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Study of Mortality Among Chemical Workers in the Kanawha Valley of West Virginia

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To assess the mortality experience of a cohort of chemical workers in the Kanawha Valley of West Virginia, 29,139 males who worked at any one of three facilities over a 39-year period were followed-up for vital status. The facilities include two chemical manufacturing plants and a research and development center. From this cohort, 5,785 men were found to have died as of the study end, December 31, 1978. This was less than the 6,148.5 men expected to have died, based upon the United States white male population (standardized mortality ratio (SMR) = 94, 95% confidence interval (CI) = 92–96). Eighty-six specific causes of death were examined. Statistically significant increased deaths were observed for two causes; cancers of the liver (not specified as primary or secondary) (SMR = 174; CI = 102–280) and lympho- and reticulosarcoma (SMR = 140; CI = 104–187). When all biliary and liver cancer was examined by duration and time since initial employment, the SMR for those who worked at least 25 years and whose deaths occurred 30 years or more after first employment was 301 (95% confidence limit = 168–497). The identification and follow-up of this complete cohort provides the basis for future study of subcohorts with specific chemical and process exposures and case control studies of specific causes of death.

Key words: liver cancer, lymphosarcoma, reticulosarcoma, vinyl chloride

INTRODUCTION

In 1979, researchers from the Union Carbide Corporation (UCC) and the National Institute for Occupational Safety and Health (NIOSH) agreed to conduct a study of the mortality experience of Union Carbide Corporation employees at three facilities in the Kanawha Valley of West Virginia: the South Charleston plant, the Institute plant, and the Technical Center.

The South Charleston plant began operations in 1925, around the process of stripping ethylene from natural gas. It evolved into a chemical and plastics facility, producing a wide variety of chemical substances, including ethylene oxide, polyeth-

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ylene, vinyl chloride resins, and polyols. The Institute plant was originally built by the U.S. Government (U.S. Rubber Reserve Corporation) to produce styrene/butadiene rubber to replace the supply of natural rubber cut off during World War II. This facility was bought by Union Carbide in 1947. The plant was then used as a larger production facility for materials developed at the South Charleston plant, such as acetone, isopropanol, butanol, and acetaldehyde. In recent years, the Institute plant has grown to include the manufacture of agricultural chemicals and a wide range of ethylene oxide and propylene oxide based products. The Technical Center was built in 1949 as a facility to house one of UCC's chemical and plastics research departments. In 1958, the facility was expanded to include a development and engineering department.

There were a number of reasons for undertaking a study of the three UCC facilities, including an interest in the mortality experience of employees exposed to ethylene oxide and others who worked in a coal hydrogenation process. Accurate identification of these subgroups from the UCC workforce required review of payroll records of approximately 42,000 individuals. In addition, there was interest in conducting a study of the overall mortality experience at the plants, following a preliminary epidemiologic study that found disproportionate cancer mortality among 819 deceased Union Carbide employees [Marsh, 1979] and concerns that chemical workers in general might be at increased risk of brain and other cancers [Thomas et al., 1980]. Since its inception, the study has involved cooperation among UCC, NIOSH researchers, and two locals of the International Association of Machinists and Aerospace Workers that represent workers at the plants.

This report is the first in a series that will examine the mortality patterns of UCC chemical workers in the Kanawha Valley of West Virginia. It reports the mortality experience of the chemical workers without reference to either process or exposure. It will be followed by reports on cohorts with specific exposures to chemicals and processes, and by case-control analyses of persons who died of causes of death of interest.

METHODS

The study population consisted of all salaried and hourly male employees ever employed at three Union Carbide facilities in the Kanawha Valley of West Virginia (South Charleston, Institute, and the Technical Center). Historical records of employment, consisting primarily of pay location cards, were reviewed and abstracted by UCC personnel. Name, social security number, sex, date of birth, overall begin and end employment dates, and indication of each unique department worked were abstracted to code sheets. The total cohort consisted of 41,127 individuals. There were 1,105 (3.7%) persons whose sex was not known, 92 of whom were judged to be female on the basis of first name. The remainder were assumed to be male. Race was recorded on only 47.4% of the records. Among those men for whom race was recorded, 96.1% were white, 3.4% were black, and 0.5% were of other race or ethnic groups. Because of the high percentage of whites among those whose race was known, the mortality experience of the entire population was compared with the corresponding white male population.

