% confidence intervals.

## Operating Segments

The following operating segments are not discussed in detail since very few deaths (N) occurred: agricultural chemicals (N = 13), building products (N = 3), coal/minerals (N = 2), and unknown (N = 30). Office workers contributed 384 deaths, but no disease categories showed significant excesses, while significant deficits were present for most major non-malignant cause of death categories, as well as total malignancies.

**Refinery locations.** This operating segment is the largest, consisting of 9,276 male workers, of which 7,437 (80%) were ever employed in an HC-exposed job. Overall mortality is slightly below national rates (N = 1805, SMR = 0.91, 95% CI = 0.86, 0.95, see Table V), as are most major disease categories. Significant deficits of "other heart disease" (SMR = 0.65) and acute myocardial infarction (SMR =

**TABLE II. Mortality Results 1964–1983 in Total Cohort of All Operating Segments in a Petroleum Company (N = 34,597)**

| Cause of death (ICD codes, 8th) | Observed | Expected | SMR | 95% Conf. Int. Lower limit | 95% Conf. Int. Upper limit |
|---|---|---|---|---|---|
| All causes (001–999) | 3,909 | 4,603.6 | 0.85 | 0.82 | 0.88 |
| Infectious Dx[a] (001–136) | 17 | 28.6 | 0.60 | 0.35 | 0.95 |
| Endocrine Dx (240–279) | 65 | 86.7 | 0.75 | 0.58 | 0.96 |
| Blood Dx (280–289) | 10 | 11.2 | 0.90 | 0.43 | 1.65 |
| Circulatory Dx (390–458) | 2,085 | 2,309.0 | 0.90 | 0.87 | 0.94 |
| Respiratory Dx (460–519) | 250 | 321.5 | 0.78 | 0.68 | 0.88 |
| Digestive Dx (520–577) | 143 | 189.5 | 0.76 | 0.64 | 0.89 |
| Genitourinary Dx (580–629) | 48 | 64.1 | 0.75 | 0.55 | 0.99 |
| Symptoms/ill-defined (780–796) | 26 | 36.8 | 0.71 | 0.46 | 1.04 |
| Accidents/violence (800–999) | 239 | 421.9 | 0.57 | 0.50 | 0.64 |
| Malignant neoplasms (140–209) | 911 | 1,025.9 | 0.89 | 0.83 | 0.95 |
| Esophagus Ca[b] (150) | 22 | 22.3 | 0.99 | 0.62 | 1.50 |
| Stomach Ca (151) | 57 | 83.1 | 0.69 | 0.52 | 0.89 |
| Large Intestine Ca (153) | 94 | 93.7 | 1.00 | 0.81 | 1.23 |
| Rectum Ca (154) | 39 | 41.4 | 0.94 | 0.67 | 1.29 |
| Liver Ca (155) | 8 | 8.6 | 0.93 | 0.40 | 1.83 |
| Pancreas Ca (157) | 48 | 57.7 | 0.83 | 0.61 | 1.10 |
| Bronchus/Lung Ca (162.1) | 243 | 283.1 | 0.86 | 0.75 | 0.97 |
| Bone Ca (170) | 6 | 4.4 | 1.37 | 0.50 | 2.97 |
| Malignant melanoma (172) | 16 | 8.6 | 1.87 | 1.07 | 3.04 |
| Breast Ca (174) | 20 | 21.3 | 0.94 | 0.57 | 1.45 |
| Cervix uteri Ca (180) | 3 | 4.0 | — | — | — |
| Corpus uteri Ca (182.0) | 3 | 1.9 | — | — | — |
| Ovary Ca (183.0) | 6 | 5.7 | 1.05 | 0.38 | 2.28 |
| Prostate Ca (185) | 89 | 85.7 | 1.04 | 0.83 | 1.28 |
| Kidney Ca (189.0–189.2) | 23 | 23.4 | 0.98 | 0.62 | 1.47 |
| Bladder Ca (188) | 26 | 32.9 | 0.79 | 0.52 | 1.16 |
| Brain (malignant) Ca (191) | 25 | 25.5 | 0.98 | 0.64 | 1.45 |
| Nervous System (all) (191–192, 225, 238–239) | 32 | 32.5 | 0.98 | 0.67 | 1.39 |
| Reticulum-cell sarcoma (200.0) | 9 | 6.2 | 1.45 | 0.66 | 2.75 |
| Lymphosarcoma (200.1) | 6 | 8.7 | 0.69 | 0.25 | 1.51 |
| Non-Hodgkin's lymphoma (200,202) | 30 | 27.7 | 1.08 | 0.73 | 1.55 |
| Multiple myeloma (203) | 12 | 13.8 | 0.87 | 0.45 | 1.52 |
| Leukemias (204–207) | 39 | 37.7 | 1.03 | 0.74 | 1.41 |
| Benign neoplasms (210–228) | 8 | 4.1 | 1.96 | 0.84 | 3.86 |
| Diabetes (250) | 53 | 70.9 | 0.75 | 0.56 | 0.98 |
| Cerebrovascular Dx (430–438) | 333 | 374.5 | 0.89 | 0.80 | 0.99 |
| Myocardial infarction (410) | 700 | 816.8 | 0.86 | 0.80 | 0.92 |
| Other heart Dx (420–429) | 78 | 109.2 | 0.72 | 0.57 | 0.89 |
| Dx of arteries/capillary (440–448) | 156 | 137.0 | 1.14 | 0.97 | 1.33 |
| Aortic aneurysm (441) | 69 | 48.3 | 1.43 | 1.11 | 1.81 |
| Cirrhosis (571) | 62 | 86.2 | 0.72 | 0.55 | 0.92 |
| Dx of pancreas (577) | 10 | 8.6 | 1.16 | 0.56 | 2.14 |
| Kidney diseases (580–593) | 33 | 46.7 | 0.71 | 0.49 | 0.99 |

[a]Dx = Disease/Diseases
[b]Ca = Cancer

0.89) were offset by a statistically significant excess for diseases of arteries, capillaries, and veins (SMR = 1.34). This category subsumes aortic aneurysms, which are also elevated (N = 32, SMR = 1.34, 95% CI = 0.98, 2.02). No malignant

