Mortality Among Unionized Construction Plasterers and Cement Masons

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Background *Plasterers perform a variety of duties including interior and exterior plastering of drywall, cement, stucco, and stone imitation; the preparation, installation, and repair of all interior and exterior insulation systems; and the fireproofing of steel beams and columns. Some of the current potential toxic exposures among plasterers include plaster of Paris, silica, fiberglass, talc, and 1,1,1-trichloroethylene; asbestos had been used by the plasterers in the past. Cement masons, on the other hand, are involved in concrete construction of buildings, bridges, curbs and gutters, sidewalks, highways, streets and roads, floors and pavements and the finishing of same, when necessary, by sandblasting or any other method. Exposures include cement dust, silica, asphalt, and various solvents.*

Methods *Proportionate mortality ratios (PMRs) and proportionate cancer mortality ratios (PCMRs) were calculated for 99 causes of death among 12,873 members of the Operative Plasterers' and Cement Masons' International Association who died between 1972 and 1996 using United States age-, race-, and calender-specific death rates. Statistical significance (P value) of results was based upon the Poisson distribution.*

Results *Among plasterers, statistically significant elevated mortality was observed for asbestosis, where the PMR reached 1,657 ($P < 0.01$) with eleven observed deaths and less than one death expected, for lung cancer ($PCMR = 124$, $P < 0.01$), and for benign neoplasms ($PMR = 210$, $P < 0.05$). Among cement masons, statistically significant elevated mortality was observed for cancer of the stomach ($PCMR = 133$, $P < 0.01$), benign neoplasms ($PMR = 132$, $P < 0.01$), and poisonings ($PMR = 159$, $P < 0.05$). Except for poisonings, which were not thought to be occupationally related, all of the statistically significant results occurred among those members who entered the union prior to 1950. However, the risk for lung cancer among plasterers was still elevated among those entering the union after 1970 as was the risk for stomach cancer among cement masons who entered the union after 1950.*

Conclusion *The present study suggests that plasterers and cement masons still have elevated risks for certain diseases, especially lung and stomach cancer. Therefore, union members currently living should be screened for asbestos-related diseases and educated about the future risks for these diseases.* Am. J. Ind. Med. 39:373–388, 2001.
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KEY WORDS: plasterer; cement mason; construction; proportionate mortality; lung cancer; stomach cancer; asbestos; cement dust; silica

INTRODUCTION

The National Plasterers Union was founded just before the Civil War with a handful of members; by 1883 the

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membership totaled 1,647 within 12 local unions. In 1914, in recognition of the growing number of cement workers who had come into the union, the name was officially changed to Operative Plasterers' and Cement Finishers' (OPCM) International Association. By 1930, the union had a membership of nearly 40,000. Reflecting the fact that members did more than finish cement, the union changed its name in 1951 to the current one, Operative Plasterers' and Cement Masons' International Association (of the United States and Canada). For this study we will use the name OPCM. It is one of the 15 Building and Construction Trades Departments which, in total, represent 3.5 million members affiliated with the AFL-CIO. Currently, OPCM has 120 local unions with approximately 36,000 members throughout the United States and Canada. Approximately 60% of the union membership are cement masons, 35% are plasterers, and 5% are shophands.

Within the construction industry, plasterers and cement masons work in a variety of projects from highway construction to assisting in the construction and renovation of industrial, commercial, and residential buildings. Plasterers, in general, are responsible for all interior and exterior plastering of drywall, cement, stucco, and stone imitation as well as the taping and pointing of all joints, nail holes and bruises on wallboard or drywall. Plasterers are also responsible for the preparation, installation, and repair of all interior and exterior insulation systems, and the fireproofing of steel beams and columns. Approximately 10% of the plasterers' duties involve insulation work (personal correspondence, Bill Shell, Executive Vice President, OPCM). Cement masons, on the other hand, are involved in concrete construction of buildings, bridges, curbs and gutters, sidewalks, highways, streets and roads, floors and pavements and the finishing of same, when necessary, by sandblasting or any other method. Shophands work in large warehouses and pre-manufacture items to be shipped to the job site such as ornate plaster columns and cornices and large pre-cast concrete items such as walls. A description of the job classifications of plasterers is presented in Appendix A. For a more detailed description of the job classifications and duties of the members of the OPCM, contact the authors.

This proportionate mortality ratio (PMR) study of OPCM was conducted in response to a request from the Center to Protect Workers' Rights, a research arm of the Building and Construction Trades Departments, AFL-CIO. This study is part of a larger initiative to examine the health and safety risks of construction workers in general, undertaken in 1991 by the National Institute for Occupational Safety and Health (NIOSH). The purpose of this PMR study is to identify those causes of death in excess among members of OPCM as a starting point for possible prevention and intervention activities.