We excluded from the analysis 7,408 individuals who terminated employment prior to January 1, 1940, because of the difficulty in determining vital status for individuals without Social Security numbers. We also excluded 4,579 individuals who were female. The reason for excluding this large number of females was the difficulty in vital status follow-up and the impression that until recent years most were not employed in the chemical process areas. The final study cohort consists of 29,139 males who worked at least 1 day any time between January 1, 1940 and December 31, 1978.

For individuals whose vital status as of December 30, 1978 was not known from UCC personnel records, vital status was determined through the Social Security Administration, the West Virginia Department of Motor Vehicles, and by reviewing listings of war deaths from West Virginia (West Virginia Blue Book—Honor List of Dead and Missing). Death certificates were obtained for persons known to be deceased. Underlying causes of death were determined from death certificate indication by a qualified nosologist, according to the rules of the International Classification of Disease in effect at the time of death. When no death certificate was available, the cause of death was considered "unkown."

We used the NIOSH Life Table Analysis System to generate expected numbers of cause-specific deaths based on person-years at-risk (PYARs) of dying within 5-year age and 5-year calendar time intervals [Waxweiler et al., 1983]. PYARs were accumulated for an individual starting on his first day of employment or January 1, 1940, whichever was later, and continued to accumulate until the date of death or December 31, 1978, whichever was earlier. For individuals whose vital status could not be determined, PYARs were accumulated only until the last date they were observed alive (usually their last day of work). Age- and calendar-year-specific US white male death rates were used as the referent rates. Applying the referent rates to the total PYARs in each age and calendar time period yielded expected numbers of cause-specific deaths. The number of observed deaths was divided by the number of expected deaths to provide a standardized mortality ratio (SMR). Upper and lower 95% confidence intervals (CIs) were developed, assuming a Poisson distribution. SMRs and confidence intervals were calculated for 86 causes of death.

For selected causes of death, PYARs were divided into 5-year duration of employment and latency periods (time from first day employed to date of death or study end date). A category of less than 1 year employed was added, so that expected and observed deaths for persons with this minimal exposure could be considered separately.

RESULTS

There were 29,139 males who worked at least 1 day at one of the three facilities between January 1, 1940 and December 31, 1978. (Table I) These individuals contributed a total of 691,183 PYARs. About 20% of the cohort members worked less than 1 year. Approximately 54% of the cohort had greater than 5 years' duration of employment. Vital status is known for 28,104 individuals, 96.4% of the study cohort. Of the 5,785 men known to be deceased as of the study end date (December 31, 1978), death certificates were obtained and causes of death determined for 5,521 (95.4%). Of the 6,148.4 deaths expected to have occurred in this cohort, 1265.9 (20.6%) were in persons with greater than 25 years of latency and duration of employment (Table II).

The number of deaths (5,785) that occurred was less than the 6,148.5 deaths

TABLE I. Distributions of Year First Employed and Total Years of Employment (persons with at least one day of employment from January 1, 1940 to December 31, 1978)

Years	Alive	Deceased	Unknown	Total
Year first employed				
Before 1930	295	495	11	801
1930 to 1940	1,575	1,422	56	3,053
1940 to 1950	8,024	3,249	539	11,812
1950 to 1960	5,754	501	253	6,508
1960 to 1970	3,756	96	136	3,988
1970 through 1978	2,915	22	40	2,977
Total	22,319	5,785	1,035	29,139
Total years employed				
< 1	4,242	1,216	484	5,942
1–4	5,983	1,169	323	7,475
5–9	2,481	702	112	3,295
10–14	1,760	577	49	2,386
15–19	1,126	524	35	1,685
20–24	1,422	483	11	1,196
> 25	5,305	1,114	21	6,440
Total	22,319	5,785	1,035	29,139

TABLE II. Distribution of Expected Number of Deaths (latency by duration of employment)

	Duration of employment (years)							
Latency	< 1	1–4	5–9	10–14	15–19	20–24	> 25	Total
< 5	145	186						331
5–9	120	78	224					423
10–14	150	96	56	262				564
15–19	198	124	73	64	312			771
20–24	253	146	84	75	77	349		985
25–29	279	150	86	77	82	85	340	1,100
> 30	264	162	162	151	153	155	925	1,974
Total	1,140	942	686	629	625	590	1,266	6,148

TABLE III. Summary of Observed and Expected Deaths in Kanawha Valley Chemical Workers (comparison rates or proportions in use: US deaths)

