

# Exhibit 311

## Mortality Surveillance in a Large Chemical Company: The Union Carbide Corporation Experience, 1974-1983

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The mortality experience of 88,000 Union Carbide Corporation employees from 1974 to 1983 is presented using a population-based surveillance system. The study included many long-term employees, with most deaths contributed by those retired or terminated. The total population exhibited 30% lower mortality overall and 10% lower cancer mortality, as compared with the general U.S. population. Excesses of benign neoplasms and malignant melanoma of the skin were observed in both hourly and salaried males. Mortality rates for lymphosarcoma and reticulosarcoma were significantly elevated due to higher rates among hourly male employees and a cluster in one location. This same location also exhibited an excess of liver cancer associated with vinyl chloride operations. There were no other significant excesses in the hourly male workers and fewer deaths than expected due to brain cancer, respiratory cancer, and nonmalignant respiratory diseases. Salaried, and particularly hourly, women experienced favorable mortality, although for the women, time since hire was relatively short. Location-specific findings were similar to what had been observed in the company's previously conducted cohort studies. Future value lies in the development of a database that will have greater power to address possible effects of past exposures and outcomes related to more recent lower level exposures.

**Key words:** chemical workers, mortality surveillance, occupational epidemiology, lymphosarcoma, reticulosarcoma, melanoma of skin

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### INTRODUCTION

Epidemiology studies within industry are typically conducted when there is concern that a certain agent or work environment may be having an adverse effect on the health of exposed workers. Confining efforts to such ad hoc studies unnecessarily restricts the potential value of epidemiology in an occupational setting. By using data normally generated in company operations, supplemented by certain additional in-

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formation, a surveillance system can be developed that can greatly enhance the effectiveness of epidemiological research in a working population.

Surveillance can be described as the ongoing use of resources distinguished by their practicality and rapidity to monitor the health of population groups [Thacker and Berkelman, 1988]. In the occupational setting, surveillance can be used to monitor the health outcomes of a given workforce. It is distinguishable from more focused cohort, case control, or cross-sectional studies, in that specific research questions are not addressed. Since a surveillance system frequently contains information on a very large population, the collection of several data items that address questions of causality is not generally feasible. Rather, a surveillance system can be thought of as a monitoring or screening device that can: (1) provide an indicator of the health status of the workforce; (2) identify unusual disease occurrences that may require additional study to assess work-relatedness; (3) provide data to facilitate more focused research; and (4) enable a rapid response to health issues that may arise.

Occupational surveillance programs have traditionally been based on mortality as an end point. This has resulted from the availability of methods for determining vital status and for obtaining cause of death information. It is ultimately desirable to develop morbidity and medical surveillance programs to detect the early warning signs of an increased occurrence of diseases and to monitor trends in nonfatal diseases. With the exception of cancer incidence surveillance programs, the advances in this area within industry and within the federal government have been limited due to problems of data availability, quality, and confidentiality [Pell et al., 1978; O'Berg et al., 1987; Howe et al., 1980; Committee on Government Operations 1986].

The mortality surveillance program currently in place at Union Carbide Corporation (UCC) has been evolving since the mid-1960s. Initially, information was collected only for decedents; unusual disease occurrence had to be assessed using a proportionate mortality ratio (PMR) approach [Austin, 1981]. Relative distributional changes in disease outcomes could be identified; however, it was not possible to determine whether the changes were due to an elevation of one particular cause of death or to relative deficits in other causes [Kupper et al., 1978].

The mortality surveillance program has now been converted to one that tracks a well-defined employee population and that uses the standardized mortality ratio (SMR) as a measure of risk. This statistic better reflects the true underlying disease rate in the employee population relative to that of a standard control population. The feasibility and utility of this population-based mortality surveillance system were reported using one of the company's divisions [Teta et al., 1987]. Using this approach, the mortality experience of more than 88,000 UCC employees is now presented over a 10-year observation period.

## METHODS AND MATERIALS

The current UCC surveillance population consists of domestic employees who were active in 1974 or hired thereafter and who have completed 1 or more years of company service as of December, 31, 1983. These 88,289 employees have been identified from a series of computerized files maintained by the UCC Benefits Department, beginning in 1974. These files contain demographic and work history data, which are limited to division, location, termination reason, continuous service date, pay point, and wage status at the end of each year.