TABLE III. Mortality Results 1964–1983 in All Females of All Operating Segments in a Petroleum Company (N = 8,249)

| Cause of death (ICD Codes, 8th) | Observed | Expected | SMR | 95% Conf. Int. Lower limit | 95% Conf. Int. Upper limit |
|---|---|---|---|---|---|
| All causes (001–999) | 207 | 285.0 | 0.73 | 0.63 | 10.83 |
| Infectious Dx (001–136) | 0 | 2.1 | — | — | — |
| Endocrine Dx (240–279) | 4 | 8.2 | 0.49 | 0.13 | 1.24 |
| Blood Dx (280–279) | 0 | 1.0 | — | — | — |
| Circulatory Dx (390–458) | 78 | 114.1 | 0.68 | 0.54 | 0.85 |
| Respiratory Dx (460–519) | 7 | 13.2 | 0.53 | 0.21 | 1.09 |
| Digestive Dx (520–577) | 12 | 12.6 | 0.95 | 0.49 | 1.66 |
| Genitourinary Dx (580–629) | 3 | 3.9 | — | — | — |
| Symptoms/ill-defined (780–796) | 2 | 2.6 | — | — | — |
| Accidents/violence (800–999) | 11 | 32.2 | 0.34 | 0.17 | 0.61 |
| Malignant neoplasms (140–209) | 80 | 83.5 | 0.96 | 0.76 | 1.19 |
| Esophagus Ca (150) | 2 | 0.8 | — | — | — |
| Stomach Ca (151) | 2 | 3.9 | — | — | — |
| Large intestine Ca (153) | 8 | 8.8 | 0.90 | 0.39 | 1.78 |
| Rectum Ca (154) | 2 | 2.5 | — | — | — |
| Liver Ca (155) | 0 | 0.5 | — | — | — |
| Pancreas Ca (157) | 3 | 3.6 | — | — | — |
| Bronchus/lung Ca (162 1) | 11 | 7.9 | 1.39 | 0.69 | 2.48 |
| Bone Ca (170) | 0 | 0.3 | — | — | — |
| Malignant melanoma (172) | 1 | 1.1 | — | — | — |
| Breast Ca (174) | 20 | 20.1 | 1.00 | 0.6 | 1.54 |
| Cervix uteri Ca (180) | 3 | 4.0 | — | — | — |
| Corpus uteri Ca (182.0) | 3 | 1.9 | — | — | — |
| Ovary Ca (183.0) | 6 | 5.7 | 1.05 | 0.38 | 2.28 |
| Kidney Ca (189.0–189.2) | 1 | 1.3 | — | — | — |
| Bladder Ca (188) | 1 | 0.9 | — | — | — |
| Brain (malignant) Ca (191) | 2 | 2.6 | — | — | — |
| Nervous system (all) (191–192, 225, 238–239) | 2 | 3.4 | — | — | — |
| Reticulum-cell sarcoma (200.0) | 1 | 0.5 | — | — | — |
| Lymphosarcoma (200.1) | 1 | 0.7 | — | — | — |
| Non-Hodgkin's lymphoma (200,202) | 3 | 2.4 | — | — | — |
| Multiple myeloma (203) | 1 | 1.0 | — | — | — |
| Leukemias (204–207) | 3 | 3.2 | — | — | — |
| Benign neoplasms (210–228) | 1 | 0.6 | — | — | — |
| Diabetes (250) | 3 | 6.6 | 0.46 | 0.09 | 1.33 |
| Cerebrovascular Dx (430–438) | 20 | 27.4 | 0.73 | 0.45 | 1.13 |
| Myocardial infarction (410) | 15 | 33.2 | 0.45 | 0.25 | 0.75 |
| Other heart Dx (420–429) | 5 | 7.3 | 0.69 | 0.22 | 1.60 |
| Dx arteries/capillary (440–448) | 8 | 6.5 | 1.23 | 0.53 | 2.42 |
| Aortic aneurysm (441) | 3 | 1.3 | — | — | — |
| Cirrhosis (571) | 7 | 5.4 | 1.29 | 0.52 | 2.66 |
| Dx of pancreas (577) | 0 | 0.6 | — | — | — |
| Kidney diseases (580–593) | 3 | 3.3 | — | — | — |

neoplasm subsite was statistically elevated, except mesothelioma (N = 5, SMR = 11.11, 95% CI = 3.61, 25.93).

The benign neoplasms category shows an elevated SMR of 3.24, but it is very

TABLE IV. Mortality Patterns by Latency and Employment Duration for Selected Causes of Death in Male Workers of All Operating Segments of a Petroleum Company (N = 26,348)*

| Cause of death | Years employed | Observed | Expected | SMR | 95% Conf. Int. |
|---|---|---|---|---|---|
| Malignant melanoma | 0–4 | 0 | 0.28 | 0.0 | 0–13.18 |
| | 5–14 | 0 | 0.77 | 0.0 | 0–4.79 |
| | 15–24 | 9 | 1.96 | 4.59 | 2.10–8.72 |
| | 25–34 | 3 | 2.32 | 1.29 | 0.27–3.78 |
| | 35+ | 2 | 1.04 | 1.92 | 0.23–6.95 |
| Leukemia | 0–4 | 0 | 0.57 | 0.0 | 0–6.47 |
| | 5–14 | 1 | 1.96 | 0.51 | 0.13–2.84 |
| | 15–24 | 8 | 8.42 | 0.95 | 0.41–1.87 |
| | 25–34 | 14 | 13.57 | 1.03 | 0.56–1.73 |
| | 35+ | 8 | 7.09 | 1.13 | 0.48–2.22 |
| Kidney cancer | 0–4 | 0 | 0.27 | 0.0 | 0–13.66 |
| | 5–14 | 1 | 1.10 | 0.91 | 0.23–5.07 |
| | 15–24 | 7 | 5.79 | 1.21 | 0.48–2.49 |
| | 25–34 | 9 | 9.15 | 0.98 | 0.45–1.87 |
| | 35+ | 4 | 5.08 | 0.79 | 0.22–2.02 |
| Non-Hodgkin's lymphoma | 0–4 | 1 | 0.45 | 2.22 | 0.56–12.38 |
| | 5–14 | 2 | 1.56 | 1.28 | 0.16–4.63 |
| | 15–24 | 5 | 6.45 | 0.78 | 0.25–1.81 |
| | 25–34 | 14 | 9.63 | 1.45 | 0.79–2.44 |
| | 35+ | 5 | 5.31 | 0.94 | 0.30–2.20 |

*All results for a latency period of 10 years.

imprecise based on a wide 95% CI (1.04, 7.55) and is not significant (N = 3, SMR = 2.52, 95% CI = 0.52, 7.37) in exposed workers. The anatomic sites included two large intestine tumors and one unspecified tumor of the digestive system, but the malignant large intestine cancer SMR (0.91) was not elevated in refinery workers.

