

*Mortality Among Workers
at the Mound Facility:
A Preliminary Report*

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MORTALITY AMONG WORKERS AT THE MOUND FACILITY: A PRELIMINARY REPORT

by

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Abstract

Mortality among 4697 white males who were employed at the Mound Facility between 1943 and 1979 was compared with expected mortality based on US white male death rates. Standardized mortality ratios (SMRs) of 96 were observed for both all causes and all cancers. SMRs for digestive cancers and unintentional injuries were significantly less than 100. No SMR was significantly greater than 100 for these workers. A significantly elevated lung cancer SMR was observed for the subcohort of workers employed from 1943-1959, a period during which polonium-210 was processed at the plant. To determine the potential impact of wartime selection factors, this time period was further divided into two periods, 1943-1945 and 1946-1959. In the 1943-1945 period, the SMR for lung cancer was 204 (90% CI=140, 290), while in the later period the lung cancer SMR was 105 (90% CI=77, 140). Similar results were observed for all causes, all cancers, cancers of the rectum, nonmalignant respiratory diseases, and all injuries for which the SMRs were elevated during the wartime period but were not elevated after the war. Additional analyses considering workers hired in the period 1960-1979, during which plutonium-238 was processed at the facility, yielded little information. Generally, a strong healthy worker effect was observed and was attributed to the limited follow-up time and small numbers of deaths among this subcohort.

KEY WORDS: Mortality, Radiation, Nuclear Workers, Cohort, Cancer Mortality

INTRODUCTION

This report presents results for a study of mortality among employees of the Mound Facility located near Dayton, Ohio. This facility has been operated by the Monsanto Chemical Corporation for the Department of Energy (DOE) since 1943. From 1943 to 1948, when the current Mound Facility was started, the activity was called the Dayton Project and was carried out at several locations in Dayton. The work force at Mound is unique because it is one of the largest known cohorts of persons who may have been occupationally exposed to the alpha emitters Po-210 or Pu-238. Both of these radionuclides have been associated with adverse health effects in animal studies (Moroz and Parfenov, 1972; ICRP 1986).

Early Mound employees (1943-1959) were responsible for the separation, chemistry and metallurgy of Po-210, which was used in some of the first atomic weapons. This work began originally as part of the Manhattan Project and was conducted at secret locations in the Dayton area. In 1948, these operations were consolidated into the Central Mound Facility which is still in operation today. In the 1960s, work with polonium began to be phased down and eventually ended in the early 1970s. Processing of Pu-238 for heat sources in satellites, artificial hearts, and neutron sources began as the polonium work was decreasing. A variety of minor research projects since 1960 have involved other isotopes, including radium and thorium. However, during the periods 1943-1959 and 1960-1980, the Po-210 and Pu-238 operations were respectively the major processes at the Mound Facility. Accordingly, two subcohorts (1943-1959 and 1960-1979) defined by working within these time periods are the focus of this paper.

Polonium-210 (Po-210) is a naturally occurring alpha emitter of the uranium decay series. Po-210 is ubiquitous in our environment and contributes a substantial fraction of the total dose from naturally occurring radionuclides to humans (BEIR, 1980). Inhaled Po-210 deposits in the lung via inhalation, translocates to the blood stream, and then deposits in soft tissues, particularly the lung, liver,

kidney, spleen, and gonads (Holtzman, 1967; Holtzman, 1966). However, due to its short half-time in humans (60 days), the accumulation in tissue is limited. No studies of health effects among human populations exposed to this radionuclide have been completed. However, results of animal experiments suggest that inhaled Po-210 from cigarette smoke may be a cause of lung cancer in humans (Little, et al., 1985; Little and O'Toole, 1974; Shami, et al., 1982; Lisco, et al., 1974; Radford and Hunt, 1962.).

No studies of health effects among persons exposed to Pu-238 have yet been reported. The isotope plutonium-239 (Pu-239) has been found in tracheobronchial lymph nodes, lung, liver, and bone in measurable concentrations in occupationally exposed workers (McInroy, 1976). Although studies of these radionuclides in animals have demonstrated experimentally induced cancers at the above-mentioned organ sites, little is known regarding health effects in humans. A long-term clinical follow-up study of 26 men exposed to Pu-239 at Los Alamos has observed one bone cancer and three cases of lung cancer. All three of the lung cancer cases occurred in heavy smokers (Voelz, 1990). An epidemiologic investigation of mortality among plutonium-exposed workers at Rocky Flats observed elevated rate ratios for lymphopoietic/hematopoietic neoplasms (Wilkinson, et al., 1987).