Data from large cohort mortality studies conducted at three major company locations were used to evaluate independently the completeness of the surveillance population base [Teta and Ott, 1988; Austin and Schnatter, 1983a; Rinsky et al., 1988]. These cohort studies themselves had undergone prior completeness verifications using the UCC quarterly returns to the Social Security Administration (SSA). Using a match based on social security number (SSN), 99% of eligible persons from the studies were found in the surveillance database. The percentage missing is a maximum, since misspecification of SSNs on either file could account for the 1% discrepancy rate.

The surveillance population was followed for mortality from 1974–1983, producing more than 644,000 person-years of observation. Study members contributed person years from 1 year after hire or from January 1, 1974, whichever was more recent, until death, the close of the study, or date last observed (if lost to follow-up). Vital status was ascertained for 94% of the population using files of the SSA, the U.S. National Death Index, the company's mortality register, and the Benefits Department's file of retired and vested employees. Death certificates were obtained for more than 99% of the decedents. They were then coded for underlying cause of death, according to the International Classification of Diseases revision in effect at the time of death, by a trained contract nosologist.

The mortality experience of white and nonwhite employees was compared with that of the general U.S. white population. Information from company records was insufficient to permit race-specific analyses. The data were analyzed using a modified life-table technique and sex-, age-, and calendar year-specific U.S. death rates for 1970–1982 [Marsh and Preninger, 1980]. The summary measure of risk was the ratio of the observed to expected deaths multiplied by 100, that is, the standardized mortality ratio (SMR). Assuming that the observed number of deaths follows a Poisson distribution, 95% confidence intervals (CI) were calculated [Rothman and Boice, 1979].

The total UCC domestic population as well as several subpopulations have been examined for 62 causes of death. These include the four sex/wage-status groups (male hourly workers, female hourly workers, male salaried workers, female salaried workers). For employees active in 1974, no wage status or location assignment data prior to that year were available in the surveillance database. Persons were assigned to the salary category, only if they were never an hourly worker between 1974 and 1983. UCC's three business groups [Chemicals and Plastics (C&P), Industrial Gases, Carbon Products] and their locations with 10 or more deaths were studied separately. Employee assignment to a business group was based on first assignment during 1974–1983. In this report, results are presented for the UCC domestic population and C&P hourly males with reference to relevant location-specific findings.

## RESULTS

The surveillance population included 46,464 hourly and 41,825 salaried employees (Table I). At the end of 1983, approximately 50% were actively employed, 40% had terminated employment, and 10% had retired. There was a large number of long-term male workers in the study group, with almost 60% of all men active in 1974, compared with only 37% of all female employees. At the close of the study observation period, 20 or more years had elapsed since hire for over 20,000 (33%) of

**TABLE I. UCC Surveillance Population Working and Vital Status as of December 31, 1983 by Wage Status**

Working and vital status	Hourly (%)	Salary (%)	
Currently employed	23,339 (50)	22,319 (54)	
Terminated	17,933 (39)	15,490 (37)	
Alive	14,451	12,717	
Dead	634	283	
Unknown status	2,848	2,490	
Retired	4,524 (10)	3,465 (8)	
Alive	4,039	3,230	
Dead	480	228	
Unknown status	5	7	
Died while employed	668 (1)	551 (1)	
Total	46,464	41,825	88,289

the male employees, some of whom joined the company as early as 1925. By contrast, only 10% of the female workforce were 20 or more years from their hire date.

The mortality experience of current and former employees was found to be generally favorable compared with that of the U.S. population. The hourly and salaried total population exhibited 30% lower mortality overall (Table II). There were 2,844 deaths observed (obs.) in this study group and 4,046 expected (exp.) deaths. The cancer death rate was also significantly lower (SMR = 90; 95% CI: 84, 96). The mortality rates for major noncancer causes of death such as heart disease, nonmalignant respiratory disease, and external causes (e.g., accidents, homicides, suicides) were noticeably lower by comparison with the U.S. for all subgroups (Tables II–VI).

In the total population, there was a statistically significant excess of deaths due to malignant melanoma of the skin, based on 32 obs. and 20.3 exp. deaths (SMR = 158; 95% CI: 108, 223) and benign neoplasms, based on 22 obs. and 12.6 exp. deaths (SMR = 175; 95% CI: 110, 265). These causes were elevated in both hourly and salaried male employees and did not exhibit location-specific clusters (Tables III–IV). The excess of malignant melanoma deaths among salaried males was statistically significant (95% CI: 101, 297), based on 15 observed and 8.3 expected deaths.