For cancers of interest from previous studies, leukemia mortality is not elevated (SMR = 0.84). Reticulum cell sarcoma (RCS) rates are elevated (N = 6, SMR = 2.44, 95% CI = 0.89, 5.31), but no patterns with latency or duration of employment are evident. Interestingly, there are no lymphosarcomas (LSA), which are commonly grouped with RCS in mortality studies. Non-Hodgkin's lymphoma (viz. RCS, LSA, and unspecified lymphomas) shows a moderate, non-significant elevation (N = 14, SMR = 1.27, 95% CI = 0.69, 2.13). There is also a deficit of multiple myeloma (SMR = 0.51). There were very few observed and expected malignant melanoma deaths, resulting in an imprecise SMR of 1.01. The kidney cancer SMR is not elevated (N = 9, SMR = 0.91, 95% CI = 0.42, 1.73).

**Marketing/transportation locations.** This operating segment consists of 6,823 male employees, of whom 5,041 (74%) are classified as ever exposed (Table VI). These workers are potentially exposed to finished product (viz. gasoline, diesel, and heating fuel), primarily via inhalation. Mortality relative to the general population is again significantly low (N = 1157, SMR = 0.88, 95% CI = 0.83, 0.93) as are most major disease categories. There was an excess of aortic aneurysms (N = 25, SMR = 1.78, 95% CI = 1.15, 2.63), which was decidedly more pronounced in non-exposed (including unknown exposure) marketing workers (N = 13, SMR = 2.64, 95% CI = 1.40, 4.51). The SMR was 1.31 among exposed workers and did not show trends with employment duration and latency.

TABLE V. Mortality Results 1964–1983 in Refinery Workers of a Petroleum Company (N = 9,276)

| Cause of death (ICD Codes, 8th) | Observed | Expected | SMR | 95% Conf. Int. Lower limit | 95% Conf. Int. Upper limit |
|---|---|---|---|---|---|
| All causes (001–999) | 1,805 | 1,994.3 | 0.91 | 0.86 | 0.95 |
| Infectious Dx (001–136) | 8 | 12.1 | 0.66 | 0.28 | 1.30 |
| Endocrine Dx (240–279) | 37 | 36.6 | 1.01 | 0.71 | 1.39 |
| Blood Dx (280–289) | 8 | 4.8 | 1.66 | 0.71 | 3.27 |
| Circulatory Dx (390–458) | 1,008 | 1,047.4 | 0.96 | 0.90 | 1.02 |
| Respiratory Dx (460–519) | 133 | 150.4 | 0.88 | 0.74 | 1.05 |
| Digestive Dx (520–577) | 61 | 77.7 | 0.78 | 0.60 | 1.01 |
| Genitourinary Dx (580–629) | 22 | 29.8 | 0.74 | 0.46 | 1.12 |
| Symptoms/ill-defined (780–796) | 17 | 15.5 | 1.10 | 0.64 | 1.76 |
| Accidents/violence (800–999) | 82 | 147.0 | 0.56 | 0.44 | 0.69 |
| Malignant neoplasms (140–209) | 388 | 430.9 | 0.90 | 0.81 | 0.99 |
| Esophagus Ca (150) | 7 | 9.8 | 0.71 | 0.29 | 1.47 |
| Stomach Ca (151) | 29 | 37.4 | 0.78 | 0.52 | 1.11 |
| Large intestine Ca (153) | 36 | 39.6 | 0.91 | 0.64 | 1.26 |
| Rectum Ca (154) | 22 | 18.2 | 1.21 | 0.76 | 1.83 |
| Liver Ca (155) | 4 | 3.6 | — | — | — |
| Pancreas Ca (157) | 20 | 24.8 | 0.81 | 0.49 | 1.24 |
| Bronchus/lung Ca (162.1) | 115 | 122.8 | 0.94 | 0.77 | 1.12 |
| Bone Ca (170) | 3 | 1.8 | — | — | — |
| Malignant melanoma (172) | 3 | 3.0 | — | — | — |
| Prostate Ca (185) | 46 | 43.1 | 1.05 | 0.77 | 1.40 |
| Kidney Ca (189.0–189.2) | 9 | 9.9 | 0.91 | 0.42 | 1.73 |
| Bladder Ca (188) | 14 | 15.6 | 0.90 | 0.49 | 1.51 |
| Brain (malignant) Ca (192) | 9 | 9.2 | 0.98 | 0.48 | 1.86 |
| Nervous system (all) (191–192, 225, 238–239) | 12 | 11.8 | 1.02 | 0.53 | 1.78 |
| Reticulum-cell sarcoma (200.0) | 6 | 2.5 | 2.44 | 0.89 | 5.31 |
| Lymphosarcoma (200.1) | 0 | 3.6 | — | — | — |
| Non-Hodgkin's lymphoma (200,202) | 14 | 11.0 | 1.27 | 0.69 | 2.13 |
| Multiple myeloma (203) | 3 | 5.9 | 0.51 | 0.10 | 1.49 |
| Leukemias (204–207) | 13 | 15.5 | 0.84 | 0.45 | 1.44 |
| Benign neoplasms (210–228) | 5 | 1.5 | 3.24 | 1.04 | 7.55 |
| Diabetes (250) | 34 | 30.3 | 1.12 | 0.78 | 1.57 |
| Cerebrovascular Dx (430–438) | 160 | 172.5 | 0.93 | 0.79 | 1.08 |
| Myocardial infarction (410) | 315 | 353.6 | 0.89 | 0.79 | 0.99 |
| Other Heart Dx (420–429) | 31 | 47.4 | 0.65 | 0.44 | 0.93 |
| Dx arteries/capillary (440–448) | 87 | 65.2 | 1.34 | 1.07 | 1.65 |
| Aortic aneurysm (441) | 32 | 22.4 | 1.34 | 0.98 | 2.02 |
| Cirrhosis (571) | 27 | 32.6 | 0.83 | 0.55 | 1.21 |
| Dx of pancreas (577) | 5 | 3.4 | — | — | — |
| Kidney diseases (580–593) | 14 | 20.9 | 0.67 | 0.37 | 1.12 |

Mortality for all malignant neoplasms is below that of the comparable general population segment (N = 254, SMR = 0.89, 95% CI = 0.78, 1.01). Two cancer subsites are moderately elevated: malignant melanoma (N = 6, SMR = 2.56, 95% CI = 0.94, 5.57) and multiple myeloma (N = 7, SMR = 1.81, 95% CI = 0.73, 3.73). There was also a multiple myeloma death in a female marketing worker, bringing the multiple myeloma SMR for *all* marketing workers to 1.89 (N = 8, 95% CI = 0.81, 3.73).