	Observed	Expected		95% CI	
Cause of death	deaths	deaths	SMR	Lower	Upper
All deaths	5,785	6,148.5	94	92	96
Malignant neoplasms	1,083	1,158.7	93	88	99
Of brain and other nervous system	28	41.2	67	45	98
Of biliary passages and liver specified as primary	22	19.5	112	70	171
Of liver not specified as primary or secondary	17	9.7	174	102	280
Lymphosarcoma and reticulosarcoma	48	34.0	140	104	187

TABLE IV. Liver Cancer Deaths. Latency by Duration of Employment

	Duration of employment (years)							
Latency[a]	Under 1	1–5	5–10	10–15	15–20	20–25	> 25	Total
< 5	1/0.44[b]	1/0.53						2/0.97
	(227)[c]	(189)						(206)
5–10	1/0.50	0/0.31	0/0.81					1/1.62
	(200)	—	—					(61)
10–15	0/0.68	0/0.42	1/0.27	1/1.10				2/2.48
	—	—	(370)	(91)				(81)
15–20	1/0.93	0/0.57	0/0.35	0/0.34	1/1.42			2/3.61
	(107)	—	—	—	(70)			(55)
20–25	0/1.21	0/0.69	2/0.41	0/0.37	0/0.43	2/1.66		4/4.78
	—	—	(488)	—	—	(120)		(84)
25–30	1/1.36	1/0.73	0/0.42	0/0.37	1/0.43	0/0.47	1/1.70	4/5.50
	(73)	(137)	—	—	(232)	—	(59)	(73)
Over 30	0/1.33	1/0.82	2/0.84	3/0.76	1/0.76	2/0.77	15/4.99	24/10.28
	—	(122)	(238)	(395)	(131)	(259)	(301)	(234)
Total	4/6.46	3/4.09	5/3.11	4/2.95	3/3.03	4/2.91	16/6.68	39/29.24
	(62)	(73)	(160)	(135)	(99)	(137)	(239)	(133)

[a]Time from first employment to date of death or study end date.
[b]Observed/expected death.
[c](SMR)

expected (SMR = 94; CI = 92–96) (Table III). There were 1,083 observed cancer deaths, compared with 1,158.7 expected (SMR = 93, CI = 88–99). In most cause-specific categories, there were fewer deaths than expected. This reflects the phenomenon often referred to as the healthy worker effect. Of the 86 causes of death categories examined, 19 showed statistically significant deficits of deaths. Among these causes was the death category "malignant neoplasms of the brain and other parts of the nervous system," a category of a priori concern in this study.

The SMR for malignant neoplasms of the liver and biliary passages was 112 (CI = 70–170). The SMR for malignant neoplasms of the liver, not specified, was 174 (CI = 102–280). The SMR for these two categories combined was 133 (CI = 95–182).[1] Henceforth, we will refer to the two categories combined as "liver cancer."

An excess in liver cancer deaths was observed among persons who worked at least 25 years and who died 30 years or more after first employment (Table IV). Among these persons there were 15 observed cases vs. 4.99 expected (SMR = 301; CI = 168–497). The excess liver cancer deaths appeared in the time period 1970 through 1978 (24 observed vs. 12.96 expected, SMR = 185; CI = 119–275) (Table V). The largest risk appeared between 1975 and 1978, the last time period examined in the study (15 observed vs. 6.23 expected; SMR = 241; CI = 135–397). However,

[1]The category "malignant neoplasm of biliary passages and liver" includes the following codes: ICD 46F in the fifth revision, 155 and 156A in the sixth revision, 155 in the seventh revision, and 155 and 156 in the eighth revision. The category "malignant neoplasm of the liver, not specified" includes the following codes: ICD 156A in the seventh revision and 197.8 in the eighth revision. We have combined these two categories because the same trends with time, duration, and latency were present in both and because there seems to be considerable overlap between the classifications. For example, angiosarcomas of the liver have been coded under both categories [Baxter and Fox, 1975].