There was also a statistically significant excess of deaths in the total population due to lymphosarcoma and reticulosarcoma (one of several causes of death within the broader category of lymphatic and hematopoietic tissue cancers), based on 28 obs. and 15.8 exp. deaths (SMR = 177; 95% CI: 118, 256). This finding was attributable to elevated rates among hourly male employees (18 obs. vs. 7.5 exp.) for whom the SMR was 241 (95% CI: 143, 381). When specific locations were examined, a cluster of cases was seen at the South Charleston C&P location in the Kanawha Valley (KV) of West Virginia. This location contributed 5 deaths to this cause of death category (0.8 exp.). These diseases were investigated as part of a joint UCC/National Institute for Occupational Safety and Health study which also identified more deaths than expected due to lymphosarcoma and reticulosarcoma at this same location [Rinsky et al., 1988]. There did not appear to be any other locations with notable excesses of this cause of death.

The overall mortality rate for the 34,795 hourly males was 17% lower than that of U.S. males, while cancer mortality for this group was slightly higher (SMR =

**TABLE II. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Males and Females, 1974–1983 (N = 88,289)\***

Cause of death (ICDA-8)	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes (000–999)	2844/4046.4	70 <sup>b</sup>	68,73
Malignant neoplasms, total (140–209)	927/1029.8	90 <sup>b</sup>	84,96
Digestive organs and peritoneum (150–159)	217/237.8	91	80,104
Large intestine (153)	94/85.9	109	88,134
Biliary passages and liver (155–156)	16/18.0	89	51,144
Pancreas (157)	44/50.4	87	63,117
Respiratory system (160–163)	290/381.1	76 <sup>b</sup>	68,85
Breast (174)	39/39.3	99	71,136
All uterine (180–182)	10/9.5	106	51,194
Other female genital organs (183–184)	11/11.6	95	47,170
Prostate (185)	48/35.6	135	99,179
Kidney and other urinary organs (189)	30/25.7	117	79,167
Bladder (188)	19/18.9	101	61,157
Malignant melanoma of the skin (172.0–172.4, 172.6–172.9)	32/20.3	158 <sup>c</sup>	108,223
Central nervous system (191–192)	32/35.7	90	61,127
Lymphatic and hematopoietic tissue (200–209)	105/94.9	111	91,134
Benign neoplasms (210–239)	22/12.6	175 <sup>c</sup>	110,265
Cerebrovascular disease (430–438)	119/164.3	72 <sup>b</sup>	60,87
All heart disease (390–398, 400.1, 400.9)	1062/1460.0	73 <sup>b</sup>	68,77
Nonmalignant respiratory diseases (460–519)	77/204.1	38 <sup>b</sup>	30,47
External causes of death (800–999)	354/572.5	62 <sup>b</sup>	56,69
All other causes of death	283/603.1	47 <sup>c</sup>	42,53

\*Expected deaths based on U.S. white mortality rates through 1982.

<sup>a</sup>SMR = (observed/expected) × 100.

<sup>b</sup>p < 0.01.

<sup>c</sup>p < 0.05.

106; 95% CI: 97, 115) (see Table III). Other than the finding for lymphosarcoma and reticulosarcoma, there were no statistically significant excesses among hourly males. There were fewer deaths than expected due to central nervous system (CNS) cancer (SMR = 97), respiratory cancer (SMR = 98), and nonmalignant respiratory diseases (SMR = 40). The nonsignificantly elevated numbers of kidney (SMR = 149), prostatic (SMR = 137), and liver (SMR = 129) cancers were accounted for by two specific locations and are described more fully in the results for the C&P Business Group. The nine deaths due to benign neoplasms included six deaths due to brain tumors, two deaths due to polycythemia vera, and one death due to myeloproliferative disorder. The brain tumor cases were each assigned to different locations.

The domestic population included 11,669 hourly female workers and 136 deaths which were a statistically significant deficit (SMR = 69; 95% CI: 58,81) (Table V). This group also exhibited a significantly lower rate of death due to all cancers combined (SMR = 73; 95% CI: 55, 95). There were no unusual disease excesses in this subgroup. The “salaried” female designation included both exempt and nonexempt (secretaries, clerical workers) women. Their total cancer SMR was 97 and there were no significant excesses for any specific causes of death (Table VI). Salaried, but not hourly, women did have higher numbers of deaths due to breast and uterine cancers than expected. Neither group experienced elevated rates of ovarian cancer that was coded to the residual category of female genital cancers.

