

Exhibit 3 13

A Cohort Mortality Study of Two California Refinery and Petrochemical Plants

Shan P. Tsai, PhD

Elizabeth L. Gilstrap, MS

Sally R. Cowles, MD, DrPH

Philip J. Snyder, MPH, JD

Charles E. Ross, DO, MS

This study examined the 1973 to 1989 mortality experience of Shell's two California manufacturing locations' employees who worked more than 6 months before December 31, 1989 and pensioners who were alive as of January 1, 1973. Vital status of each employee as of December 31, 1989 was determined from various sources including company records, the National Death Index, and the Social Security Administration's Master Beneficiary Record file. The study included many long-term employees, with more than half (57%) of the total population working 20 years or longer. The total population exhibited 11% lower all causes mortality and 20% lower cancer mortality, as compared with the California general population. There were no significant excesses of any cause-specific mortality including cancer. Among total employees, mortality for several cancer sites showed a statistically nonsignificant increase, for example, cancer of the kidney (8 observed deaths and 6.02 expected), cancer of the bladder (11 observed deaths and 9.17 expected), and Hodgkin's disease (2 observed deaths and 1.01 expected). A review of these work histories revealed no predominant work area or job assignment. In contrast to the ecologic studies based on local county rates, lung cancer mortality in this study was significantly lower (Standardized Mortality ratio [SMR] = 0.73). In addition, statistically significant deficits in mortality were found for cirrhosis of the liver (SMR = 0.63) and all external causes of death (SMR = 0.74). This study also failed to show an increased mortality rate for cancers of the brain, stomach, and prostate—causes which have been reported to be elevated in other refinery and petrochemical employee studies.

Occupational and environmental health issues occupy a major role in our societies. In the last two decades, a large number of epidemiologic studies of petroleum refinery employees have been conducted to address the question of possible excess cancer mortality among these workers. Although overall mortality and major causes of death have been shown to be substantially lower among refinery employees than those for the general population, deaths from a number of cancer sites have been elevated.¹⁻¹⁷ These include cancer of the lung, stomach, skin, kidney, brain, and lymphopoietic system.

To investigate whether these mortality risks existed in Shell's manufacturing employees in California, we conducted a mortality follow-up study. This study includes two Shell refinery and petrochemical manufacturing facilities in California, ie, Martinez Manufacturing Complex (MMC) in Contra Costa County and Wilmington Manufacturing Complex (WMC) in the Los Angeles Basin. The two populations combined provided a data base for evaluating the mortality patterns of these employees.

Both Shell facilities studied include refinery operations and smaller chemical production units. The original oil refining processes associated with these facilities generally date back to the early 1920s. While differences in potential employee exposure did exist periodically because of different crude feedstocks, additives, and chemical operations, generally long-term employees would have had similar opportunities for exposure to many chemicals common to the petrochemical industry.

From the Corporate Medical Department, Shell Oil Company, P.O. Box 2463, Houston, TX 77252-2463.

Address correspondence to: Shan P. Tsai, PhD.

0096-1736/93/3504-0415\$03.00/0

Copyright © by American College of Occupational and Environmental Medicine

Examples would have included both high and low boiling point refinery intermediate streams that result from distillation, treatment, cracking, and other processes. These include pitch, fuel oil, gas oils, propane, benzene, toluene, etc. Additionally, certain process streams would have contained polynuclear aromatics. Depending on the process involved, various catalysts were used (eg, nickel). Other occupational exposures possibly encountered by MMC/WMC employees include asbestos insulation, sulfur dioxide, ketones and alcohols, various solvents used by maintenance workers, and chemicals used by laboratory technicians.