TABLE V. Cancer Deaths by Age and Calendar Time

	Year Span			
Age	1940–1969	1970–1974	1975–1978	Total
< 45	0/2.0[a]	0/0.27	0/0.08	0/2.40
	—	—	—	—
45–49	3/1.76	1/0.40	0/0.25	4/2.42
	(170)[b]	(250)	—	(165)
50–54	4/2.5	1/0.75	0/0.59	5/3.85
	(160)	(133)	—	(130)
55–59	3/2.93	2/1.19	0/0.91	5/5.03
	(102)	(168)	—	(99)
60–64	2/2.73	0/1.40	7/1.29	9/5.43
	(73)	—	(543)	(166)
65–69	0/2.10	3/1.15	4/1.27	7/4.53
	—	(261)	(315)	(155)
70–74	1/1.36	0/0.77	2/0.91	3/3.04
	(73)	—	(220)	(100)
75–79	2/0.63	2/0.51	1/0.52	5/1.67
	(317)	(392)	(192)	(301)
> 80	0/0.19	0/0.29	1/0.33	1/0.83
	—	—	(303)	(120)
Total	15/16.28	9/6.73	15/6.23	39/29.24
	(92)	(134)	(241)	(133)

[a]Observed/expected deaths.
[b](SMR)

while this pattern of excess liver cancer deaths was apparent for time period, it was not apparent for age group.

As a preliminary assessment of the liver cancer excess observed in this analysis, the 1,195 persons who had been assigned to the four vinyl chloride resins departments of the South Charleston plant were identified. The vital status was known for 1,164 (97.4%) persons, 253 (21.2%) of whom were deceased. Expected liver cancer deaths were calculated from this subcohort of 1,195 persons. A total of 12 liver cancer deaths were observed, with only 1.2 expected.

SMRs for lymphosarcoma and reticulosarcoma were significantly in excess, with 48 observed deaths vs. 34.04 expected (SMR = 140; CI = 104–187) (Table III). When deaths from lymphosarcoma and reticulosarcoma were examined in greater detail, elevated SMRs were observed in most employment duration and latency strata, but no apparent trends with increasing employment duration or latency were observed (Table VI). Similarly, for these two categories, excess deaths were distributed over most time periods and age groups examined.

In addition to analyzing deaths from liver cancer and lympho-reticulosarcoma by latency, duration of employment, age, and calendar time period, we similarly examined 55 other causes of death (Table VII). For these causes of death, no other excess risk or trends were readily apparent.

DISCUSSION

Surveillance has been described as a practice composed of three elements: 1) detecting and reporting cases; 2) analyzing and synthesizing the incoming reports into

TABLE VI. Lymphosarcoma and Reticulosarcoma Deaths. Latency by Duration of Employment

	Duration of employment (years)							
Latency[a]	Under 1	1–5	5–10	10–15	15–20	20–25	> 25	Total
< 5	1/0.36[b]	1/0.57						2/0.93
	(280)[c]	(173)						(214)
5–10	0/0.55	0/0.36	2/0.88					2/1.80
	—	—	(226)					(110)
10–15	1/0.86	0/0.55	2/0.25	1/1.24				4/2.90
	(116)	—	(811)	(80)				(137)
15–20	3/1.16	0/0.73	0/0.39	0/0.31	2/1.74			5/4.34
	(257)	—	—	—	(114)			(115)
20–25	1/1.52	1/0.87	1/0.47	2/0.39	1/0.40	4/2.16		10/5.81
	(65)	(114)	(214)	(515)	(251)	(185)		(172)
25–30	2/1.73	0/0.92	2/0.49	1/0.40	0/0.43	2/0.47	5/2.21	12/6.64
	(115)	—	(411)	(252)	—	(421)	(226)	(180)
Over 30	0/1.66	1/1.02	1/0.96	3/0.83	1/0.77	1/0.78	6/5.58	13/11.61
	—	(97)	(103)	(362)	(129)	(127)	(107)	(112)
Total	8/7.85	3/5.03	8/3.44	7/3.17	4/3.34	7/3.42	11/7.79	48/34.04
	(101)	(59)	(232)	(221)	(119)	(204)	(141)	(141)

[a]Time from first employment to date of death or study end.
[b]Observed/expected deaths.
[c](SMR)

an understanding of the situation; and 3) acting appropriately [Millar, 1984]. Employers are showing increased interest in developing surveillance systems from the records they maintain on agents, workplace conditions, and indices of employees' health [Halperin and Frazier, 1985]. This report describes the mortality experience of a large cohort of chemical workers employed by a single corporation at three localities within the Kanawha Valley of West Virginia. Given the diversity of jobs and exposures in a group of complex chemical plants, there should be little expectation that such an effort would detect associations of disease and employment, should they exist. More directed analyses are needed that take into account the employee's specific exposures, duration of exposure, and latency since initial exposure. This study has provided an overall assessment of the mortality experience of employees of Union Carbide in the Kanawha Valley and a foundation for future surveillance activities. In addition, two causes of death have been identified as being in excess.