MATERIALS AND METHODS

Name, social security number, education, and dates of birth, hire and termination were obtained from the personnel records at Mound for all persons ever employed between the beginning of the Dayton Project in 1943 through December 31, 1979. When available, race and sex were added to the file from health physics, medical records, and personnel identification photographs. A listing of persons with unknown race or sex was independently reviewed by three Mound employees. When there was agreement between all reviewers, race and/or sex was assigned in our study file. Persons of still undetermined race (n=589) or sex (n=17) were classified as white males. Females (n=1702), nonwhite males (n=345), military personnel (n=12), and visiting corporate personnel (n=7) were not considered in this analysis. Persons employed less than 30 days who therefore did not receive the required security clearance to work onsite at the plant were also excluded. Fifteen persons with unknown birthdates and six with unknown dates for both hire and termination were deleted from the cohort. Individuals with missing dates of hire (n=11) or termination from employment (n=2) were assumed to have worked at the plant one day prior to termination and one day after date of hire, respectively.

To verify the completeness of our study cohort, we compared our study file with the first quarter 941A Employee Quarterly Earnings records from the Social Security Administration for the years 1943-1979 (Marsh and Enterline, 1979). We identified 18 additional white male employees who were added to the cohort. This resulted in a final study cohort of 4697 white males. A 10% random sample of all records was edited. None of the study variables demonstrated an error rate greater than 1.1%.

Vital status through 1979 was determined using the following sources: the Social Security Administration (SSA), the Ohio Department of Motor Vehicles, the Mound retirement and benefits office, Mound medical records, telephone tracing, and coworker interviews. The SSA follow-up was the most

comprehensive, but only included earnings reports through the first quarter of 1980. Therefore, the end-of-study date was defined as December 31, 1979. The combination of these activities yielded vital status for approximately 98% (n=4588) of our study cohort. Death certificates were obtained for 803 of the 826 known deaths (97%) from state vital statistics offices or from the company. These were coded by a nosologist according to the 8th Revision of the International Classification of Diseases (ICDA). Persons known to be deceased but for whom no death certificate could be obtained (n=23) were assumed deceased, cause of death unknown. For 22 of these deaths, we knew the year of death and assigned July 1 as the month and day of death. Date of termination was assigned as the death date for the remaining death.

Utilizing the computer program developed by Monson (1974), we calculated standardized mortality ratios (SMRs) comparing observed mortality with mortality expected based on US white male mortality rates adjusted for age and calendar years in five-year intervals. Ninety percent exact confidence intervals for the SMR were calculated using an adaptation of a procedure reported by Rothman and Boice (1979). Person-years-at-risk were counted from the first date of hire until the earlier of death date or end-of-study date. Workers lost-to-follow-up contributed person-years only until their termination date. We carried out additional analyses by employment duration (degree of exposure) and time-since-first-employment (latency). For duration of employment, three mutually exclusive categories (less than two, two to five, and greater than five years) were examined. For time-since-first-employment, we considered three latency models. In the three models, persons entered the study at hire date, hire date plus 10 years, and hire date plus 20 years. These models did not represent mutually exclusive groups.

RESULTS

Among the entire cohort of 4697 white males described in Table I, the average age at hire was 31.4 years. The distribution of the cohort by year of hire revealed that 53% were hired during the period 1943-1959 and 47% were hired after 1959. These two hiring periods were selected to reflect two major operational periods at the facility. For the purpose of this report, we considered workers employed between 1943-1959 potentially exposed to Po-210 and those workers employed as of 1960 or later potentially exposed to Pu-238. Because of the gradual phaseout of polonium operations, some workers in the later time period could have had polonium exposure. All workers were potentially exposed to external radiation.

Length of follow-up averaged 19.7 years for the entire cohort, 25.8 years for workers hired before 1960 and 12.7 years for workers hired in 1960 or later. Among the entire cohort, 18% (826 persons) were deceased; but this figure was weighted by the large majority of deaths (765 persons) occurring among people hired before 1960 and the comparatively fewer deaths (61 persons) among workers hired after 1960.