**TABLE III. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Hourly Males, 1974–1983 (N = 34,795)\***

Cause of death category	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes	1646/1992.8	83 <sup>b</sup>	79,87
Malignant neoplasms, total	510/482.7	106	97,115
Digestive organs and peritoneum	119/115.6	103	85,123
Large intestine	47/40.6	116	85,154
Biliary passages and liver	11/8.5	129	64,230
Pancreas	26/24.6	106	69,155
Respiratory system	190/194.8	98	84,112
Prostate	28/20.4	137	91,198
Kidney and other urinary organs	19/12.8	149	90,232
Bladder	10/10.0	100	48,183
Malignant melanoma of the skin	13/9.3	140	74,239
Central nervous system	16/16.5	97	55,157
Lymphatic and hematopoietic tissue	53/44.9	118	88,154
Benign neoplasms	9/5.7	158	72,301
Cerebrovascular disease	64/78.3	82	63,104
All heart disease	635/750.7	85 <sup>b</sup>	78,91
Nonmalignant respiratory diseases	41/103.7	40 <sup>b</sup>	28,54
External causes of death	222/284.0	78 <sup>b</sup>	68,89
All other causes of death	165/287.7	57 <sup>c</sup>	49,67

\*Expected deaths based on U.S. white male mortality rates through 1982.

<sup>a</sup>SMR = (observed/expected) × 100.

<sup>b</sup>p < 0.01.

<sup>c</sup>p < 0.05.

## BUSINESS GROUPS

### Industrial Gases

There were 12,399 persons in the Industrial Gases Business Group that accounted for 374 deaths. One third of these were employees from the large Tonawanda, N.Y., facility of this Business Group. This research, engineering, and metal fabrication facility was the subject of a cohort study that covered 1946–1981 [Teta and Ott, 1988]. The surveillance study findings, which included far fewer total years of observation, but 2 more years of data, were similar to those reported earlier as part of the previous study at that location. There were no notable findings in the remaining Industrial Gases population who were employed in more than 140 very small gas-packaging and air-separation plants with fewer than 30 employees each.

### Carbon Products

The Carbon Products Business Group served as the pilot for the population-based approach to mortality surveillance and covered the same observation period. The test population was restricted, however, to men with 10 or more years service as of 1974. The more complete surveillance population produced similar findings to those already reported [Teta et al., 1987].

### Chemicals and Plastics (C&P)

The C&P surveillance population included 7,901 hourly and 5,389 salaried males. Both groups experienced significantly lower overall mortality than the corre-



**TABLE IV. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Salary Males, 1974–1983 (N = 29,033)\***

Cause of death category	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes	895/1638.4	55 <sup>b</sup>	51,58
Malignant neoplasms, total	287/394.6	73 <sup>b</sup>	65,82
Digestive organs and peritoneum	77/93.8	82	65,103
Large intestine	34/32.7	104	72,145
Biliary passages and liver	3/6.9	43	9,127
Pancreas	14/20.0	70	38,117
Respiratory system	78/159.0	49 <sup>b</sup>	39,61
Prostate	20/15.2	131	80,203
Kidney and other urinary organs	9/10.7	84	39,160
Bladder	7/7.8	90	36,184
Malignant melanoma of the skin	15/8.3	180 <sup>c</sup>	101,297
Central nervous system	13/14.3	91	49,156
Lymphatic and hematopoietic tissue	41/37.4	110	79,149
Benign neoplasms	9/4.8	189	87,359
Cerebrovascular disease	36/62.6	58 <sup>b</sup>	40,80
All heart disease	359/614.7	58 <sup>b</sup>	53,65
Nonmalignant respiratory diseases	25/81.5	31 <sup>b</sup>	20,45
External causes of death	88/238.1	37 <sup>b</sup>	30,46
All other causes of death	91/242.1	38 <sup>c</sup>	30,46

\*Expected deaths based on U.S. white male mortality rates through 1982.

<sup>a</sup>SMR = (observed/expected) × 100.

<sup>b</sup>p < 0.01.

<sup>c</sup>p < 0.05.

sponding general U.S. population. Salaried males also had lower total cancer death rates. There was, however, a statistically significant excess of deaths due to cancer of the large intestine based on 18 observed and 7.9 expected deaths (SMR = 229; 95% CI: 136, 362). This pattern was not seen in hourly workers and did not cluster in one or a few locations.