One cause of death found to be in excess was cancer of the liver (including angiosarcoma). It is known that vinyl chloride departments were operated at the South Charleston facility and that cases of angiosarcoma of the liver have occurred among South Charleston employees. In a preliminary assessment of the liver cancer excess observed in this analysis, expected liver cancer deaths were calculated for the persons who had been assigned to the vinyl chloride departments. A total of 12 liver cancer deaths were observed, with only 1.2 expected. They occurred primarily in persons with greater than 10 years of employment in vinyl chloride resins production, indicating that this could very well be the cause of the excess liver cancer seen in the overall cohort. The vinyl chloride units were partially discontinued in the mid 1970s and entirely by 1982. Since follow-up of this cohort stops in 1978, it is not yet known if the rate of liver cancer continues in excess to the present. Although the preliminary assessment indicated that the liver cancer excess was largely associated with vinyl

TABLE VII. Summary of Observed and Expected Deaths in Kanawha Valley Chemical Workers (comparison rates or proportions in use: US deaths)

Cause of death	Observed deaths	Expected deaths	SMR	95% CI	
				Lower	Upper
Tuberculosis	41	76.5	53	38	73
Malignant neoplasms	1,083	1,158.7	93	88	99
Of buccal cavity and pharynx	28	39.0	71	48	104
Lip	1	1.2	83	2	463
Tongue	6	9.2	64	24	141
Buccal cavity	8	10.3	77	33	153
Pharynx	13	18.2	71	38	122
Of digestive organs and peritoneum	285	332.5	85	76	96
Esophagus	21	27.2	77	48	118
Stomach	52	65.6	79	59	104
Intestine except rectum	87	103.4	84	67	104
Rectum	24	38.1	62	40	94
Biliary passages and liver	22	19.5	112	70	171
Liver not specified	17	9.7	174	102	280
Pancreas	59	63.5	92	71	120
Peritoneum and digestive organs	3	5.4	55	11	161
Of respiratory system	365	387.3	94	85	104
Larynx	6	18.0	33	12	72
Trachea, bronchus, and lung	352	364.8	96	87	107
Other parts of respiratory system	7	4.5	156	63	323
Of breast	1	1.8	54	1	303
Of male genital organs	73	72.2	101	79	127
Prostate	70	62.2	112	88	142
Other male genital organs	3	9.9	30	6	88
Of urinary organs	57	62.1	91	69	119
Kidney	30	29.6	101	68	144
Bladder and other urinary organs	27	32.4	83	55	121
Of other and unspecified sites	145	147.5	98	83	116
Skin	26	22.8	113	74	167
Eye	0	1.1	—	—	—
Brain and other nervous system	28	41.2	67	45	98
Thyroid gland	1	2.5	40	1	223
Bone	6	6.2	96	35	209
Connective	5	4.8	103	33	242
Other unspecified sites (Minor)	79	68.7	114	91	143
Of lymphatic and hematopoietic tissue	129	116.4	110	92	132
Lymphosarcoma and reticulosarcoma	48	34.0	140	104	187
Hodgkin's disease	9	16.9	53	24	101
Leukemia and aleukemia	54	49.1	109	82	143
Other	18	16.3	110	65	174
Brain neoplasms	10	12.8	77	37	143
Benign	3	3.8	78	16	231
Unspecified	7	9.0	77	31	160
Diabetes mellitus	45	85.4	52	38	70
Diseases of the blood and blood-forming organs	7	16.3	42	17	88
Mental, psychoneurotic and personality disorders	29	36.4	79	53	114

(Continued)

TABLE VII. Summary of Observed and Expected Deaths in Kanawha Valley Chemical Workers (comparison rates or proportions in use: US deaths) (continued)