SMRs for white males are presented in Table II. SMRs did not differ significantly from 100 for all causes of death or all cancers. SMRs above 100 were observed for several cancer sites including thyroid, 290 (90% CI = 15, 1376); oral and pharyngeal, 129 (90% CI = 61, 242); lung, 123 (90% CI = 98, 152); and prostate, 117 (90% CI = 66, 194). SMRs above 100 were also observed for benign and unspecified neoplasms, genitourinary diseases, diabetes mellitus, and suicide. Significantly low SMRs were observed for digestive system cancers, 71 (90% CI = 52, 95), and unintentional injuries, 75 (90% CI = 59, 94). SMRs below 100 were also observed for cancers of the stomach, large intestine, liver, pancreas, larynx, bladder, and brain/central nervous system and for lymphosarcoma, Hodgkin's disease, and leukemia.

Table III presents SMRs for workers who were hired between 1943-1959. These workers were employed during the years that Po-210 was used extensively. Especially interesting was the SMR of 131 (90% CI = 104, 163) for lung cancer. None of the remaining SMRs in this table differed significantly from expected, although the SMR for thyroid cancer was 334 (90% CI = 17, 1584) based on only one death.

Among the subcohort of workers hired since 1960, only 61 deaths were observed (Table IV). Although SMRs above 100 were present for cancers of the skin and kidney and for lymphosarcoma, Hodgkin's disease, suicide, and genitourinary diseases, the number of observed deaths in each category was small and the confidence intervals were wide indicating that our point estimates had limited precision.

SMRs are presented in Table V for employees who worked less than 2, 2 to 5, and greater than 5 years, during the years 1943-1959. Significantly elevated SMRs were observed for all causes of death, lung cancer, and injuries among those who worked less than 2 years. However, these excesses were not repeated among those who worked more than 2 years. The SMR for prostate cancer was significantly high for workers employed greater than 5 years.

Table VI provides an analysis of cause-specific worker mortality by time since initial employment for males hired between 1943 and 1959. For each of the three latency models evaluated, the SMRs for all causes showed slight increases. In contrast, no deviation from expected was observed for all cancers combined. Lung cancer, on the other hand, was consistently about 30% higher than expected and was generally statistically significant. The SMR for genitourinary diseases was almost 200 for persons followed for at least 10 years after initial employment. The SMRs for diabetes mellitus, nonmalignant respiratory diseases, and nonmalignant digestive diseases were somewhat elevated for each of the three latency models.

For workers hired between 1960 and 1979, analyses by duration of employment and time since initial employment are presented in Tables VII and VIII, respectively. These analyses were severely limited by the small numbers of observations in each subcategory. No SMRs were significantly elevated in either analysis. The SMRs for all causes were significantly low for two of the duration of employment categories, less than 2 years and greater than 5 years. The SMR for all causes among workers employed between 2 and 5 years was close to expected. Similarly, SMRs for all causes and circulatory diseases were significantly less than 100 for each of the two latency models considered in this analysis.

DISCUSSION

In this study, we have found an excess of lung cancer among workers who were first employed at Mound between 1943 and 1959, years during which operations involved the handling and processing of polonium-210. One possible explanation was revealed when duration of employment was examined. Persons who worked less than two years demonstrated elevated SMRs for lung cancer, injuries, and for all causes of death, while those employed longer did not. Several investigations of nuclear workers have employed a two-year work restriction (Wilkinson, et al., 1987; Gilbert and Marks, 1979). The rationale for such a restriction is that employees who work for short periods of time differ in many respects from more permanent employees (Gilbert and Marks, 1979). It is thought that transient employees are more likely to reflect poorer health status and behavior, and at the same time have less opportunity for experiencing significant exposures (Gilbert and Marks, 1979). However, until the exposure data for Mound employees are analyzed, it is impossible to determine if there is an association between duration of employment and exposure.