METHODS AND MATERIALS

Study Population

Our study population consisted of all deceased OPCM members who (1) had been active dues-paying members; (2) were actively paying into the death benefit fund at the time of their death whether they were currently employed (a requirement when employed), unemployed or retired; and (3) had died in the United States between 1 January 1972 and 31 December 1996, excluding 1981–1983.

The OPCM computerized master file of demographic, work history, and death claim data which we obtained from the union consisted of records on all actively employed and deceased members from 1984 to 1996. Over 8,000 deaths which occurred between 1984 and 1996 were included in this file. The union had purged its computerized death claim file for claims filed prior to 1984. Information regarding demographics, work history, and death claim data for members who had died between 1972 and 1980 had been previously obtained by NIOSH in a Health Hazard Evaluation that was conducted by London and Rinsky [1986]. This accounted for approximately 4,800 additional deaths. Therefore, this study includes deaths occurring between 1 January 1972 and 31 December 1996 with the exclusion of those deaths which occurred during the years 1981–1983. Approximately 13,000 deaths were included in this study which provided adequate statistical power to examine even relatively rare causes of death as well as examining recent mortality trends.

For each union member we obtained name, Social Security number, membership number, date of birth, date entering and leaving the union, the various locals in which each member had worked, the time period for which dues were paid, date of death, death claim number, and type of trade held (plasterer, cement mason, or shophand). For deaths which had occurred between 1972 and 1980, the underlying and contributory causes of death had previously been coded by a qualified nosologist according to the revision of the International Classification of Diseases (ICD) in effect at the time of death. For the deaths which occurred between 1984 and 1996, the ICD codes for underlying and contributory causes of death were obtained from the National Center for Health Statistics, National Death Index, NDI Plus file. We actually had in our possession 9,449 hard-copy death certificates for this cohort.

Potential Exposures

Plasterers and cement masons have potential for exposure to numerous chemical, physical, and biological agents due to the work they perform. The National Occupational Exposure Survey (NOES) conducted during 1981–1983 on a representative sample of 4,500 U.S. industrial facilities with eight or more employees (employing nearly 1.8 million workers) recorded potential worker exposures [NIOSH 1988, 1990a,b; Sieber et al., 1991]. The result of the survey for plasterers and cement masons is presented in Appendix B. Some of the potential toxic exposures among plasterers include plaster of Paris, cement dust, silica, fiberglass, talc, and 1,1,1-trichloroethylene. Asbestos was also known to have been used by the plasterers in the past (up until about the late 1960s) in the spackling compound. Cement masons have been potentially exposed at the job site to various agents including cement dust, silica, talc, asphalt, and various solvents [Fowler, 1978; Gunter, 1979; Sanderson and Davidson, 1982; Linch et al., 1998]. Based on the aforementioned exposures, causes of death hypothesized to be elevated in this cohort included lung cancer and various other malignant and nonmalignant respiratory diseases, and cancers of the stomach, colon and esophagus. Because plasterers and cement masons often work side by side with persons from other construction trades, whose tasks and work practices vary widely, they may also have been exposed to other hazardous materials.

Statistical Analyses

Due to the limitations of the records available from the union, the only records available were for dues-paying members at the time of their deaths. Proportionate mortality ratios (PMR) were chosen as the method of analysis using the NIOSH Life Table Analysis System (LTAS). Three types of PMR analysis were used: one that evaluated the overall proportionate mortality; a second that evaluated the proportionate cancer mortality [Steenland et al., 1990], (both of these analyses using underlying cause of death); and a third that evaluated the proportionate mortality using all causes of death (multiple causes) as listed on the death certificate [Steenland et al., 1992]. A total of 99 causes of death utilizing the LTAS was examined.

The number of deaths in the study population, by cause, was compared with the number of deaths expected. Expected rates were based on the race and cause-specific mortality experience of the U.S. male population for the 5-year calendar-time periods beginning in 1970–1974 and ending with 1990–1994, the last category being duplicated for time period 1995–1996, and by 5-year age groups from 15 to 19 through 80–84, and then for 85 and older. The PMRs were then calculated by dividing the observed number of deaths by the number expected and multiplying the results by 100. Statistical significance of each cause-specific result was determined using the Poisson distribution. If the observed number of deaths was greater than six, the Byar approximation to the exact test was used; if the observed number of deaths was less than or equal to six, the exact test was used [Rothman and Boice, 1979]. PMRs were also calculated for trade (plasterer, cement mason, shophand), race, and by 10-year intervals based on calendar years of employment in the union. This last analysis was an attempt to account for changes in the formulas of materials or changes in work practices over time. Although we would have liked to have conducted analyses by duration of time worked in the union as well as duration of time worked in each local, the union did not maintain records to distinguish between periods of active employment versus active union membership (active employment and retired status). Therefore, analyses by duration of total employment and employment in union locals were not performed.