Contra Costa County has been the focus of ongoing controversy regarding the role of industrial pollution in the distribution and magnitude of cancer rates in the county.¹⁸⁻²⁵ The original National Cancer Institute Cancer Atlas¹⁸ showed that both Contra Costa County and Los Angeles County had significantly higher lung cancer mortality than all US counties combined. Since then, a series of ecologic studies in Contra Costa County has demonstrated a consistently increased lung cancer rate for residents in the more industrialized northern part of the county compared to the southern, less industrialized and somewhat more affluent, part of the county.²¹⁻²³ This gradient in cancer incidence has been variously attributed to industrial activity or socioeconomic and life-style factor differences. Since 1970, lung cancer mortality rates in both Contra Costa and Los Angeles counties have not been any higher than the rates for surrounding counties nor significantly higher than the United States as a whole.²⁵ Studies of occupational groups within Contra Costa County have failed to show an excess of any cancer.^{12,24} Because employees of an industrial complex experience potentially far more exposure to industrial process chemicals and chemical emissions than the general public, examination of occupational groups employed by manufacturing facilities offers the opportunity to help determine whether industrial activity is producing cancer or

whether other factors are more important.

Method and Materials

The study population consisted of 4,585 Shell MMC and WMC full-time male employees who worked more than 6 months before December 31, 1989 and who were actively employed as of January 1, 1973 and pensioners who were alive as of January 1, 1973. These study subjects were identified from the Shell Health Surveillance System which contains demographic, mortality, morbidity, physical examination, and work history data.²⁶

Vital status as of December 31, 1989 for each employee, including those who terminated employment for reasons other than retirement, was determined from a number of sources. Company records were supplemented with results of data-linkage searches from the National Death Index and the Social Security Administration's Master Beneficiary Record file. Terminated employees not identified by the National Death Index search or the Social Security Administration search were assumed to be alive.

Because a death certificate is required as proof of death before the payment of benefits to a surviving spouse, most certificates are received by the company. Those not received were requested from the appropriate state vital statistics department. Death certificates were obtained for more than 99% of the decedents. For each death certificate, the underlying cause of death was coded according to the revision in effect at the time of death and then converted to the eighth revision of the International Classification of Diseases by trained Shell nosologists.²⁷

A person entered the study on January 1, 1973 if he began employment at least 6 months before that date. Otherwise, he entered the study 6 months after the day of employment at these two facilities. Person-years were accumulated from the entry date to the date of death or the study end date, whichever came first.

Mortality excesses and deficits in this report are expressed as standard-

ized mortality ratios (SMRs) which were calculated using the Occupational Cohort Mortality Analysis Program.²⁸ The SMR is the number of observed deaths in the study group divided by the number of deaths that would be expected if the death rates for a comparison population were in effect. The number of deaths that are expected is adjusted for age and race of the study population and for the time period during which the study takes place. In addition, to help accommodate for geographic variations in mortality, the California mortality rates were used as the comparison. The 95% confidence interval for each SMR was calculated and its deviation from 1.00 was tested assuming a Poisson distribution for the observed deaths, using a two-sided test of significance.²⁹

Results

A total of 4,585 individuals who satisfied the cohort criteria were included in the analysis. The average age at entry into the cohort was 45 years. The total number of person-years of observation was 57,657, and the subjects were followed for an average of 12.6 years. A total of 3,534 employees (77%) were still alive at the end of the study period. White males are the largest grouping, representing 95% of the study population. Employees, primarily pensioners, whose race could not be determined (11%) were included in the "white" category. At the end of 1989, retirees comprised 50% of the study population, followed by active employees (29%) and termines (19%). A total of 1,051 individuals had died during the study period. Ninety-one percent of these deaths occurred in retirees (Table 1). Sixty-two percent of the total population were employed after 1950 while 86% of the deaths occurred among individuals hired before 1950 (Table 2). Approximately three fourths of the employees worked 10 years or longer and 57% worked 20 years or longer. The average duration of employment for Martinez employees was 20.8 years and for Wilmington employees, 20.4 years.

Table 3 shows the observed deaths, SMRs, and their 95% confidence intervals. For both refineries, the total mortality was significantly lower than the corresponding California population (SMR = 0.89 at Martinez, 0.90 at Wilmington; 0.89 for the combined). Since the operations of these two facilities were similar, our presentation will focus on the total combined cohort. Mortality from all cancer for the combined population was also significantly lower than the California experience (SMR = 0.80).