Another possible explanation for the lung cancer excess concerns the type of workers or the working conditions they encountered while employed during World War II. It is possible that those who were employed at that time differed from the rest of the work force because of not meeting requirements for military service. It is also possible that workers during the war years may have received large exposures. We investigated the possibility that employees hired during the war years have differed from those hired after the war years by calculating SMRs for those employed between 1943 and 1945, and those employed between 1946 and 1959. Both periods included the years that polonium-210 was routinely used. Table IX shows that the SMRs for all causes of death and all cancers for those hired between 1943 and 1945 were 118 (90% CI = 106, 131) and 130 (90% CI = 101, 164), respectively. Furthermore, the SMR for lung cancer was 204 (90% CI = 140, 290). On the other hand, the SMRs among those hired from 1946 to

1959 for all causes of death, all cancers and lung cancers were 98 (90% CI = 91, 105), 89 (90% CI = 74, 105) and 105 (90% CI = 77, 140), respectively. These findings suggest that workers hired between 1943 and 1945 may have been less healthy than workers hired later, had different life style characteristics such as smoking habits, or received greater exposures than those hired later. Unfortunately, without specific exposure information, health history data or information on smoking habits, we can only speculate.

Any consideration of health effects involving neoplastic disease and a hypothesized putative exposure must consider latency time (Rothman, 1981). For the subcohort hired between 1943 and 1959, we observed elevated SMRs for lung cancer for each of the three latency models considered. This finding is inconsistent with most research on lung cancer and other solid tumors where a minimal latent period of 10 years is generally accepted (Shore, 1984). In fact, the point estimated for the latency model of at least 20 years was somewhat lower than those for the other two models. In general, no latency relationship for lung cancer was observed.

With respect to lung cancer, the major confounder with which we should be concerned is smoking. The use of tobacco exerts such a strong effect on risk estimates of lung cancer that considerations which do not account for cigarette smoking are of limited utility. This problem was compounded by the lack of specific exposure estimates which were unavailable for these analyses. As has been previously mentioned, Mound employees in certain occupations had the potential to be exposed to external radiation, polonium-210 (for those hired from 1943 until the early 1970s) and plutonium-238 (for those hired from 1960-1979). Subsequent analyses need to compare exposed with unexposed employees.

Interpretation of our analyses was limited due to the small number of person-years accumulated by the population being followed. The observed number of deaths was small among the subcohort of workers hired since 1960, especially in the cause-specific categories. A possible explanation is that this

subcohort was still young, which results in a limited period of follow-up and a small percentage of deceased workers. Additional years of follow-up are necessary to study mortality among this subgroup.

Another limitation in this study was the use of national death rates to calculate the SMRs. SMRs based on US rates can incorrectly estimate mortality among a working population employed in a limited geographic area due to geographic differences in regional rates. However, this problem was offset somewhat by the greater stability that characterizes US rates compared with local rates.

Among industrial worker cohorts, a selection bias known as the Healthy Worker Effect (HWE) has been well recognized. The factors contributing to this have been summarized in detail (McMichael, 1976) and probably result from the selection of healthy persons into the work force. Among nuclear worker cohorts, the screening procedures necessary for security purposes and the intensive monitoring of worker health may also contribute to the HWE (Wilkinson, et al., 1987; Hadjimichael, et al., 1983). The HWE is usually strongest among young industrial cohorts with a brief period of follow-up and a small percentage of deceased workers.

When compared with US rates, a deficit in mortality among nuclear worker cohorts has been observed and ranges from 9% to 34% (Lushbaugh, 1982). The overall impact of the HWE among the 1943-1979 Mound work force appeared to be minimal (all causes SMR = 96) due to the long follow-up period (20-year average) and the percentage of deceased workers (18%). However, the HWE did not apply equally to the subcohorts within our study population, a result we believe due to the large differences in years of follow-up and percentage of deceased workers. The 1943-1959 subcohort averaged 25.8 years of follow-up, with over 30% dead and an all causes SMR = 103. In contrast, the subcohort of workers hired since 1960 averaged only 12.7 years of follow-up, less than 3% dead and an all causes SMR = 51. These

results support the observation that as the percent of deaths in a cohort increases, the SMR reflects a lessening of the HWE (Acquavella, et al., 1985).

Future analyses of the Mound cohort will take into account estimates of exposure to external radiation, polonium-210 and plutonium-238. These data will, in turn, allow us to estimate the cause-specific risk among exposed or burdened workers compared with unexposed or unburdened workers.