Proportionate cancer mortality ratios (PCMRs) were calculated using cancer-specific observed deaths among our study population and comparing these results with the cancer-specific proportionate mortality of the U.S. population with similar time periods and age groups as previously used for the PMR analysis. PCMR analyses were conducted to correct for possible biases in the cancer mortality PMR due to elevations or deficits from other nonmalignant causes, particularly a possible deficit of deaths from heart disease due to the "healthy worker effect."

A multiple-cause analysis using all causes of death as coded on the death certificate was conducted to examine any disease excesses not identified using only underlying causes of death. This analysis includes the usual underlying cause of death as well as contributory causes and other significant conditions at time of death which the physician or other medical provider noted on the death certificate and compares these results with those expected using multiple cause-of-death referent rates. Good candidates for multiple cause analyses are diseases which are of long duration, not necessary fatal, yet serious enough to be noted on the death certificate.

RESULTS

Characteristics of the Study Population

A total of 13,371 male deaths among members of OPCM was initially identified from the union's membership files we had received. We eliminated 332 members (2.4%) who had died after 1996. We also eliminated 147 members for whom we could not obtain a death certificate (1%) and 19 members for whom dates of birth could not be ascertained, leaving a total of 12,873 members.

Of the 12,873 members eligible for analysis, 11,442 were white (89%) and 1,431 (11%) were nonwhite males. This percentage is similar to that in the construction industry as a whole which is approximately 91% white and 9% nonwhite [CPWR, 1997]. The mean age on entering the union among all members in the study was 34.9 years (standard deviation {SD} = 10.4) and the average age at death was 72.3 years (SD = 13.2). The age distribution of the deaths ranged from 20 to 94 years. Cement masons comprised 61% (7,811) of the cohort, plasterers 34% (4,386), and shophands 5% (676).

Mortality Analysis for Entire Cohort

The numbers of observed and expected selective nonmalignant deaths and their corresponding PMRs are presented in Table I. There was a statistically significant elevation in PMRs due to benign neoplasms, asbestosis, other respiratory diseases, and poisonings. Significant deficits were observed for alcoholism, diseases of the heart, cerebrovascular diseases, and diseases of the genitourinary system.

The number of observed deaths, PMRs, and proportionate cancer mortality ratios (PCMRs) for cancer-specific deaths are shown in Table II. There was a statistically significant elevation in the PMR for all cancers combined (PMR = 125, $P < 0.05$) and six of the cancer sites shown had statistically significant elevated PMRs: trachea, bronchus and lung; stomach; intestine; rectum; brain and nervous system; and lymphatic and hematopoietic systems. The PCMR analysis by definition sets the ''all cancers'' category PCMR at 100 which, consequently, affects the site-specific cancer PCMR results. When this was done only two cancer sites had significantly elevated PCMRs; cancers of the stomach (N = 161, PCMR = 118, $P < 0.01$) and cancers of the trachea, bronchus and lung (N = 1,386, PCMR = 111, $P < 0.01$). Mesothelioma, a relatively rare cancer death, was observed for four members and the risk was elevated, PMR = 188, but not statistically significant.

Mortality Analysis by Trade (Plasterer, Cement Mason, Shophand)

PMR analyses were conducted for members by trade: plasterer, cement mason, shophand (Table III). For plasterers, significant elevations were seen for asbestosis, where the PMR reached 1,657 ($P < 0.01$) with eleven observed deaths and less than one death expected, and for benign neoplasms. There were significant deficits for heart and cerebrovascular diseases. For cement masons, significant excesses were seen for benign neoplasms and for poisonings and significant deficits for alcoholism, heart disease, and genitourinary disease. Shophands showed a significant excess in risk from poisonings and a deficit of respiratory system deaths.

Significant excess PCMRs were observed among plasterers for cancers of the trachea, bronchus, and lung; among cement masons for stomach cancers; and among shophands for non-Hodgkin's lymphoma (Table IV). Plasterers had a significantly reduced PCMR for lymphatic and hematopoietic cancers.