Statistically significant deficits were seen for cancer of the lung (SMR = 0.73), cirrhosis of the liver (SMR = 0.63), and all external causes of death (SMR = 0.74). The SMR for brain cancer was low with only two observed deaths (SMR = 0.34). Of over

50 causes of death examined, none was statistically significantly increased. The observed number of deaths from all diseases of the respiratory system was less than that expected (SMR = 0.88), while emphysema was slightly elevated with an SMR of 1.11.

Among the total males, the SMR for several cancer sites showed a non-significant increase, for example, cancer of the skin (SMR = 1.07), cancer of the bladder (SMR = 1.20), and cancer of the kidney (SMR = 1.33). The excesses came primarily from the Wilmington refinery that experienced an SMR of 1.16 for skin cancer, 1.42 for bladder cancer, and 2.16 for kidney cancer. The SMR for total lymphopoietic tissue cancer was slightly elevated (SMR = 1.04). This increase

was mainly due to the elevated mortality from Hodgkin's disease (SMR = 1.99 based on two observed deaths) and cancer of other lymphatic tissue (SMR = 1.22). A statistically nonsignificant elevation was noted for leukemia at Wilmington (SMR = 1.47).

Long latent periods are usually required for chronic diseases to develop. To assess the possible effect of latency, a subgroup of employees ($n = 3,335$) who worked for a minimum of 10 years was also examined. This group contributed 69% of the total person-years in the total cohort. The SMR for all causes was 0.91 ($P < .05$) with 1019 observed deaths and 1124 expected. The number of observed deaths for all cancer was less than expected (213 observed deaths and 264 expected, SMR = 0.81). The cause-specific mortality pattern is virtually the same as that exhibited by the total cohort in Table 3. There were no statistically significant excesses in any cause of death. Similar to the total cohort results, nonsignificantly elevated SMRs were noted for several cancer sites including cancer of the kidney (8 observed and 5.81 expected, SMR = 1.38), cancer of the bladder (11 observed and 9.05 expected, SMR = 1.22), malignant melanoma of the skin (4 observed and 3.34 expected, SMR = 1.20), Hodgkin's disease (2 observed and 0.82 expected, SMR = 2.43), and all lymphopoietic tissue cancer (25 observed and 23.77 expected, SMR = 1.05). A statistically

TABLE 1
Vital Status by the End of the Study

Vital Status	Martinez		Wilmington		Combined	
	No.	%	No.	%	No.	%
Currently Employed	731	29.5	610	28.8	1341	29.2
Terminated	464	18.8	407	19.3	871	19.0
Alive	454	(97.8)*	391	(96.1)	845	(97.0)
Dead	10	(2.2)	16	(3.9)	26	(3.0)
Retired	1236	50.0	1064	50.4	2300	50.2
Alive	727	(58.8)	621	(58.4)	1348	(58.6)
Dead	509	(41.2)	443	(41.6)	952	(41.4)
Died while employed	43	1.7	30	1.4	73	1.6
Total	2474	100.0	2111	100.0	4585	100.0

* Number in parentheses represents percentage within subgroup.

TABLE 2
Distribution of Study Population by Year of Hire

Year of Hire	Martinez		Wilmington		Combined	
	Employee* (%)	Deaths† (%)	Employee (%)	Deaths (%)	Employee (%)	Deaths (%)
1910-1919	13 (0.5)	12 (2.1)	4 (0.2)	4 (0.8)	17 (0.4)	16 (1.5)
1920-1929	219 (8.9)	165 (29.4)	192 (9.1)	160 (32.7)	411 (9.0)	325 (30.9)
1930-1939	240 (9.7)	123 (21.9)	154 (7.3)	93 (19.0)	394 (8.6)	216 (20.6)
1940-1949	502 (20.3)	191 (34.0)	395 (18.7)	152 (31.1)	897 (19.6)	343 (32.6)
1950-1959	278 (11.2)	41 (7.3)	317 (15.0)	44 (9.0)	595 (13.0)	85 (8.1)
1960-1969	358 (14.5)	21 (3.7)	272 (12.9)	22 (4.5)	630 (13.7)	43 (4.1)
1970-1979	463 (18.7)	6 (1.1)	364 (17.2)	9 (1.8)	827 (18.0)	15 (1.4)
1980-1989	401 (16.2)	3 (0.5)	413 (19.6)	5 (1.0)	814 (17.7)	8 (0.8)
Total	2474 (100.0)	562 (100.0)	2111 (100.0)	489 (99.9)	4585 (100.0)	1051 (100.0)