There were 200 cancer deaths among C&P hourly males and 174.7 expected (SMR = 114; 95% CI: 99, 132) (Table VII). This included an almost three-fold excess of liver cancer (SMR = 288; 9 obs./3.1 exp.). This finding was also observed in a cohort study of a C&P location and has been related to vinyl chloride operations [Rinsky et al., 1988]. There were no other statistically significant excesses in C&P hourly workers. Fewer deaths than expected were observed due to respiratory cancer (SMR = 91) and nonmalignant respiratory diseases (SMR = 34), among other causes of death. Although not statistically elevated, mortality rates for cancers of the pancreas (SMR = 155), prostate (SMR = 177), kidney (SMR = 174), lymphosarcoma and reticulosarcoma (SMR = 233), CNS cancers (SMR = 127), and malignant melanoma of the skin (SMR = 240) were above expectation in this group. Malignant melanoma mortality rates were somewhat elevated across most C&P facilities, with no unusual clustering by location. The pancreatic and prostatic cancer excesses were confined to one location, CNS cancer to another, and kidney cancer and lymphosarcoma and reticulosarcoma to the location included in the Kanawha Valley (KV) Study. These excesses were either statistically significant or of borderline significance at the location level and have already been investigated or are



**TABLE V. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Hourly Females, 1974–1983 (N = 11,669)\***

Cause of death category	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes	136/198.1	69 <sup>b</sup>	58,81
Malignant neoplasms, total	54/74.0	73 <sup>c</sup>	55,95
Digestive organs and peritoneum	9/13.5	67	31,127
Large intestine	5/6.0	83	27,195
Biliary passages and liver	2/1.2	—	20,589
Pancreas	2/2.7	—	9,264
Respiratory system	10/13.3	75	36,138
Breast	13/19.0	69	37,117
All uterine	3/4.6	—	13,190
Other female genital organs	5/5.7	89	29,206
Kidney and other urinary organs	1/1.1	—	2,516
Bladder	1/0.4	—	5,1159
Malignant melanoma of the skin	2/1.3	—	18,548
Central nervous system	0/2.4	—	0,155
Lymphatic and hematopoietic tissue	6/6.0	100	37,217
Benign neoplasms	3/1.0	288	60,843
Cerebrovascular disease	8/11.0	73	31,143
All heart disease	34/43.9	78	54,108
Nonmalignant respiratory diseases	4/8.9	45	12,115
External causes of death	24/24.1	99	64,148
All other causes of death	9/35.2	26 <sup>c</sup>	12,48

\*Expected deaths based on U.S. white female mortality rates through 1982.

<sup>a</sup>SMR = (observed/expected) × 100. Not calculated when observed and expected numbers of deaths are <5.

<sup>b</sup>p <0.01.

<sup>c</sup>p <0.05.

currently under investigation for possible work relatedness as part of more detailed studies at these locations [Ott et al., 1989a,b; Austin and Schnatter, 1983a].

## DISCUSSION

Examination of the total domestic corporation is primarily useful as an indicator of the general health status of the company's employees. It also addresses the issue of whether the same agent in multiple locations has caused increases in mortality or whether multiple agents have been influencing similar adverse health effects. The mortality experience of hourly males is the most informative for the detection of possible work-related effects. The patterns observed in this investigation do not indicate widespread cancer increases as a result of working with chemicals. Overall cancer mortality was comparable to that of the United States. This is similar to what has been seen in other large industrial cohorts [Bond et al., 1985; Bond et al., 1987b; Divine et al., 1985; Pifer et al., 1986; Hanis et al., 1985].

The generally favorable mortality findings, particularly from noncancer causes of death, reflect, in part, the known "healthy worker effect" but may also result from a healthier life-style and greater access to medical care, which are consequences of continued employment [Fox and Collier, 1976; Ott et al., 1976; Wen et al., 1983]. The bias associated with the healthy worker effect has been found to diminish as time

**TABLE VI. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Salary Females, 1974–1983 (N = 12,792)\***

Cause of death category	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes	167/217.1	77 <sup>b</sup>	66,90
Malignant neoplasms, total	76/78.5	97	76,121
Digestive organs and peritoneum	12/15.0	80	41,140
Large intestine	8/6.7	120	52,237
Biliary passages and liver	0/1.4	—	0,271
Pancreas	2/3.1	—	8,236
Respiratory system	12/14.0	86	44,150
Breast	24/19.3	124	80,185
All uterine	7/4.9	144	58,297
Other female genital organs	6/6.0	100	37,220
Kidney and other urinary organs	1/1.2	—	2,475
Bladder	1/0.6	—	5,996
Malignant melanoma of the skin	2/1.3	—	18,542
Central nervous system	3/2.5	—	25,348
Lymphatic and hematopoietic tissue	5/6.6	76	25,177
Benign neoplasms	1/1.1	—	2,500
Cerebrovascular disease	11/12.4	89	44,159
All heart disease	34/50.8	67 <sup>c</sup>	46,94
Nonmalignant respiratory diseases	7/10.0	70	28,144
External causes of death	20/26.3	76	47,118
All other causes of death	18/38.0	47 <sup>c</sup>	28,75

\*Expected deaths based on U.S. male mortality rates through 1982.