Mortality Analysis by Time-Period of Entry into Union Within Trade

Figures 1–4 illustrate the PMRs for those nonmalignant causes of death that were significantly elevated in the overall analysis by the time period of entry in the union. Figure 1 shows that asbestosis was statistically significantly elevated

TABLE I. Plasterers and Cement Masons Union-Proportionate Mortality Ratios (PMRs): Selected Categories of Nonmalignant Causes of Death, 1972–1980, 1984–1996

Causes of death (ICDA-9)	Observed deaths	Expected deaths	PMR
All Causes (000–999)	12873	12873	100
Benign neoplasms (210–239)	60	34	174**
Alcoholism (303)	10	34	29**
Diseases of the heart (390–429)	4658	4968	94**
Cerebrovascular diseases (430–438)	789	903	87**
Diseases of respiratory system (460–519)	1157	1155	100
Emphysema (492)	159	148	107
Asbestosis (501)	15	2	693**
Silicosis (502)	4	2	179
Other Pneumoconioses (500,503,505)	7	13	52
Other respiratory (470–478,494–499,504,506–519)	550	502	109*
Diseases of digestive system (520–579)	449	464	97
Cirrhosis of the liver (571)	214	194	110
Diseases of the genitourinary system (580–629)	176	216	82**
Injuries (E800–869, E880–929)	405	434	93
Poisoning (E850–869)	35	24	145*

* $P < 0.05$; ** $P < 0.01$.

TABLE II. Plasterers and Cement Masons Union-Proportionate Mortality Ratios (PMRs) and Proportionate Cancer Mortality Ratios (PCMRs): Selected Categories of Cancer, 1972–1980, 1984–1996

Causes of death (ICDA-9)	Observed deaths	PMR	PCMR
All Cancers (140–208)	3745	125**	100
Cancers of the digestive organs (150–159)	919	121**	97
Cancer of esophagus (150)	87	112	80
Cancer of stomach (151)	161	149**	118**
Cancer of intestine except rectum (152–153)	329	115*	92
Cancer of rectum (154)	86	145**	115
Cancers of the respiratory system (160–165)	1446	139**	111**
Cancer of larynx (161)	44	122	96
Cancer of trachea, bronchus, lung (162)	1386	139**	111**
Mesothelioma (163)	4	188	NA[a]
Cancers of other & unspecified sites (170–199)	416	124**	100
Cancer of brain & nervous system (191–192)	79	134*	108
Cancer of bone (170)	12	177	82
Cancer of lymphatic & hematopoietic system (200–208)	309	119*	96
Non-Hodgkin's lymphoma (200,202)	30	133	103
Leukemia (204–208)	127	119	96

$^{*}P < 0.05$; $^{**}P < 0.01$
[a]Rates not available.

only for those who entered the union prior to 1950. The PMR reached as high as 1,115 (over 11 times expected) for those joining in 1940–1949. For those entering the union prior to 1950, the PMR for asbestosis was 10.5 times that expected (ob = 13, exp = 1.24, PMR = 1,050). Only two deaths due to asbestosis occurred among those entering the union between 1950 and 1969, with no deaths occurring among those who entered the union after 1969. A somewhat

TABLE III. Plasterers and Cement Masons Union-Proportionate Mortality Ratios (PMRs): Selected Categories of Non-malignant Causes of Death, 1972–1980, 1984–1996, by Job Type

	Plasterers			Cement masons			Shophands		
Cause of death (ICDA-9)	OBS deaths	EXP deaths	PMR	OBS deaths	EXP deaths	PMR	OBS deaths	EXP deaths	PMR
All Causes (000–999)	4386	4386	100	7811	7811	100	676	676	100
Benign neoplasms (210–239)	24	11	210*	36	21	132**	0	—	—
Alcoholism (303)	1	—	—	8	25	32**	1	—	—
Diseases of the heart (390–429)	1673	1772	94*	2741	2937	93**	244	259	94
Cerebrovascular diseases (430–438)	260	347	75**	469	503	93	60	53	113
Diseases of respiratory system (460–519)	448	422	106	676	686	99	33	47	70*
Emphysema (492)	64	55	117	90	85	106	5	9	57
Asbestosis (501)	11	1	1657**	4	1	275	0	—	—
Silicosis (502)	0	—	—	3	1	236	1	—	—
Other pneumoconioses (500,503,505)	2	5	38	5	7	67	0	—	—
Other respiratory (470–478,494–499,504,506–519)	194	173	112	343	314	109	13	15	87
Diseases of digestive system (520–579)	145	145	100	281	292	96	23	27	84
Cirrhosis of the liver (571)	54	51	106	151	130	116	9	13	68
Diseases of genitourinary system (580–629)	67	79	85	101	126	80*	8	10	81
Injuries (E800–869, E880–929)	99	118	84	261	272	96	45	43	105
Poisoning (E850–869)	1	—	—	26	16	159*	8	3	304*

$^{*}P < 0.05$; $^{**}P < 0.01$.