* Employees hired during specific employment period.

† Deaths of employees hired during specific employment period.

TABLE 3

Standardized Mortality Ratio (SMR) for Selected Causes of Death by Location*

Cause of Death (ICDA, 8th revision)	Martinez			Wilmington			Combined		
	Observed	SMR	95% CI†	Observed	SMR	95% CI	Observed	SMR	95% CI
All causes (000-999.8)	562	0.89‡	0.81-0.96	489	0.90‡	0.82-0.98	1051	0.89‡	0.84-0.95
All malignant neoplasms (140-209)	108	0.74‡	0.60-0.89	109	0.87	0.71-1.05	217	0.80‡	0.69-0.91
Buccal cavity and pharynx (140-149)	1	0.28	0.01-1.55	1	0.32	0.01-1.81	2	0.30	0.04-1.08
Digestive organs and peritoneum (150-159)	33	0.89	0.61-1.25	22	0.69	0.43-1.04	55	0.80	0.60-1.04
Esophagus (150)	2	0.80	0.07-2.17	3	1.05	0.22-3.08	5	0.81	0.26-1.89
Stomach (151)	5	0.84	0.27-1.97	1	0.20	0.01-1.09	6	0.54	0.20-1.19
Large intestine (153)	14	1.02	0.56-1.70	10	0.85	0.41-1.56	24	0.94	0.60-1.40
Rectum (154)	4	1.22	0.33-3.12	0			4	0.66	0.18-1.68
Liver and biliary passages (155, 156)	2	0.63	0.08-2.26	1	0.36	0.01-2.03	3	0.51	0.10-1.47
Pancreas (157)	6	0.79	0.29-1.73	7	1.08	0.44-2.23	13	0.93	0.49-1.58
Respiratory system (160-163)	31	0.63‡	0.43-0.89	37	0.88	0.62-1.21	68	0.74‡	0.58-0.94
Lung (162)	30	0.63‡	0.43-0.90	34	0.84	0.58-1.18	64	0.73‡	0.58-0.93
Skin (172.0-172.4, 172.6-172.9)	2	0.98	0.12-3.55	2	1.16	0.14-4.19	4	1.07	0.29-2.73
Prostate (185)	10	0.60	0.29-1.11	13	0.91	0.49-1.56	23	0.75	0.47-1.12
Bladder (188, 189.9)	5	1.02	0.33-2.37	6	1.42	0.52-3.09	11	1.20	0.60-2.15
Kidney (189.0, 189.1, 189.2)	2	0.62	0.07-2.22	6	2.16	0.79-4.71	8	1.33	0.57-2.62
Brain and central nervous system (191, 192)	1	0.32	0.01-1.76	1	0.37	0.01-2.07	2	0.34	0.04-1.23
Thyroid (193, 194)	0			0			0		
Lymphatic and hematopoietic tissue (200-209)	14	1.04	0.57-1.74	12	1.04	0.54-1.82	26	1.04	0.68-1.52
Lymphosarcoma and reticulosarcoma (200)	2	1.21	0.15-4.37	0			2	0.65	0.08-2.36
Hodgkin's disease (201)	2	3.66	0.44-13.23	0			2	1.99	0.24-7.18
Leukemia (204-207)	2	0.36	0.04-1.29	7	1.47	0.59-3.02	9	0.87	0.40-1.65
Other lymphatic tissue (202, 203, 208, 209)	8	1.39	0.60-2.75	5	1.02	0.33-2.39	13	1.22	0.65-2.09
Benign neoplasms (210-239)	1	0.64	0.02-3.59	3	2.26	0.47-6.60	4	1.39	0.38-3.55
Diabetes mellitus (250)	5	0.65	0.21-1.53	4	0.61	0.17-1.56	9	0.63	0.29-1.20
Heart disease (390-398, 400.1, 400.9, 402, 404, 410-414, 420-429)	236	0.95	0.84-1.09	215	1.01	0.88-1.15	451	0.98	0.89-1.08
Cerebrovascular disease (430-438)	46	0.99	0.73-1.33	34	0.84	0.58-1.18	80	0.92	0.73-1.15
Respiratory disease (460-519)	51	0.89	0.66-1.17	43	0.87	0.63-1.18	94	0.88	0.71-1.08
Cirrhosis of liver (571)	13	0.84	0.45-1.44	5	0.38‡	0.12-0.88	18	0.63‡	0.37-0.99
External causes of death (E800-E999.8)	31	0.78	0.53-1.10	23	0.69	0.44-1.03	54	0.74‡	0.55-0.96
Accidents (E800-E949)	21	0.90	0.56-1.37	13	0.66	0.35-1.13	34	0.79	0.55-1.10
Motor vehicle accidents (E810-E823)	11	1.10	0.55-1.98	3	0.37	0.08-1.07	14	0.77	0.42-1.30
Suicides (E950-E959)	10	0.89	0.43-1.63	6	0.64	0.23-1.38	16	0.77	0.44-1.26