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Table I. Selected Characteristics Of White Male Mound Employees By First Hire Date

	1943-1979	1943-1959	1960-1979
Number	4697	2489	2208
Alive	3762	1633	2129
Lost-to-follow-up	109	91	18
Deaths	826	765	61
Death certificates missing	23	22	1
Average age at hire	31.4	32.9	29.7
Average age at death	61.1	62.0	49.2
Average duration of employment	6.4	5.8	7.2
Average years of follow-up	19.7	25.8	12.7
Average year of hire	1957.5	1949.1	1967.0
Average year at death	1967.7	1967.2	1974.8

Table II. Standardized Mortality Ratios Among White Male Employees Hired By Mound Between 1943-1979

Cause (ICDA-8)	Observed	SMR	90% Exact CI
All causes (1-999)	826	96	91 - 102
All cancers (140-209)	157	96	84 - 110
Oral and pharynx (140-149)	7	129	61 - 242
Digestive system (150-159)	33	71	52 - 95
Stomach (151)	6	64	28 - 126
Large intestine (153)	13	91	54 - 145
Rectum (154)	6	111	48 - 219
Liver (155)	1	31	02 - 147
Pancreas (157)	4	45	15 - 103
Larynx (161)	2	80	14 - 252
Lung (162,163)	62	123	98 - 152
Bone (170)	1	107	05 - 508
Skin (172,173)	4	122	42 - 279
Prostate (185)	11	117	66 - 194
Bladder (188)	3	64	17 - 165
Kidney (189)	4	96	33 - 220
Brain/central nervous system (191,192)	2	36	06 - 113
Thyroid (193)	1	290	15 - 1376
All lymphopoietic and hematopoietic (200-209)	14	82	50 - 128
Lymphosarcoma (200)	2	53	09 - 167
Hodgkin's (201)	2	78	14 - 246
Leukemia (204-207)	6	88	38 - 174
Benign neoplasms (210-239)	3	125	34 - 323
Diabetes mellitus (250)	16	133	83 - 202
Circulatory diseases (390-458)	406	94	86 - 102
Arteriosclerotic heart disease (410-413)	285	94	85 - 104
Cerebrovascular diseases (430-438)	55	98	77 - 123
Respiratory diseases (460-519)	48	104	81 - 132
Digestive diseases (520-577)	45	101	78 - 129
Liver cirrhosis (571)	23	97	66 - 137
Genitourinary diseases (580-629)	17	137	87 - 205
All injuries (800-998)	88	85	71 - 101
Unintentional injuries (800-949)	53	75	59 - 94
Suicide (950-959)	27	116	82 - 160

Table III. Standardized Mortality Ratios Among White Male Employees Hired By Mound Between 1943-1959

Cause (ICDA-8)	Observed	SMR	90% Exact CI
All causes (1-999)	765	103	97 - 109
All cancers (140-209)	142	100	87 - 115
Oral and pharynx (140-149)	7	148	69 - 278
Digestive system (150-159)	31	75	54 - 101
Stomach (151)	6	70	30 - 138
Large intestine (153)	12	94	54 - 152
Rectum (154)	6	123	54 - 243
Liver (155)	1	33	02 - 157
Pancreas (157)	4	51	17 - 117
Larynx (161)	2	91	16 - 286
Lung (162,163)	57	131	104 - 163
Bone (170)	1	127	07 - 602
Skin (172,173)	2	77	14 - 242
Prostate (185)	11	124	70 - 205
Bladder (188)	3	70	19 - 181
Kidney (189)	3	84	23 - 217
Brain/central nervous system (191,192)	2	45	08 - 142
Thyroid (193)	1	334	17 - 1584
All lymphopoietic and hematopoietic (200-209)	11	78	44 - 129
Lymphosarcoma (200)	1	31	02 - 147
Hodgkin's (201)	1	51	03 - 242
Leukemia (204-207)	5	87	34 - 183
Benign neoplasms (210-239)	3	147	40 - 380
Diabetes mellitus (250)	15	143	88 - 220
Circulatory diseases (390-458)	385	100	92 - 109
Arteriosclerotic heart disease (410-413)	269	100	90 - 111
Cerebrovascular diseases (430-438)	55	107	84 - 134
Respiratory diseases (460-519)	47	114	88 - 145
Digestive diseases (520-577)	43	115	88 - 148
Liver cirrhosis (571)	21	110	74 - 158
Genitourinary diseases (580-629)	15	133	82 - 205
All injuries (800-998)	72	100	81 - 122
Unintentional injuries (800-998)	46	92	71 - 118
Suicide (950-959)	20	119	79 - 173

Table IV. Standardized Mortality Ratios Among White Male Employees Hired By Mound Between 1960-1979