<sup>a</sup>SMR = (observed/expected) × 100. Not calculated when observed and expected numbers of deaths are < 5.

<sup>b</sup>p < 0.01.

<sup>c</sup>p < 0.05.

from hire increases and as the workforce ages [Fox and Collier, 1976]. It is also of less importance in interpreting cancer mortality findings [Enterline, 1975; McMichael, 1976; Monson, 1986]. In a comparison of several large cohort studies, Monson observed that the lowest SMRs occurred for nonmalignant respiratory disease during the first 10 years of observation [Monson, 1986]. The SMR for rubber workers was 40 during this period, the same as that for the surveillance population of hourly males. The importance of including nonactive employees to minimize potential selection biases has also been pointed out [Wen et al., 1983]. The surveillance population included both retired and other terminated employees, who together contributed 47% of the population and 57% of the deaths. To date, the follow-up period covers only 10 years, yet approximately one third of the males were over 50 years of age and 20+ years from hire. Those active in 1974 were a censored population, thus the mortality experience of those who terminated prior to that year is unknown. Information on mortality at ages greater than 74 years and with longer latency will only become available as the cohort ages.

The ability to detect small increases in risk in a subset of a large plant population is limited using the surveillance methodology. The smallest population examined is hourly males at a given location, most of whom are unlikely to have been exposed to a particular suspect agent and who have potential exposures to a multitude of agents. A special studies component is therefore utilized to target specific exposure or dis-

**TABLE VII. UCC Mortality Surveillance Observed and Expected Deaths by Cause: Hourly Males, 1974–1983: Chemicals and Plastics (N = 7,901)\***

Cause of death category	Observed/expected deaths	SMR <sup>a</sup>	95%CI
All causes	589/685.8	86 <sup>b</sup>	79,93
Malignant neoplasms, total	200/174.7	114	99,132
Digestive organs and peritoneum	48/42.4	113	83,150
Large intestine	16/14.9	107	61,174
Biliary passages and liver	9/3.1	288 <sup>b</sup>	132,547
Pancreas	14/9.0	155	85,260
Respiratory system	65/71.7	91	70,116
Prostate	14/7.9	177	97,297
Kidney and other urinary organs	8/4.6	174	75,343
Bladder	4/3.8	—	29,269
Malignant melanoma of the skin	7/2.9	240	97,495
Central nervous system	7/5.5	127	51,262
Lymphatic and hematopoietic tissue	21/15.2	138	85,211
Benign neoplasms	4/1.9	—	56,527
Cerebrovascular disease	16/28.7	56 <sup>c</sup>	32,91
All heart disease	243/274.1	89	78,101
Nonmalignant respiratory diseases	13/38.4	34 <sup>b</sup>	18,58
External causes of death	55/69.4	79	60,103
All other causes of death	58/98.6	59 <sup>c</sup>	45,76

\*Expected deaths based on U.S. white male mortality rates through 1982.  
\*SMR = (observed/expected) × 100. Not calculated when both observed and expected numbers of deaths are <5.  
<sup>b</sup>p <0.01.  
<sup>c</sup>p <0.05.

eased subgroups for hypothesis testing. The surveillance database serves as a useful adjunct to this effort by identifying deaths and providing information on vital status and general employment history.

Large increases in mortality, particularly for a rare disease, in a plant subpopulation can often be observed in an examination of the data for the entire plant population, even over a restricted risk period. The known excess of angiosarcoma of the liver associated with vinyl chloride exposures at the South Charleston facility was clearly evident in the surveillance database. There was an almost sixfold excess of malignant neoplasms of the biliary passages and liver among hourly males at the C&P South Charleston location (SMR = 592; 95% CI: 217, 1289). This cause of death category also exemplifies the dilution of important effects that can occur when the at-risk population is overwhelmed by persons not exposed to the risk factor. There was a deficit of neoplasms of the biliary passages and liver within the total company population of hourly and salaried employees (SMR = 89); a nonsignificant elevation in hourly males (SMR = 129; 95% CI: 64, 230); and a statistically significant excess in hourly males from the C&P Business Group (SMR = 288; 95% CI: 123, 547).