* White and nonwhite combined.

† Confidence interval.

‡ $P < .05$.

significant deficit was observed for lung cancer (63 observed and 85.58 expected, SMR = 0.74). The observed number of deaths from nonmalignant respiratory disease was lower than that of expected (94 observed and 105.12 expected, SMR = 0.89).

Discussion

Mortality and morbidity data are routinely collected as part of occupational health surveillance programs at Shell Oil Company in order to detect potential adverse health effects due to occupational hazards or personal risk factors. The Health Surveillance System has been used to monitor the health outcomes of a given work force.^{15,30,31} Examination of data from a health surveillance system can provide an indicator of both the current and past health status of the work force and identify unusual disease occurrences that may require additional study to assess possible work associations.

Like most other epidemiology studies of employees, the overall mortality rate seen in this study is lower than the comparable mortality rate for the California general population. The lower overall mortality can be translated into longer life expectancy. Based on the mortality experience of these employees, the life expectancy at age 25 among cohort members is estimated to be 1.5 years longer than their counterparts.³² Such a difference may seem small, but the magnitude of this relative advantage is equivalent to the potential gain in life expectancy if 52% of all cancers were eliminated as a cause of death in the US male population.³³ The favorable mortality findings and better life expectancy experienced by these employees is probably due to a combination of the healthy worker effect, the relative absence of risks related to employment, and the beneficial effects of continuing employment, such as greater access to medical care and regular income.³⁴⁻³⁷

The SMR for all causes of death in this study was somewhat higher than the usually observed SMR of approximately 0.80 in oil refinery and petro-

chemical cohort studies.^{16,17} This is primarily due to the inclusion of a large group of retirees ($n = 1,257$) at the beginning of the study. When these retirees were excluded from the analysis, the mortality was much lower than the general population. In fact, the SMR for all causes was 0.71 (250 observed and 352 expected) for the combined cohort after exclusion of the pre-1973 retirees as compared with an SMR of 0.89 when these retirees were included. It is interesting to note that this diminishing of the "healthy worker effect" by inclusion of pre-1973 retirees does not have much impact on cancer death; the SMR for all cancer increases only slightly from 0.77 (70 observed and 91 expected) to 0.80.