TABLE VI. Mortality Results 1964–1983 in Marketing/Transportation Workers of a Petroleum Company (N = 6,823)

| of death Codes, 8th) | Observed | Expected | SMR | 95% Conf. Int. Lower limit | 95% Conf. Int. Upper limit |
|---|---|---|---|---|---|
| All causes (001–999) | 1,157 | 1,317.2 | 0.88 | 0.83 | 0.93 |
| Infectious Dx (001–136) | 6 | 8.3 | 0.74 | 0.27 | 1.60 |
| Endocrine Dx (240–279) | 13 | 24.0 | 0.54 | 0.29 | 0.93 |
| Blood Dx (280–289) | 3 | 3.1 | — | — | — |
| Circulatory Dx (390–458) | 651 | 670.0 | 0.97 | 0.90 | 1.05 |
| Respiratory Dx (460–519) | 67 | 93.7 | 0.72 | 0.55 | 0.91 |
| Digestive Dx (520–577) | 35 | 54.8 | 0.64 | 0.44 | 0.89 |
| Genitourinary Dx (580–629) | 13 | 18.6 | 0.70 | 0.37 | 1.20 |
| Symptoms/ill-defined (780–796) | 10 | 10.5 | 0.96 | 0.46 | 1.76 |
| Accidents/violence (800–999) | 72 | 118.8 | 0.61 | 0.47 | 0.76 |
| Malignant neoplasms (140–209) | 254 | 285.8 | 0.89 | 0.78 | 1.01 |
| Esophagus Ca (150) | 9 | 6.5 | 1.38 | 0.63 | 2.63 |
| Stomach Ca (151) | 18 | 24.1 | 0.75 | 0.44 | 1.18 |
| Large intestine Ca (153) | 31 | 25.7 | 1.20 | 0.82 | 1.71 |
| Rectum Ca (154) | 6 | 11.8 | 0.51 | 0.19 | 1.10 |
| Liver Ca (155) | 2 | 2.5 | — | — | — |
| Pancreas Ca (157) | 15 | 16.4 | 0.92 | 0.51 | 1.51 |
| Bronchus/lung Ca (162.1) | 64 | 82.8 | 0.77 | 0.60 | 0.99 |
| Bone Ca (170) | 2 | 1.2 | — | — | — |
| Malignant melanoma (172) | 6 | 2.3 | 2.56 | 0.94 | 5.57 |
| Prostate Ca (185) | 28 | 25.9 | 1.08 | 0.72 | 1.56 |
| Kidney Ca (189.0–189.2) | 9 | 6.7 | 1.34 | 0.61 | 2.54 |
| Bladder Ca (188) | 5 | 9.7 | 0.52 | 0.17 | 1.21 |
| Brain (malignant) Ca (191) | 7 | 7.1 | 0.99 | 0.40 | 2.03 |
| Nervous system (all) (191–192, 225, 238–239) | 8 | 9.0 | 0.89 | 0.38 | 1.75 |
| Reticulum-cell sarcoma (200.0) | 1 | 1.7 | — | — | — |
| Lymphosarcoma (200.1) | 2 | 2.5 | — | — | — |
| Non-Hodgkin's lymphoma (200,202) | 7 | 7.7 | 0.91 | 0.36 | 1.87 |
| Multiple myeloma (203) | 7 | 3.9 | 1.81 | 0.73 | 3.73 |
| Leukemias (204–207) | 14 | 10.6 | 1.33 | 0.73 | 2.23 |
| n neoplasms (210–228) | 1 | 1.1 | — | — | — |
| tes (250) | 9 | 19.6 | 0.46 | 0.21 | 0.87 |
| ebrovascular Dx (430–438) | 90 | 106.4 | 0.85 | 0.68 | 1.04 |
| Myocardial infarction (410) | 230 | 237.5 | 0.97 | 0.85 | 1.10 |
| Other Heart Dx (420–429) | 28 | 31.0 | 0.91 | 0.60 | 1.31 |
| Dx arteries/capillary (440–448) | 46 | 39.9 | 1.15 | 0.84 | 1.54 |
| Aortic aneurysm (441) | 25 | 14.1 | 1.78 | 1.15 | 2.63 |
| Cirrhosis (571) | 14 | 25.4 | 0.55 | 0.30 | 0.93 |
| Dx of pancreas (577) | 2 | 2.5 | — | — | — |
| Kidney diseases (580–593) | 9 | 13.4 | 0.67 | 0.31 | 1.28 |

We next examined the mortality patterns for these two diseases in exposed marketing workers by employment duration, latency, year of death, and hire date (see Tables VIIA and VIIB). For multiple myeloma, higher SMRs were evident for workers with longer latencies and employment. The risk concentrates in employees hired before 1950. No such trends were apparent for malignant melanoma, although workers hired between 1940–1949 seemed to be at highest risk. The detection of trends is hindered by extremely small numbers.

TABLE VIIA. Multiple Myeloma Mortality Analyses in Marketing Workers of a Petroleum Company

| ncy sl | Deaths obs. | SMR | Employment (years) | Deaths obs. | SMR |
|---|---|---|---|---|---|
| 0–4 | 0 | 0 | 0–4 | 0 | 0 |
| 5–14 | 0 | 0 | 5–14 | 0 | 0 |
| 15–24 | 0 | 0 | 15–24 | 0 | 0 |
| 25–34 | 3 | 4.27 | 25–34 | 4 | 3.66 |
| 35+ | 2 | 1.59 | 35+ | 1 | 2.09 |
| Year of death | Deaths obs. | SMR | Hire year | Deaths obs. | SMR |
| 1964 | 0 | 0 | <1930 | 2 | 2.54 |
| 1965–1969 | 1 | 2.42 | 1930–1939 | 1 | 2.04 |
| 1970–1974 | 2 | 3.48 | 1940–1949 | 2 | 1.57 |
| 1975–1979 | 1 | 1.39 | 1950+ | 0 | 0 |
| 1980–1983 | 1 | 1.31 | | | |