The excess of deaths due to cancers of the breast and uterus among salaried females are consistent with the findings of Roman et al. [1985], who noted elevated mortality rates due to cancers of the breast, corpus uteri, and ovary among professional and clerical workers. This was attributed to a likely high proportion of nulliparous women and women who delay childbearing in these groups. Nulliparity and

older age at first birth have been associated with increased risk of these cancers [Threlfall et al., 1985].

In the Dow female surveillance population, breast cancer mortality rates were elevated for hourly and nonexempt women, and ovarian cancer mortality for hourly and exempt women [Bond et al., 1987a]. There was a deficit of uterine cancer mortality in hourly and nonexempt women. Breast, cervical, and other genital cancer incidence rates were elevated for salaried active female employees in the Du Pont Company [O'Berg et al., 1987]. Breast cancer incidence was not higher than expected in the hourly women. Breast, cervix uteri, corpus uteri, and ovarian cancer incidence rates were significantly elevated for Connecticut cosmetologists who were first licensed between 1925 and 1934 [Teta et al., 1984]. These also may have been women with low parity and/or later age at first childbirth.

In the present investigation, hourly women exhibited fewer deaths than expected from all three causes, in contrast to the salaried women. Interstudy comparisons are complicated by different groupings of female genital cancers and wage status subsets and varying employee inclusion criteria. It is reasonable to conclude, however, that there are differences in the frequency of hormonally related cancers between classes of working women and between working women and the general female population that may confound attempts to relate occupational risk factors to these diseases.

Employee race data were unavailable for the surveillance population whose mortality rates were compared to those of the U.S. white population. In 1977, 21% of the company workforce was nonwhite. Inclusion of nonwhites in the study group would tend to artificially inflate SMRs for all causes, total cancer and specific cancers for which nonwhites have higher mortality (esophagus, stomach, lung, pancreas, prostate, multiple myeloma) [Schottenfeld and Fraumeni, 1982].

Smoking data on individuals were also unavailable. The frequency of lung cancers in hourly males (190) was slightly less than expected (194.8). This could result if these employees smoked less than U.S. white males. Smoking has traditionally been restricted in chemical plants for safety reasons, raising the possibility that this may have occurred. However, an examination of medical records from the six major C&P plants from 1978 to 1987, for men under surveillance in accordance with the Occupational Safety and Health Administration (OSHA) asbestos standard, suggested similar smoking patterns as the general U.S. population [U.S. Department of Health, Education and Welfare, 1980].

The inclusion of long-term employees in the surveillance population (primarily those active in 1974) provided the ability to detect those excesses seen in the company's comprehensive cohort studies, which collected more detailed information and were therefore more costly and time consuming efforts. Since these studies have been completed within the past few years at most major locations, the present analysis of the surveillance data was not expected to identify previously unknown excesses [Austin and Schnatter, 1983a,b; Teta et al., 1987; Teta and Ott, 1988; Rinsky et al., 1988]. Its present value lies primarily in providing an assessment of the mortality experience of the entire workforce and adding a few years of additional information beyond the observation period of the completed cohort studies. Future value lies in the development of a database which will have greater power to address the impacts of exposures in the past, but also possible effects of lower levels of exposure for those workers hired more recently.

It is not always necessary, however, to observe those exposed at lower levels for many years, since the absence of effects at presumably higher levels in the past can provide reassurance that those hired more recently will not be adversely affected. This is limited to processes which started up many years ago and are still operational. A progressive chemical company continually discontinues outdated technologies and develops new operations and products. These changes and the long latency period associated with most cancers require a commitment to maintain a surveillance system for many years.