Two ecologic studies of cancer in Contra Costa County have found excesses of several cancer sites, especially lung cancer, in residents near refinery and petrochemical facilities. In 1982, Austin reported results from an analysis of the relation between air pollution and lung cancer incidence rates as well as a case-control study of lung cancer.²¹ The industrialized area was found to have lung cancer rates roughly 40% higher than the nonindustrialized area. A weak association was found between lung cancer and SO_4 particulate matter in ambient air in white males, but not in white females. The subsequent case-control study concluded that differential smoking rates within the county explained the industrial to nonindustrial rate elevations. Kaldor et al in 1984 published another ecologic analysis of cancer which concluded that increasing exposure to industrial pollutants was associated with increased rates of cancer of all sites, cancer of the buccal cavity and pharynx, cancer of the stomach, cancer of the lung, cardiovascular disease, cerebrovascular disease, cirrhosis of the liver, and "other" diseases.²² Socioeconomic and lifestyle factors were not evaluated in this study.

The patterns observed in this Shell study do not show any statistically significant increased cancer mortality as a result of working at these two refineries. Overall, cancer mortality

was 20% lower than that of the California general population. This finding is similar to that seen in other large oil refinery cohort studies.⁹⁻¹⁷

It is noteworthy that cancer of the lung in this study was significantly lower than the general population. There is also no increase in nonmalignant respiratory diseases. Cigarette smoking is one of the most important risk factors for lung cancer. The smoking prevalence (between 1985 and 1989) of the present workers at these two locations was virtually the same as the US general population (30.2 vs 30.0 per 100).³⁸ However, the smoking intensity could be lower among these workers since there have always been stringent restrictions on cigarette smoking in the workplace. No other obvious reasons could account for the significantly low lung cancer death rate in this study. Similar observations have also been reported in other refinery workers studies.^{16,17} In 1986, Wong et al reported the results of a mortality study of workers at two California refineries (one in Contra Costa County and the other in Los Angeles County).¹² An analysis of lung cancer mortality by duration of employment revealed an inverse association. Ott et al in 1985 reported on the mortality of 1,919 men employed at two chemical manufacturing facilities in Contra Costa County for the period 1940-1979.²⁴ Deficits of 20% for deaths from all respiratory cancer were found. Our study also failed to show an increased mortality rate for cancers of the brain,^{4,6,12} stomach,^{5,6,10} or prostate as has been reported in other studies (R.W. Morgan, MD, and O. Wong, ScD, ENSR, unpublished report, 1985).

For kidney cancer, there was a statistically nonsignificant excess of deaths (8 observed and 6.03 expected deaths), primarily due to the WMC deaths. The SMRs for this cancer among other refinery studies ranged from 0.35 to 1.55.¹⁷ A review of the WMC work histories revealed no predominant pattern of job type, work area, or process operation. The longest jobs for the six cases included chemist/technologist (16 years), mechanic (20 years), asbestos worker (24 years),

alkylation and distilling operator (20 years), cat cracking operator (28 years), and dispatching wharfman (18 years). The average age at death for the kidney cancer cases was 72 years (range, 62–86 years), which is similar to that seen for adult kidney cancer deaths in the general population. The average duration of employment was 29 years (range, 20–40 years), and all cases were hired before 1952. This review did not suggest a common work-related component.

Bladder cancer resulted in a statistically nonsignificant elevated SMR of 1.20 based on 11 observed deaths. The average age at death for the six Wilmington bladder cancer cases was 78 years. All decedents were hired before 1946, and 29.5 years was their average duration of employment at WMC (range, 23–41 years). The cases had the following job titles: machinist/foreman (23 years), instrumentman/foreman (23 years), alkylation operator/foreman (12 years), clerk/accountant (30 years), draftsman (23 years), and truck driver (16 years).

A statistically nonsignificant excess of Hodgkin's disease was noted based only on two observed deaths. An inspection of Martinez work histories for these cases revealed no evidence of a common job or work area. A statistically nonsignificant excess of leukemia was seen in the WMC (SMR = 1.47) but not in the MMC (SMR = 0.36). The SMRs for this cancer in other studies ranged from 0.41 to 2.30, with an industrywide average of 1.10.¹⁷ The average age at death for the seven WMC employee leukemia cases was 70 years (range, 52–79 years), which is similar to the general population. Leukemia cell types included two acute myelogenous leukemia, four chronic lymphocytic leukemia, and one acute erythroleukemia. P-pump operator (25 years), storeman (16 years), distilling operator (20 years), alkylation and distilling operator/foreman (15 years), instrument repairman/foreman (25 years), maintenance foreman (18 years), and instrument repairer (2 years) were the longest jobs held for these cases. The average duration of employment was 29.3 years (range, 2–40 years), and

71% of the decedents were hired before 1947.