Cause (ICDA-8)	Observed	SMR	90% Exact CI
All causes (1-999)	61	51	41 - 63
All cancers (140-209)	15	70	43 - 108
Digestive system (150-159)	2	41	07 - 129
Esophagus (150)	1	217	11 - 1031
Large intestine (153)	1	61	03 - 289
Lung (162,163)	5	70	28 - 147
Skin (172,173)	2	283	50 - 891
Kidney (189)	1	171	09 - 811
All lymphopoitetic and hematopoietic (200-209)	3	103	28 - 266
Lymphosarcoma (200)	1	162	08 - 769
Hodgkin's (201)	1	165	08 - 783
Leukemia (204-207)	1	91	05 - 432
Diabetes mellitus (250)	1	64	03 - 304
Circulatory diseases (390-458)	21	47	31 - 68
Arteriosclerotic heart disease (410-413)	16	48	30 - 73
Respiratory diseases (460-519)	1	21	01 - 100
Digestive diseases (520-577)	2	28	05 - 88
Liver cirrhosis (571)	2	42	07 - 132
Genitourinary diseases (580-629)	2	185	33 - 582
All injuries (800-998)	16	52	33 - 79
Unintentional injuries (800-949)	7	35	16 - 66
Suicide (950-959)	7	107	50 - 201

Table V. Standardized Mortality Ratios By Duration Of Employment For White Males Hired Between 1943-1959

Cause (ICDA-8)	Duration Of Employment								
	Less than 2 years			2-5 years			Greater than 5 years		
	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI
All causes (1-999)	496	115	106 - 124	123	89	76 - 103	146	95	82 - 109
All cancers (140-209)	84	102	85 - 122	24	92	63 - 129	34	110	81 - 146
Oral and pharynx (140-149)	5	183	72 - 385	2	231	41 - 727	0	--	--
Digestive system (150-159)	17	70	44 - 104	7	94	44 - 177	7	81	38 - 152
Stomach (151)	4	77	26 - 177	0	--	--	2	121	22 - 381
Large intestine (153)	5	67	26 - 141	4	170	58 - 389	3	110	30 - 284
Rectum (154)	4	137	47 - 315	1	114	06 - 541	1	100	05 - 474
Liver (155)	1	56	03 - 264	0	--	--	0	--	--
Pancreas (157)	3	66	18 - 170	0	--	--	1	58	03 - 275
Lung (162,163)	39	157	118 - 206	8	99	49 - 179	10	99	54 - 168
Bone (170)	1	214	11 -1009	0	--	--	0	--	--
Skin (172,173)	0	--	--	0	--	--	2	363	64 -1143
Prostate (185)	4	75	25 - 170	2	120	21 - 378	5	280	110 - 589
Kidney (189)	2	97	17 - 306	1	150	08 - 712	0	0	--
All lymphopoietic and hematopoietic (200-209)	5	62	24 - 130	1	37	02 - 176	5	164	65 - 345
Leukemia (204- 207)	4	120	41 - 275	0	--	--	1	83	04 - 394
Diabetes mellitus (250)	9	145	76 - 254	3	154	41 - 398	3	138	38 - 357
Circulatory diseases (390-458)	244	107	96 - 119	64	89	72 - 110	77	95	78 - 115
Respiratory diseases (460-519)	32	132	96 - 178	7	91	43 - 171	8	90	45 - 162
Digestive diseases (520-577)	26	121	85 - 168	10	142	77 - 241	7	88	41 - 165
Genitourinary diseases (580-629)	10	146	79 - 248	2	102	18 - 321	3	153	42 - 395
All injuries (800-998)	54	131	103 - 164	8	58	29 - 105	10	75	41 - 127

Table VI. Standardized Mortality Ratios By Time Since First Employment For White Males Hired Between 1943-1959