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## REFERENCES

- Austin SG (1981): An industry-sponsored mortality surveillance program. *Banbury Rep* 9:347–358.
- Austin SG, Schnatter AR (1983a): A case-control study of chemical exposures and brain tumors in petrochemical workers. *J Occup Med* 25:313–320.
- Austin SG, Schnatter AR (1983b): A cohort mortality study of petrochemical workers. *J Occup Med* 25:304–312.
- Bond GG, Shellenberger RJ, Fishbeck WA, Cartmill JB, Lasich BJ, Wymer KT, Cook RR (1985): Mortality among a large cohort of chemical manufacturing employees. *J Natl Cancer Inst* 75: 859–869.
- Bond GG, McLaren EA, Cartmill JB, Wymer KT, Lipps TE, Cook RR (1987a): Mortality among female employees of a chemical company. *Am J Indust Med* 12:563–578.
- Bond GG, McLaren EA, Cartmill JB, Wymer KT, Sobel W, Lipps TE, Cook RR (1987b): Cause-specific mortality among male chemical workers. *Am J Indust Med* 12:353–383.
- Committee on Government Operations (1986): Occupational Health Hazard Surveillance: 72 Years Behind and Counting. Sixty-First Report. House Report 99–979. Washington, DC: Government Printing Office.
- Divine BJ, Barron V, Kaplan SD (1985): Texaco mortality study I. Mortality among refinery, petrochemical and research workers. *J Occup Med* 27:445–447.
- Enterline PE (1975): Not uniformly true for each cause of death. *J Occup Med* 17:127–128.
- Fox AJ, Collier PF (1976): Low mortality rates in industrial cohort studies due to selection for work and survival in the industry. *Br J Prev Soc Med* 30:225–230.
- Hanis NM, Shallenberger LG, Donaleski DL, Sales EA (1985): A retrospective mortality study of workers in three major U.S. refineries and chemical plants. Part I. Comparison with the U.S. population. *J Occup Med* 27:283–292.
- Howe GR, Lindsay J, Miller AB (1980): Cancer incidence and mortality in relation to occupation in 700,000 members of the Canadian labour force. *Cancer Detect Prev* 3:487–497.
- Kupper LL, McMichael AJ, Symons MJ, Most BM (1978): On the utility of proportional mortality analysis. *J Chron Dis* 31:15–22.
- Marsh GM, Preninger M (1980): OCMAP: A user-oriented occupational cohort mortality analysis program. *Am Stat* 34:245–246.
- McMichael AJ (1976): Standardized mortality ratios and the “healthy worker effect”: Scratching beneath the surface. *J Occup Med* 18:165–168.
- Monson RR (1986): Observations on the healthy worker effect. *J Occup Med* 28:424–433.
- O’Berg MT, Burke CA, Chen JL, Walrath J, Pell S, Gallie CR (1987): Cancer incidence and mortality in the Du Pont company: An update. *J Occup Med* 29:245–252.
- Ott MG, Holder BB, Langner RR (1976): Determinants of mortality in an industrial population. *J Occup Med* 18:171–177.
- Ott MG, Teta MJ, Greenberg HL (1989a): Assessment of exposure to chemicals in a complex work environment. *Am J Ind Med* 16:617–630.

- Ott MG, Teta MJ, Greenberg HL (1989b): Lymphatic and hematopoietic tissue cancer in a chemical manufacturing environment. *Am J Ind Med.* 16:631–643.
- Pell S, O’Berg MT, Karrh BW (1978): Cancer epidemiologic surveillance in the Du Pont company. *J Occup Med* 20:725–740.
- Pifer JW, Hearne FT, Friedlander BR, McDonough JR (1986): Mortality study of men employed at a large chemical part, 1972 through 1982. *J Occup Med* 28:439–444.
- Rinsky RA, Ott MG, Ward E, Greenberg HL, Halperin W, Leet T (1988): Study of mortality among chemical workers in the Kanawha Valley of West Virginia. *Am J Indust Med* 13:429–438.
- Roman E, Beral V, Inskip H (1985): Occupational mortality among women in England and Wales. *Br Med J* 291:194–196.
- Rothman KJ, Boice JD (1979): *Epidemiologic analysis with a programmable calculator.* Washington, DC: US Department of Health, Education and Welfare, 1979. (DHEW publication No. (NIH) 79-1649).
- Schottenfeld D, Fraumeni JF (eds) (1982): “*Cancer Epidemiology and Prevention.*” Philadelphia: WB Saunders Co.
- Teta MJ, Ott MG (1988): A mortality study of a research, engineering, and metal fabrication facility in Western New York State. *Am J Epidemiol* 127:540–51.
- Teta MJ, Walrath J, Meigs JW, Flannery JT (1984): Cancer incidence among cosmetologists. *J Natl Cancer Inst* 72:1051–1057.
- Teta MJ, Ott MG, Schnatter AR (1987): Population-based mortality surveillance in carbon products manufacturing plants. *Br J Ind Med* 44:344–350.
- Thacker SB, Berkelman RL (1988): Public health surveillance in the United States. *Epidemiol Rev* 10:164–190.
- Threlfall WJ, Gallagher RP, Spinelli JJ, Band PR (1985): Reproductive variables as possible confounders in occupational studies of breast and ovarian cancer in females. *J Occup Med* 27:448–450.
- US Department of Health, Education and Welfare (1980): DHEW publication No. (PHS) 80-1232. Washington, DC: Government Printing Office.
- Wen CP, Tsai SP, Gibson RL (1983): Anatomy of the healthy worker effect: A critical review. *J Occup Med* 25:283–289.