In summary, this study examined the mortality experience of two refinery employee populations over a period of 17 years from 1973 to 1989. There was no statistically significant increased risk due to any cause of death. A significantly lower mortality was found for cancer of the lung. Among total employees, deaths from Hodgkin's disease, cancer of the kidney, and cancer of the bladder were statistically nonsignificantly higher than expected based on the California general population. None of the suspected cancer risks appear to be related to particular work areas or job assignments within the refinery. Some of the increased cancer mortality risks suggested in the literature for refinery and petrochemical employees, ie, brain, stomach, and prostate, were not found in this study.¹⁷

References

- Hanis NM, Stavaky KM, Fowler JL. Cancer mortality in oil refinery workers. *J Occup Med.* 1979;21:167–174.
- Thomas TL, Waxweiler RJ, Moure-Eraso R, et al. Mortality patterns among workers in three Texas oil refineries. *J Occup Med.* 1982;24:135–141.
- Rushon L, Alderson MR. An epidemiological survey of eight oil refineries in Britain. *Br J Ind Med.* 1981;38:225–234.
- Therault G, Goulet L. A mortality study of oil refinery workers. *J Occup Med.* 1979;21:367–370.
- Therault G, Provencher S. Mortality study of oil refinery workers: five-year follow-up. *J Occup Med.* 1987;29:357–360.
- Thomas TL, Waxweiler RJ, Crandall MS, et al. Brain cancer among OCAW members in three Texas oil refineries. *Ann NY Acad Sci.* 1982;381:120–129.
- Hanis NM, Holmes TM, Shallenberger LG, et al. Epidemiologic study of refinery and chemical plant workers. *J Occup Med.* 1982;24:203–212.
- Wen CP, Tsai SP, McClellan WA, et al. Long-term mortality study of oil refinery workers. I. Mortality of hourly and salaried workers. *Am J Epidemiol.* 1983;118:526–542.
- Hanis NM, Shallenberger LG, Donaleski DL, et al. A retrospective mortality study of workers in three major US refineries and chemical plants. I. Comparisons with US population. *J Occup Med.* 1985;27:283–292.
- Nelson NA, Van Peenen PFD, Blanchard AG. Mortality in a recent oil refinery cohort. *J Occup Med.* 1986;28:514–516.
- Kaplan SD. Update of a mortality study of workers in petroleum refineries. *J Occup Med.* 1986;28:514–516.
- Wong O, Morgan RW, Bailey WJ, et al. An epidemiological study of petroleum refinery employees. *Br J Ind Med.* 1986;43:6–17.
- Wongsrichanalai C, Delzell E, Cole P. Mortality from leukemia and other diseases among workers at a petroleum refinery. *J Occup Med.* 1989;31:106–111.
- Divine BJ, Barron V, Kaplan SD. Texaco mortality study. I. Mortality among refinery, petrochemical and research workers. *J Occup Med.* 1985;27:445–447.
- McCraw DS, Joyner RE, Cole P. Excess leukemia in a refinery population. *J Occup Med.* 1985;27:220–222.
- Delzell E, Austin H, Cole P. Epidemiologic studies of the petroleum industry. *Occup Med State of the Art Rev.* 1988;3:455–474.
- Wong O, Rabbe GK. Critical review of cancer epidemiology in petroleum industry employees, with a quantitative meta-analysis by cancer site. *Am J Ind Med.* 1989;15:283–310.
- Mason TJ, McKay FW, Hoover R, et al. *Atlas of Cancer Mortality for U.S. Counties: 1950–1969.* DHEW Publication No. (NIH) 75–780. Washington, DC: Department of Health Education, and Welfare; 1975.
- Blot WJ, Brinton LA, Fraumeni JF, et al. Cancer mortality in U.S. counties with petroleum industries. *Science.* 1977;198:51–53.
- Hearey CD, Ury H, Siegelau A, et al. Lack of association between cancer incidence and residence near petrochemical industry in the San Francisco Bay area. *JNCI.* 1980;64:1295–1299.
- Austin DF. *Epidemiological Study of the Incidence of Cancer as Related to Industrial Emissions in Contra Costa County, California. Final Report to the Environmental Protection Agency, grant number R8063960-1.* Sacramento, Calif: Department of Health and Human Services, November, 1982.
- Kaldor J, Harris JA, Glazer E, et al. Statistical association between cancer incidence and major-cause mortality, and estimated residential exposure to air emissions from petroleum and chemical plants. *Environ Health Perspect.* 1984;54:319–332.
- Selvin S, Shaw G, Schulman J, et al. Spatial distribution of disease: three case studies. *JNCI.* 1987;79:417–423.
- Ott MG, Carlo GL, Steinberg S, et al.