TABLE IV. Plasterers and Cement Masons Union-Proportionate Mortality Ratios (PMRs) and Proportionate Cancer Mortality Ratios (PCMRs); Selected Categories of Cancer by Job Type, 1972–1980, 1984–1996

	Plasterers			Cement Masons			Shophands		
Cause of death (ICDA-9)	**OBS deaths**	**PMR**	**PCMR**	**OBS deaths**	**PMR**	**PCMR**	**OBS deaths**	**PMR**	**PCMR**
All Cancers (140–208)	1240	130**	100	2338	122**	100	167	124**	100
Cancer of digestive organs (150–159)	294	119**	91	582	122**	99	43	118	95
Cancer of esophagus (150)	29	132	101	58	112	91	0	—	—
Cancer of stomach (151)	44	125	96	110	164**	133**	7	116	92
Cancer of intestine (152–153)	111	115	89	202	114	93	16	126	106
Cancer of rectum (154)	26	126	98	52	146**	118	8	240*	197
Cancer of the respiratory system (160–165)	507	162**	123**	887	130**	106	52	114	87
Cancer of larynx (161)	14	129	99	27	116*	94	3	165	123
Cancer of trachea, bronchus, lung (162)	486	162**	124**	852	130**	106	48	111	85
Cancer of other & unspecified sites(170–199)	137	136**	103	253	116	95	26	171*	143
Cancer of brain & nervous system(191–192)	28	174**	129	47	119	98	4	131	111
Cancer of bone (170)	2	92	70	9	222*	180	1	—	—
Cancer of lymphatic & hematopoietic systems (200–208)	74	90	69**	219	132**	109	16	133	116
Non-Hodgkin's lymphoma (200,202)	5	64	47	17	130	104	8	445**	366**
Leukemia (204–208)	29	82	63*	95	143**	119	3	58	53

* $P < 0.05$; ** $P < 0.01$.

similar result was observed for other respiratory diseases (Fig. 2) where statistically significant excess results were seen only for those who entered the union before 1950. Among those joining after 1950, the PMR for other respiratory diseases for each time period was below 100 with a definite trend downward. Benign neoplasms PMR (Fig. 3) was elevated for each time period, with a statistically significant PMR only among those who entered by 1940.