TABLE VIIB. *Malignant* Melanoma Mortality Analyses in Marketing Workers of a Petroleum Company

| Latency (years) | Deaths obs. | SMR | Employment (years) | Deaths obs. | SMR |
|---|---|---|---|---|---|
| 0–4 | 0 | 0 | 0–4 | 0 | 0 |
| 5–14 | 1 | 2.94 | 5–14 | 1 | 2.96 |
| 15–24 | 1 | 2.15 | 15–24 | 2 | 3.99 |
| 25–34 | 1 | 2.59 | 25–34 | 1 | 2.22 |
| 35+ | 1 | 2.60 | 35+ | 0 | 0 |
| Year of death | Deaths obs | SMR | Hire year | Deaths obs. | SMR |
| 1964 | 0 | 0 | <1930 | 0 | 0 |
| 1965–1969 | 2 | 7.54 | 1930–1939 | 0 | 0 |
| 1970–1974 | 1 | 2.86 | 1940–1949 | 3 | 6.99 |
| 1975–1979 | 0 | 0 | 1950+ | 1 | 1.16 |
| 0–1983 | 1 | 1.92 | | | |

Mortality for other lymphopoietic cancers is generally consistent with expectation (see Table VI). Leukemia mortality is moderately elevated (N = 14, SMR = 1.33, 95% CI = 0.73, 2.23), but the SMR decreases in exposed marketing workers (N = 7, SMR = 0.98, 95% CI = 0.39, 2.02). Mortality for the myeloid and lymphatic cell types are about as expected for all marketing workers (N = 7, SMR = 0.96). This implies an SMR of 2.14 for seven leukemias which were not myeloid or lymphatic (not statistically significant). The vast majority of these would be expected to be leukemias unspecified as to cell type.

Kidney cancer mortality is of particular interest in this operating segment due to toxicologic findings on totally vaporized gasoline. Kidney cancer is moderately elevated in exposed marketing workers (SMR = 1.53), but the excess is not statistically significant (95% CI = 0.61, 3.16).

**Marine locations.** Marine workers have potential exposure to finished product, most often during loading and unloading operations, and are not generally ex-

posed to crude oil. This exposure pattern most closely resembles that of the marketing and transportation workers. There are relatively few employees (1,660) and deaths (176) in this group, although all-cause and external cause SMRs are significantly low. ... cancer subsite is significantly elevated. There were no deaths due to malignant anoma and only one from multiple myeloma; these two causes were elevated in ...arketing/transportation workers. There were five central nervous system cancers (benign and malignant), resulting in an SMR of 2.41 which is not significant (95% CI = 0.78, 5.63). This cause was not elevated in marketing workers (SMR = 0.87, Table VI).

**Upstream (production/drilling/exploration and pipeline) locations.** There were only 176 pipeline workers in this study. The vast majority of pipeline operations applicable to the study time period carried crude oil. Thus, pipeline workers were grouped with production, drilling, and exploration workers to form an upstream operating segment.

There were a total of 4,443 male upstream workers, but a lower percentage of workers was judged as potentially exposed to HC (2,465 of 4,443, or 55%) compared to the other operating segments. All cause mortality is significantly low in this group (SMR = 0.74, 95% CI = 0.66, 0.84), as are most non-malignant categories (see Table VIII). One cancer subsite was significantly elevated: malignant melanoma (N = 6, SMR = 6.00). Though this SMR is quite high, it is very imprecise, based on the exact 95% CI of (2.19, 13.06). None of the lymphopoietic cancer sites, or kidney cancer, were elevated.

Since most upstream workers (87.3%) resided in Alberta, we used Alberta reference rates in lieu of Canada rates. Table IX shows a slight SMR increase to 6.59 (95% CI = 2.42, 14.41) when using these rates. Thus, factors unique to Alberta do not explain the excess melanoma mortality. Of the 16 total melanomas in the entire cohort, 6 occurred on the trunk, while 9 were unspecified. Unfortunately, all six upstream cases were unspecified. Table IX shows that malignant melanoma mortality remained statistically elevated for exposed upstream workers (SMR = 5.48, 95% CI = 1.10, 16.03).

Despite extremely small numbers, there was a strong upward trend in SMRs for ...ployment duration and latency (see Table X). The SMR for employees with at least ...ears service and a 20-year latent period is 13.33, based on three deaths. Although ...mplete computerized work histories are not available, two of the three cases worked in drilling and gas production, while the third decedent was a gas plant operator.

**Petrochemical locations.** The single petrochemical location contributed too few employees (1,396) and deaths (52) for detailed analyses. However, SMRs for all causes (0.52), circulatory disease (0.55), and accidents/violence (0.28) are significantly reduced. There was only one cancer subsite with more than two deaths (lung cancer SMR = 0.52). No angiosarcoma deaths were observed despite the presence of vinyl chloride monomer at this location. Although some authors have noted an increased incidence of brain tumors in petrochemical workers [Reeve et al., 1983; Austin and Schnatter, 1983], no such deaths were observed in these workers.

## DISCUSSION

This study of petroleum workers simultaneously examined mortality according to different operating segments and exposure to hydrocarbons. A strength of this

TABLE VIII. Mortality Results 1964–1983 in Upstream Workers of a Petroleum Company (N = 4,443)