Cause of death	Observed deaths	Expected deaths	SMR	95% CI Lower	95% CI Upper
Diseases of the nervous system	303	382.7	79	70	89
Diseases of the circulatory system	2,548	2,655.7	95	92	100
Rheumatic fever	2	2.9	68	8	247
Chronic rheumatic heart disease	33	71.3	46	32	65
Arteriosclerotic heart disease	2,089	2,155.9	96	93	101
Chronic endocarditis not specified as rheumatic	10	20.9	47	23	88
Other myocardial degeneration	32	47.6	67	46	95
Other diseases of the heart	136	115.8	117	98	139
Hypertension with heart disease	77	74.5	103	81	129
Hypertension without heart disease	24	19.2	124	80	185
Diseases of the arteries and veins	145	147.4	98	83	116
Diseases of the respiratory system	259	321.5	80	71	91
Acute upper respiratory	1	1.1	87	2	489
Influenza	6	9.8	61	22	133
Pneumonia (except newborn)	91	130.2	69	56	86
Bronchitis	11	19.9	55	27	99
Other respiratory diseases	150	160.5	93	79	110
Diseases of the digestive system	161	244.1	65	56	77
Diseases of the stomach and duodenum	29	50.7	57	38	82
Hernia and intestinal obstruction	9	22.8	39	18	75
Cirrhosis of the liver	123	170.6	72	60	86
Diseases of the genito-urinary system	40	72.4	55	39	75
Acute nephritis	2	4.5	44	5	159
Nephritis with edema, including nephrosis	2	2.6	77	9	278
Unspecified and chronic nephritis and other renal sclerosis	22	38.2	57	36	87
Infection of kidney	7	14.0	49	20	103
Calculi of urinary system	1	3.7	26	1	149
Hyperplasia of prostate	4	7.4	53	15	137
Other diseases of male genital organs	2	1.9	104	13	378
Diseases of the skin	6	5.1	117	43	255
Infections of the skin	2	1.5	136	16	494
Other diseases of the skin	4	3.6	109	30	280
Diseases of the bone and organs of movement	5	5.5	90	29	210
Unknown causes	71	69.9	101	79	128
Accidents	469	535.2	87	82	96
Violence	201	219.3	91	79	105
Suicide	149	168.4	88	75	104
Homicide	52	50.9	102	76	134
Other causes	243	250.7	97	85	110
Blank ICD (no death certificate)	264				
All deaths	5,785	6,148.5	94	92	96

chloride polymerization, the risk associated with vinyl chloride monomer operations was not evaluated.

The second cause of death category found to be in excess was lymphosarcoma and reticulosarcoma. There was no apparent pattern or trend by either latency (time since initial exposure) or duration of employment. Because multiple comparisons were made without benefit of an a priori hypothesis, the occurrence of excess deaths in this category could conceivably be a matter of chance. We do not consider the absence of trends by latency or duration of employment to be evidence against an occupational association, since such trends could have been obscured by the larger cohort. We are investigating the excess further with a case control study that will allow examination of a number of variables, including individual chemical exposures and interaction of chemicals, as well as a number of temporal variables, such as age, latency, and duration of employment.

CONCLUSIONS

Overall, the mortality experience of this large population of chemical workers is similar to that of the US white male population. While this is encouraging, potentially hazardous exposures were not homogeneous, and more refined analyses are planned to utilize the information already collected in support of this overall study. In addition to a more detailed examination of the two causes of death found to be in excess, we will explore the mortality experience of subcohorts with specific exposures. Initial studies will include ethylene oxide, coal hydrogenation, and 1,3-butadiene. Given the large number of chemicals produced at these plants, there may be other opportunities to define study subcohorts from within this population to test hypotheses derived elsewhere.

REFERENCES

Baxter PJ, Fox AJ (1975): Angiosarcoma of the liver as a certified cause of death 1963–1973. Lancet 1:27–28.

Halperin W, Frazier TM (1985): Surveillance for the effects of workplace exposure. Am Rev Public Health 6:419–32.

Marsh GM (1979): A preliminary exploratory study of proportional mortality among employees and ex-employees of the Union Carbide Corporation's plant at South Charleston, West Virginia. Submitted to the Union Carbide Corporation on August 22, 1979.

Millar JD (1984): Occupational health—defining the problem. Presented at American Occupational Health Conference, Los Angeles, CA, May 2, 1984.

Thomas TL, Decouflé P, Moure-Eraso R (1980): Mortality among workers employed in the petroleum refining and petrochemical plants. J Occup Med 22:97–103.

Waxweiler RJ, Beaumont JJ, Henry JA, Brown DP, Robinson CF, Ness GO, Wagoner JK, Lemen RA (1983): A modified life-table analysis system for cohort studies. J Occup Med 25:115–124.