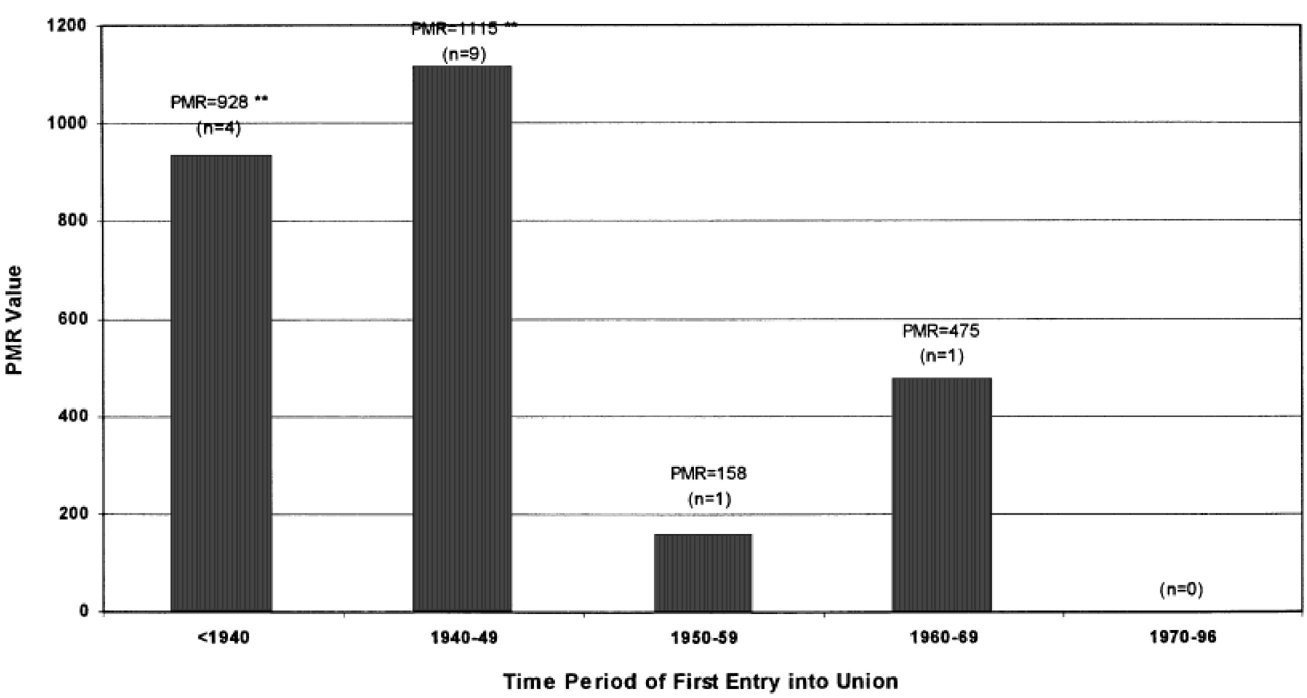

FIGURE 1. Asbestosis PMR values by time period of first entry into union.
** $p < 0.01$.

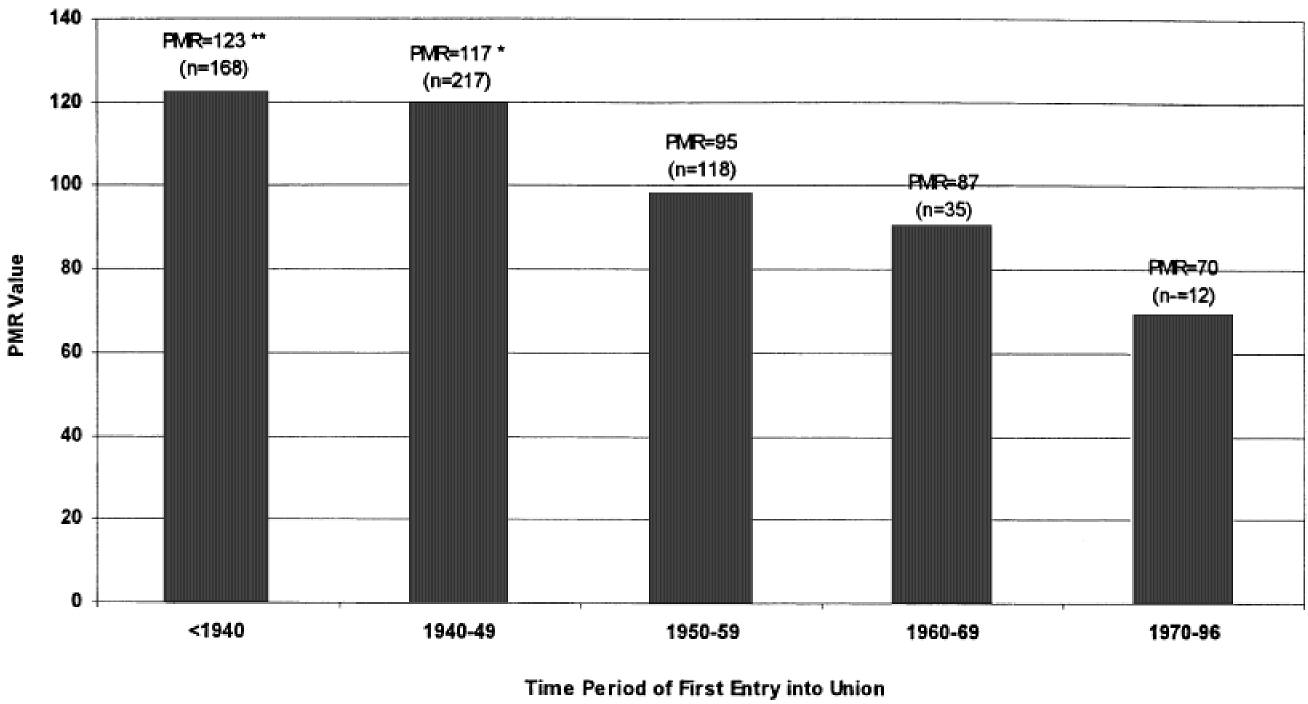

FIGURE 2. Other respiratory diseases PMR values by time period of first entry into union.
$^{*}p < 0.05$; $^{**}p < 0.01$.

PMR results for poisonings (Fig. 4) were elevated for those entering after 1960 with a statistically significant result for those who joined the union between 1970 and 1996.