| ause of death (ICD Codes, 8th) | Observed | Expected | SMR | 95% Conf. Int. Lower limit | 95% Conf. Int. Upper limit |
|---|---|---|---|---|---|
| All causes (001–999) | 275 | 369.6 | 0.74 | 0.66 | 0.84 |
| Infectious Dx (001–136) | 1 | 2.4 | — | — | — |
| Endocrine Dx (240–279) | 3 | 6.3 | — | — | — |
| Blood Dx (280–289) | 0 | 0.8 | — | — | — |
| Circulatory Dx (390–458) | 118 | 162.8 | 0.73 | 0.60 | 0.89 |
| Respiratory Dx (460–519) | 8 | 20.0 | 0.40 | 0.17 | 0.79 |
| Digestive Dx (520–577) | 18 | 18.2 | 0.99 | 0.59 | 1.56 |
| Genitourinary Dx (580–629) | 3 | 3.6 | — | — | — |
| Symptoms/ill-defined (780–796) | 4 | 3.1 | — | — | — |
| Accidents/violence (800–999) | 36 | 58.0 | 0.62 | 0.43 | 0.86 |
| Malignant neoplasms (140–209) | 80 | 84.0 | 0.95 | 0.76 | 1.19 |
| Esophagus Ca (150) | 3 | 1.9 | — | — | — |
| Stomach Ca (151) | 7 | 6.2 | 1.13 | 0.45 | 1.15 |
| Large intestine Ca (153) | 6 | 6.9 | 0.87 | 0.32 | 1.88 |
| Rectum Ca (154) | 2 | 3.2 | — | — | — |
| Liver Ca (155) | 0 | 0.8 | — | — | — |
| Pancreas Ca (157) | 3 | 4.8 | — | — | — |
| Bronchus/lung Ca (162.1) | 20 | 26.9 | 0.74 | 0.45 | 1.15 |
| Bone Ca (170) | 0 | 0.4 | — | — | — |
| Malignant melanoma (172) | 6 | 1.0 | 6.00 | 2.19 | 13.06 |
| Prostate Ca (185) | 7 | 4.6 | 1.54 | 0.62 | 3.16 |
| Kidney Ca (189.0–189.2) | 2 | 2.2 | — | — | — |
| Bladder Ca (188) | 4 | 2.1 | — | — | — |
| Brain (malignant) Ca (191) | 4 | 3.0 | — | — | — |
| Nervous system (all) (191–192, 225, 238–239) | 6 | 3.7 | 1.60 | 0.59 | 3.49 |
| Reticulum-cell sarcoma (200.0) | 0 | 0.6 | — | — | — |
| Lymphosarcoma (200.1) | 0 | 0.8 | — | — | — |
| Non-Hodgkin's lymphoma (200,202) | 2 | 2.7 | — | — | — |
| Multiple myeloma (203) | 0 | 1.1 | — | — | — |
| Leukemias (204–207) | 2 | 3.3 | — | — | — |
| :nign neoplasms (210–228) | 0 | 0.4 | — | — | — |
| Diabetes (250) | 2 | 5.0 | — | — | — |
| Cerebrovascular Dx (430–438) | 19 | 20.4 | 0.93 | 0.56 | 1.45 |
| Myocardial infarction (410) | 48 | 72.8 | 0.66 | 0 49 | 0.88 |
| Other Heart Dx (420–429) | 3 | 8.1 | 0.37 | 0 07 | 1.08 |
| Dx arteries/capillary (440–448) | 5 | 7.3 | 0.69 | 0.22 | 1.60 |
| Aortic aneurysm (441) | 2 | 3.4 | — | — | — |
| Cirrhosis (571) | 7 | 10.6 | 0.66 | 0.26 | 1.36 |
| Dx of pancreas (577) | 3 | 0.9 | — | — | — |
| Kidney diseases (580–593) | 2 | 3.0 | — | — | — |

study is its breadth; it examines different operating segments and different work environments with different exposure patterns. Stratification by operating segments allows for the assessment of mortality patterns for several fairly homogeneous work environments. Another strength of the study is its size; to our knowledge it has the largest number of employees of any single-company mortality follow-up study reported in the petroleum industry [see Wong and Raabe, 1989]. Thus, the precision of

TABLE IX. Malignant Melanoma SMRs in Upstream Workers of a Petroleum Company 1964–1983

| Referent population | Observed | Expected | SMR | 95% CI |
|---|---|---|---|---|
| Canadian population | 6 | 1.00 | 6.00 | 2.19–13.06 |
| Alberta population | 6 | 0.91 | 6.59 | 2.42–14.41 |
| **Exposure status** | **Observed** | **Expected** | **SMR** | **95% CI** |
| Exposed | 3 | 0.55 | 5.48 | 1.13–16.03 |
| Not exposed | 2 | 0.30 | 6.62 | 0.80–23.92 |
| Unknown | 1 | 0.15 | 6.64 | 0.17–36.90 |

TABLE X. Malignant Melanoma SMRs by Latency and Employment Duration for Upstream Workers in a Petroleum Company 1964-1983

| Latency (years) | Observed deaths | Expected deaths | SMR |
|---|---|---|---|
| 0–4 | 0 | 0.06 | 0 |
| 5–14 | 0 | 0.14 | 0 |
| 14–24 | 1 | 0.17 | 5.92 |
| 25–34 | 1 | 0.14 | 7.25 |
| 35+ | 1 | 0.04 | 23.80 |
| **Employment duration (years)** | **Observed deaths** | **Expected deaths** | **SMR** |
| 0–4 | 0 | 0.11 | 0 |
| 5–14 | 0 | 0.13 | 0 |
| 15–24 | 1 | 0.18 | 5.56 |
| 25–34 | 1 | 0.12 | 8.33 |
| 35+ | 1 | 0.02 | 62.50 |

the SMRs is enhanced without sacrificing data comparability that is inherent in inter-industry studies.

Among the study shortcomings are: a) the relatively young cohort examined (only 11% are deceased and 54% have less than 20 years of follow-up); b) the relatively short follow-up period; c) the incomplete job history information for some workers; and d) the absence of data on life-style risk factors such as smoking, alcohol use, and socio-economic status. The incompleteness of job histories necessitated extrapolation of last job or first known job to earlier time periods. This could result in misclassification of exposure and latency/duration in long-term exposed employees. The relatively short follow-up time and young cohort translates into less precise SMRs for longer employment duration and latency times, given the cohort's size. For example, 30% of the 109,336 person-years in refinery workers are for workers with less than 5 years worked and 10 years latency. Further follow-up of the cohort would thus be desirable.

### Overall Cohort

Overall mortality and most non-malignant causes of death were significantly lower than general population rates in the overall cohort, as well as the major oper-

ating segments. This is most likely due to the attendant survival advantages for selection into and maintenance of employment. This healthy worker effect is repeatedly observed in petroleum industry and other occupational cohort studies. Incomplete death ascertainment is not a likely explanation, since a blinded test for known deaths revealed nearly 98% death ascertainment based on the identifiers supplied for linkage to Statistics Canada's Mortality Data Base (MDB) [Schnatter et al., 1990].

### Malignant Melanoma

The most noteworthy finding is the significant excess of malignant melanoma overall, particularly for the upstream operating segment (SMR = 6.0). The excess persists when Alberta rates are used (SMR = 6.6) and for HC-exposed upstream workers (SMR = 5.5).

Although we analyzed results by operating segment to detect potential risks applicable to a fairly homogeneous work environment, the upstream operating segment remains relatively diverse. For example, drilling workers are primarily exposed to crude oil and drilling muds (made up of complex HC mixtures) via dermal absorption, but production workers can be exposed to a wider range of HCs, including natural gas and other liquid streams. Exploration workers are less likely to be exposed to hydrocarbons. Many upstream workers spend a significant part of their occupational time out-of-doors. Thus, at this time, we are unable to relate the increased melanoma SMR in upstream workers to a specific agent or HC stream.

Various HC fractions have produced squamous cell carcinomas in mice, including light and heavy vacuum and catalytically cracked distillates [IARC, 1989]. However, these findings may not be etiologically relevant to melanomas, which arise from pigment-producing melanocytes.