- Mortality among employees engaged in chemical manufacturing and related activities. *Am J Epidemiol.* 1985;122:11-22.
25. Pickle LW, Mason TJ, Howard N, et al. *Atlas of U.S. Cancer Mortality Among Whites: 1950-1980.* DHHS Publication No. (NIH) 87-2900. Washington, DC: Department of Health and Human Services; 1987.
26. Joyner RE, Pack PH. The Shell Oil Company's computerized health surveillance system. *J Occup Med.* 1982;24:812-814.
27. *International Classifications of Diseases, 9th revision, Clinical Modification (1978).* Ann Arbor, Mich: Commission on Professional and Hospital Activities.
28. Marsh GM, Preininger M. OCMAP: A user-oriented occupational cohort mortality analysis program. *Am Stat.* 1980;34:245.
29. Bailar JC, Ederer F. Significance factors for the ratio of a Poisson variable to its expectation. *Biometrics.* 1964;20:639-643.
30. Cowles SR, Bennett JM, Ross CE. Medical surveillance for leukemia at a petrochemical manufacturing complex: four-year summary. *J Occup Med.* 1991; 33:808-812.
31. Tsai SP, Cowles SR, Tackett DL, Barclay MT, Ross CE. Morbidity prevalence study of workers with potential exposure to epichlorohydrin. *Br J Ind Med.* 1990;47:392-399.
32. Tsai SP, Hardy RJ, Wen CP. The standardized mortality ratio and life expectancy. *Am J Epidemiol.* 1992;135:824-831.
33. National Center for Health Statistics. *United States Life Tables Eliminating Certain Causes of Death. U.S. Decennial Life Tables for 1979-81.* Vol. 1, No. 2. DHHS Publication No. (PHS) 88-1150-2. Washington, DC: Government Printing Office; July, 1988.
34. Fox AJ, Collier PF. Low mortality rates in industrial cohort studies due to selection for work and survival in the industry. *Br J Prev Soc Med.* 1976;30:225-230.
35. Ott MG, Holder BB, Langner RR. Determinants of mortality in an industrial population. *J Occup Med.* 1976;18:171-177.
36. Wen CP, Tsai SP, Gibson RL. Anatomy of the healthy worker effect: a critical review. *J Occup Med.* 1983;25:283-289.
37. Monson RR. Observations on the healthy worker effect. *J Occup Med.* 1986;28:425-433.
38. National Center for Health Statistics. *Health Promotion and Disease Prevention: United States, 1985.* DHHS Publication No. (PHS) 88-1591. Washington, DC: Government Printing Office; 1988.

Managing

Peter Lynch, the legendary former head of Fidelity Investments' Magellan Fund, who now donates his skills to charities, on what to look for:

"I like to buy a company any fool can manage because eventually one will."

—From "Now Hear This" in *Fortune*, 126;14:17 (December 28, 1992)