	Time Since First Employment								
	0 or more years			10 or more years			20 or more years		
Cause (ICDA-8)	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI
All causes (1-999)	765	103	97 - 109	652	109	102 - 116	396	107	98 - 116
All cancers (140-209)	142	100	87 - 115	123	103	88 - 120	79	102	84 - 123
Oral and pharynx (140-149)	7	148	69 - 278	6	152	66 - 300	2	80	14 - 252
Digestive system (150-159)	31	75	54 - 101	26	77	54 - 107	18	87	56 - 129
Stomach (151)	6	70	30 - 138	6	95	41 - 187	5	142	56 - 299
Large intestine (153)	12	94	54 - 152	11	102	57 - 169	8	113	56 - 204
Rectum (154)	6	123	54 - 243	4	103	35 - 236	2	88	16 - 277
Liver (155)	1	33	2 - 157	1	44	02 - 209	0	--	--
Pancreas (157)	4	51	17 - 117	3	45	12 - 116	2	46	08 - 145
Lung (162,163)	57	131	104 - 164	53	136	107 - 171	34	128	94 - 170
Bone (170)	1	127	7 - 602	63	180	9 - 854	0	--	--
Skin (172,173)	2	77	14 - 242	2	95	17 - 299	1	79	04 - 375
Prostate (185)	11	124	69 - 205	8	101	50 - 182	5	91	36 - 191
Kidney (189)	3	84	73 - 217	1	33	02 - 157	0	--	--
All lymphopoietic and hematopoietic (200-209)	11	78	44 - 129	10	87	47 - 148	6	86	37 - 170
Leukemia (204-207)	5	87	34 - 183	4	87	30 - 199	2	72	13 - 227
Diabetes mellitus (250)	15	143	88 - 220	14	163	99 - 255	8	150	75 - 271
Circulatory diseases (390-458)	385	100	92 - 109	334	103	94 - 113	203	101	90 - 113
Respiratory diseases (460-519)	47	114	88 - 145	45	126	97 - 162	30	125	90 - 170
Digestive diseases (520-577)	43	115	88 - 148	40	134	101 - 174	24	136	94 - 191
Genitourinary diseases (580-629)	15	133	82 - 205	14	185	112 - 289	8	190	95 - 343
All injuries (800-998)	72	100	81 - 122	48	102	79 - 130	26	112	78 - 155

Table VII. Observed And Expected Deaths And Standardized Mortality Ratios By Duration Of Employment For White Males Hired Between 1960-1979

	Duration Of Employment								
	Less than 2 years			2-5 years			Greater than 5 years		
Cause (ICDA-8)	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI
All causes (1-999)	12	52	30 - 84	18	105	68 - 156	31	53	38 - 72
All cancers (140-209)	3	81	22 - 209	5	166	65 - 349	7	60	28 - 113
Lung (162, 163)	2	178	32 - 560	1	103	5 - 489	2	48	9 - 151
Lymphopoietic and hematopoietic (200-209)	0	--	--	1	230	12 - 1091	2	149	26 - 469
Circulatory disease (390-458)	1	14	1 - 66	4	66	23 - 151	16	65	41 - 99
All injuries (800-998)	5	63	25 - 132	7	141	66 - 265	4	37	13 - 85

Table VIII. Observed And Expected Deaths And Standardized Mortality Ratios By Time Since First Employment For White Males Hired Between 1960-1979

	Time Since First Employment					
	0 or more years			10 or more years		
Cause (ICDA-8)	Observed	SMR	90% Exact CI	Observed	SMR	90% Exact CI
All causes (1-999)	61	51	41 - 63	33	65	48 - 87
All cancers (140-209)	15	70	43 - 108	8	76	38 - 137
Lung (162,163)	5	70	28 - 141	2	52	9 - 164
Lymphopoietic and hematopoietic (200-209)	3	103	28 - 266	2	172	31 - 541
Circulatory disease (390-458)	21	47	31 - 68	14	64	39 - 100
All injuries (800-998)	16	52	33 - 79	5	56	22 - 118

**Table IX. Standardized Mortality Ratios And 90% Exact Confidence Intervals
For White Males Who Were Hired Between 1943-1945 Or 1946-1959**

	Year First Hired			
	1943-1945		1946-1959	
Cause (ICDA-8)	SMR	90% Exact CI	SMR	90% Exact CI
All causes(1-999)	118	106 - 131	98	91 - 105
All cancers(140-209)	130	101 - 164	89	74 - 105
All digestive(150-159)	73	38 - 127	76	51 - 108
Rectum(154)	332	131 - 701	29	2 - 139
Lung(162,163)	204	140 - 290	105	77 - 140
All circulatory disease(390-458)	100	85 - 117	100	91 - 111
All respiratory disease(460-519)	171	115 - 247	89	62 - 123
All digestive disease(520-577)	92	48 - 160	123	91 - 164
All genitourinary disease(580-629)	107	36 - 243	145	82 - 241
All injuries(800-998)	147	101 - 206	86	66 - 109

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