Previous experimental and population-based studies give little indication that substances which may have been encountered by the upstream workers can cause melanoma. Other exposures, such as polychlorinated biphenyls [Bahn et al., 1976], gold mining [Armstrong et al., 1979], rubber industry processes [Holmberg et al., 1983], vinyl chloride [Heldas et al., 1984], aircraft worker activities [Costa et al., 1989], electrical jobs and electromagnetic fields [Olin et al., 1985; Vagero et al., 1985; Swerdlow, 1983], and the work activities of chemists [Austin et al., 1981; Hoar and Pell, 1981] have been associated with melanoma, but none of the findings are conclusive. Most refinery worker cohort studies do not show an excess of skin cancer and/or melanoma [Delzell et al., 1988], except for two [Rushton and Alderson, 1981; Nelson et al., 1987]. In experimental studies, 7,12-dimethylbenz(a)anthracene (DMBA) [Takizawa et al., 1985; Berkelhammer and Oxenhandler, 1987] and urethane [Vesselinovitch et al., 1970; Toth et al., 1961] have been shown to cause melanomas in mice and hamsters, respectively. In summary, however, the literature lends little support that hydrocarbon streams encountered in the upstream operating segment can cause melanoma.

Melanoma mortality is increasing rapidly [Osterlind and Jenson, 1986; Glass and Hoover, 1989] and is thought to be due to a real rise in incidence, rather than improved diagnosis for the disease [Elwood and Lee, 1974]. Concurrent 5-year intervals were used for observed and expected deaths in this study, so a temporal effect should be ruled out as an explanatory factor for the excess.

Several investigators have suggested that the background melanoma increase is due to ultraviolet (UV) light exposure [Fitzpatrick and Sober, 1985; Lee, 1982], and,

specifically, UV overexposure, or the number of sunburning episodes [Rhodes et al., 1987; Mackie and Atchison, 1982], especially during childhood [Gallagher et al., 1985]. Owing to the amount of occupational time spent outdoors by these workers, UV light exposure must also be considered a candidate etiologic factor for these melanomas.

A sunlight/exposure interaction is also a plausible explanation for this excess. Indeed, experimental evidence for this phenomenon exists [Epstein et al., 1967]. Hairless, pigmented mice exposed to DMBA alone developed blue nevi precursor lesions. Mice treated further with mid-UV energy light developed melanomas, while no melanomas developed in mice treated with DMBA alone, UV light alone, or neither factor. The applicability of these findings to our study is uncertain without more specific information on exposure to HC streams and UV light.

Other known risk factors for melanoma include congenital moles, dysplastic nevi syndrome, a history of cutaneous melanoma in close relatives, and the skin's sun-sensitivity [Rhodes et al., 1987]. We did not collect information on any of these risk factors, thus they remain as possible explanations of the excess. A factor more common in Alberta, where most of the upstream workers reside, is not a likely explanation, since the use of Alberta reference rates did not alter the excess.

The only other study of upstream workers did not report a melanoma or skin cancer excess [Divine and Barron, 1987]. These workers were primarily from lower latitudes, where cumulative sunlight exposure would be expected to be higher. A greater opportunity for *intermittent* sunlight exposure in Alberta, local work environment differences, or the presence of other potential confounders mentioned above may explain the differences in the two studies.

Although the malignant melanoma excess concentrated in employees ever assigned to the upstream segment, the melanoma SMR was also significant for all males in the total cohort and was elevated, though not significantly so (N = 6, SMR = 2.56) in marketing/transportation workers, and in exposed marketing/transportation workers (N = 4, SMR = 2.37). Trends with latency and duration of exposure were not evident, as in the upstream segment. While these data indicate that this finding could be due to chance, further follow-up efforts on this cohort would be useful to monitor melanoma mortality and incidence.

### Aortic Aneurysm

Aortic aneurysm mortality was also significantly elevated in the male cohort as a whole (SMR = 1.41). There are few population-based studies of aortic aneurysms, and fewer studies which examine the relationship of the disease to occupation. Mortality due to aortic disease is subject to variation since other cardiovascular diseases such as atherosclerotic heart disease (ASHD), hypertensive disease, and cerebrovascular disease often coexist, and sometimes take priority in death certificate coding [Dalen, 1987].

Melton et al. [1984] stress that improved diagnostic capabilities (roentgenograms and ultrasonography) have seemed to fuel a rise in the incidence of this disease, particularly the smaller, asymptomatic abdominal aneurysms. The majority (35 of 40 specified, 87.5%) of the aortic aneurysms in this study were abdominal aneurysms, which are more strongly influenced by this trend. If employees in this study are more likely than the general population to undergo the diagnostic procedures mentioned above, an improved detection rate for aortic aneurysms could result. This has been

labeled a "diagnostic sensitivity bias" for workers with better access to sophisticated diagnostic procedures [Greenwald et al., 1981]. Other factors that cannot be ruled out xplanatory factors are trauma, and dietary and lifestyle factors related to hyper-ion and arteriosclerosis.

### Operating Segments

**Refinery workers.** Several studies of refinery workers have been conducted and have been reviewed by Delzell et al. [1988], Wong and Raabe [1989], and IARC [1989]. The latter group classified refinery operations as probably carcinogenic largely based on findings regarding leukemia, and also on skin cancer. The findings in this study do not support this conclusion. Both of these death categories showed deficits in these refinery workers. The only significant finding in refinery workers was for benign neoplasms. However, this excess was weakened in ever-exposed workers and was not based on tumors of a common anatomical site. Furthermore, the corresponding malignant cancer sites were not elevated.

While the combined category of all lymphopoietic cancers show an SMR close to unity, mortality patterns for different lymphopoietic cancers show considerable variation. Lymphosarcoma (LSA) and multiple myeloma show very low SMRs, while reticulosarcoma (RCS) shows an elevation (SMR = 2.44). The combined RCS and LSA SMR is 0.98 (N = 6), consistent with previous refinery worker studies [see Delzell et al., 1988] which commonly group these two diseases. Possible diagnostic overlap [Percy et al., 1981; Heath, 1982] could explain disparate SMRs for lymphopoietic subtypes [Delzell et al., 1988], but non-optimal groupings of these subtypes cloud even this explanation. Future petroleum worker studies could evaluate RCS and LSA separately as a test of the elevated RCS rate found in this study. Alternately, a grouped category of "non-Hodgkin's lymphoma" is preferred because it accounts for *all* lymphomas which are not distinctly Hodgkin's disease. In other studies, unspecified lymphomas are often grouped with multiple myeloma, polycythemia vera, and myelofibrosis into an "other lymphatic cancer" category. This rubric consists of diseases with different etiologies; if the study size is sufficient, it uld seem advisable to at least evaluate unspecified lymphomas with other non-dgkin's lymphomas, and multiple myeloma separately.