In the overall PCMR analysis, only two causes of death were significantly elevated; cancers of the trachea, bronchus and lung, and cancers of the stomach. When we examined these results by trade, we observed that plasterers had an

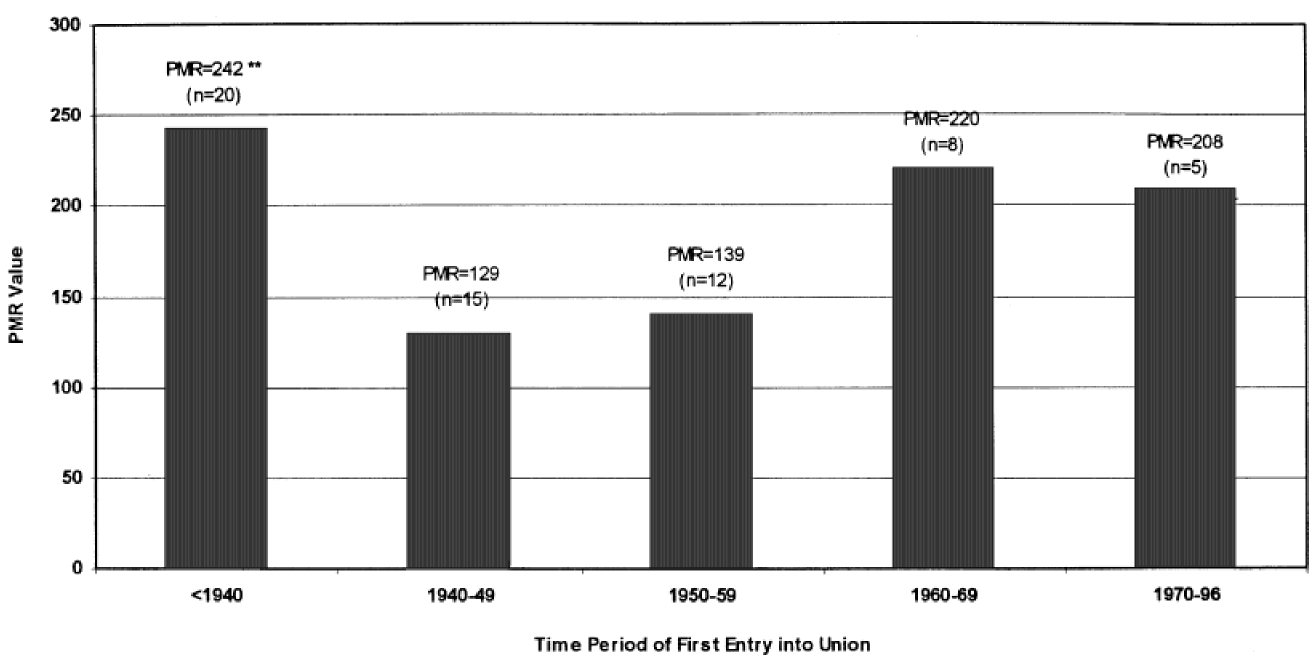

FIGURE 3. Benign neoplasm PMR values by time period of first entry into union.
$^{**}p < 0.01$.