**Marketing/transportation workers.** Marketing/transportation workers show significantly elevated mortality for aortic aneurysms, which is stronger in non-exposed workers. In the only other study among similar employees, Rushton and Alderson [1983] report results for "diseases of the arteries" which includes aortic aneurysms as well as atherosclerosis. This cause was not elevated based on 81 observed deaths (SMR = 0.91).

Multiple myeloma mortality was elevated, though not significantly, in exposed as well as total marketing workers in the present study. This excess concentrated among employees with long employment duration and latency, and most of the decedents died within 5 years of last employment. Rushton and Alderson [1983] reported a multiple myeloma SMR of 1.17, based on 11 cases. This does not support our finding, although additional analyses by duration of employment were not performed for this cause. Rushton and Alderson [1983] do report a significant excess of another lymphopoietic disorder, myelofibrosis, although we did not observe any such deaths. Rushton and Alderson [1983] also found a non-significantly elevated SMR for Hodgkin's disease (1.39), but we found a deficit (N = 2, SMR = 0.84).

Some finished products such as gasoline contain benzene, a known cause of leukemia. Leukemia mortality was moderately raised in all marketing workers (N = 14, SMR = 1.33) but was lower for exposed marketing workers (N = 7, SMR = 98). We have no knowledge of how many workers were actually exposed to benzene, nor the level at which they may have been exposed. However, Rushton and Alderson [1983] also report results (SMR = 1.04) similar to our exposed workers.

Other investigators have suggested that multiple myeloma may be related to benzene exposure [Rinsky et al., 1987; Decouflé et al., 1983]. The leukemia results described above would argue against a benzene relationship, although Rinsky et al. [1987] suggest that benzene exposure at lower levels may cause multiple myeloma. Without more specific job histories relating to possible benzene exposure, we cannot evaluate this further.

Different lymphopoietic cancers share possible etiologies (e.g., aromatic solvents and radiation), and even diagnostic characteristics [e.g., lymphocytic leukemias/lymphomas, Heath, 1981]. Theoretically, an insult to a multipotent stem cell could manifest as different lymphopoietic cancers, which justifies the examination of an aggregate category of lymphopoietic cancers. However, several investigators argue against this [Crane et al., 1989; Linet, 1985], pointing out different etiologies even within specific leukemia subtypes (e.g., genetic factors, radiation, chromosomal aberrations). Although the authors of this paper share different views on the issue, we have examined a category of combined lymphopoietic cancer for reader convenience. For non-Hodgkin's lymphoma, multiple myeloma, leukemias, and myelofibrosis, a slight, non-significant (N = 21, SMR = 1.22) excess was observed in exposed marketing workers. A similar finding can be calculated for the U.K. workers (N = 57, SMR = 1.15) (Rushton and Alderson, 1983), suggesting no, or at most a slight, relationship in the total marketing work environment.

Renal carcinogenicity has been reported in male rats exposed to totally vaporized gasoline [Kitchen, 1984], although subsequent studies ascribe this to accumulation of a protein not found in humans. Exposure potentials of marketing/distribution workers are characterized by intermittent gasoline vapor inhalation, mostly during loading/unloading operations. For all HC-exposed workers in this segment, the kidney cancer SMR shows a 34% elevation, which is not statistically significant. Rushton and Alderson [1983] also report an elevation (SMR = 1.21), based on 23 deaths in marketing workers which is not statistically significant. While neither study alone is persuasive of an effect, a small effect cannot be ruled out. Ongoing studies in different downstream populations should shed further light on whether exposures encountered in this setting might be related to kidney cancer mortality.

**Upstream workers.** With the exception of malignant melanoma, upstream workers did not show other significantly elevated SMRs. Specifically, there were no thyroid cancer deaths or deaths from benign or unspecified neoplasms as in Divine and Barron [1987]. However, the upstream group in this study is relatively small, and the study has insufficient statistical power for excluding risks as large as five-fold for these rare causes of death.

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APPENDIX: ICDA Codes Examined for Mesothelioma and Angiosarcoma Deaths

| Mesothelioma | |
|---|---|
| Primary codes | Secondary codes |
| 7th Revision (1958–1968) | |
| 227—Mesothelioma, unspecified | Same |
| 212—Mesothelioma lung, pleura, mediastinum | |
| 211—Mesothelioma peritoneum | |
| 197—Malignant mesothelioma, unspecified or diaphragm | |
| 163—Malignant mesothelioma, lung or pleura | |
| 164—Malignant mesothelioma, mediastinum | |
| 158—Malignant mesothelioma, peritoneum | |
| 8th Revision (1969–1978) | |
| 228—Mesothelioma, unspecified | Same, except for 199 |
| 212—Mesothelioma, lung | |
| 158—Mesothelioma, peritoneum | |
| 211—Mesothelioma, peritoneum | |
| 163—Mesothelioma, pleura | |
| 212—Mesothelioma, pleura, fibrous or mediastinum | |
| 215—Mesothelioma, diaphragm | |
| 199—Malignant mesothelioma | |
| 162—Malignant mesothelioma, lung | |
| 171—Malignant mesothelioma, mediastinum | |
| 163—Malignant mesothelioma, mediastinum | |
| 9th Revision (1979+) | |
| 199—Mesothelioma, unspecified | Same, except for 199 |
| 162—Mesothelioma, lung | |
| 229—Benign mesothelioma | |
| 212—Benign mesothelioma, lung, pleura or mediastinum | |
| 215—Benign mesothelioma, diaphragm | |
| 211—Benign mesothelioma, peritoneum | |
| 171—Mesothelioma, diaphragm | |
| 164—Mesothelioma, mediastinum | |
| 58—Mesothelioma, peritoneum | |
| 53—Mesothelioma, pleura | |

| Angiosarcoma | |
|---|---|
| Primary codes | Secondary codes |
| 7th Revision (1958–1968) | |
| 197—Angiosarcoma | Same |
| 156—Angiosarcoma, liver | |
| 155—Primary angiosarcoma, liver | |
| 156—Primary angiosarcoma, bile ducts | |
| 8th Revision (1969–1978) | |
| 171—Angiosarcoma | Same |
| 197—Angiosarcoma, liver | |
| 155—Primary angiosarcoma, liver or intra-hepatic bile ducts | |
| 156—Primary angiosarcoma, extrahepatic bile ducts | |
| 9th Revision (1979+) | |
| 171—Angiosarcoma | Same |
| 155—Angiosarcoma, liver | |
| 155—Primary angiosarcoma, liver | |
| 156—Primary angiosarcoma, bile ducts | |