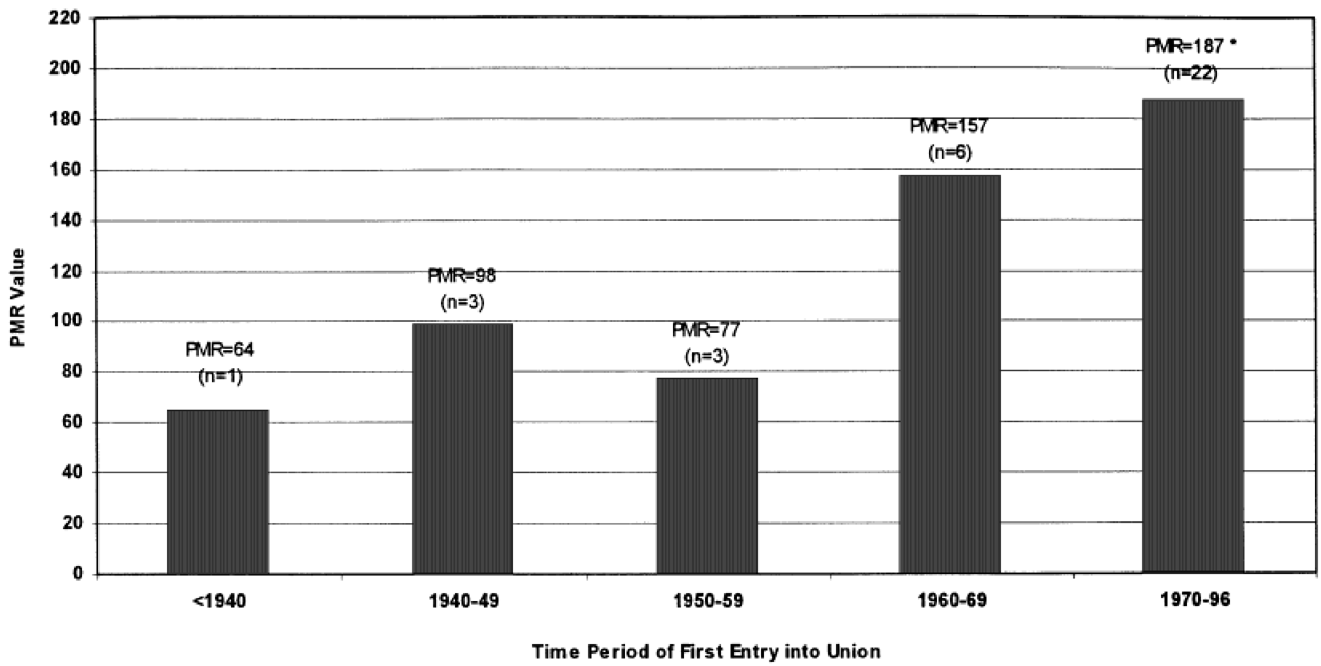

FIGURE 4. Poisoning PMR values by time period of first entry into union.
$^{*}p < 0.05$.

elevated risk for cancers of the trachea, bronchus and lung, whereas cement masons had an elevated risk for cancers of the stomach. Figure 5 presents the PCMRs for cancers of the trachea, bronchus and lung by trade and by time period of entry in the union. All time periods showed elevated PCMRs for lung cancer for both trade groups. However, only the earliest time period (members joining by 1940) for plasterers was statistically significant.

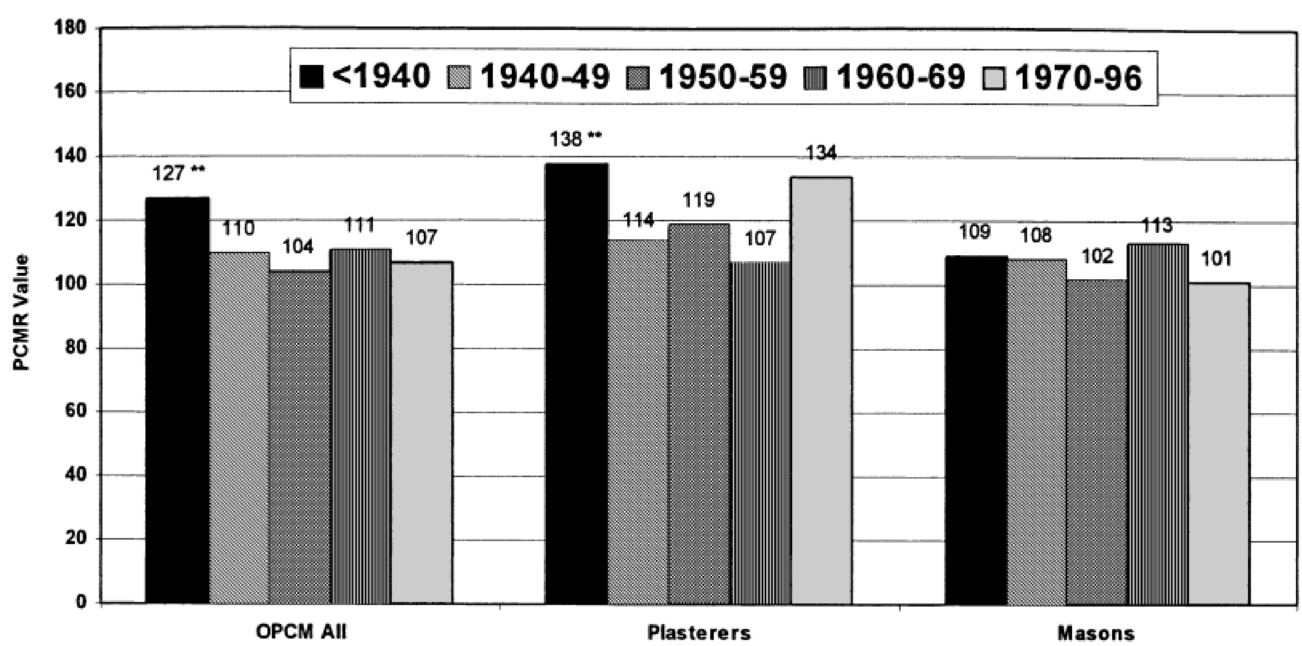

FIGURE 5. Lung/bronchus/trachea cancer PCMR values by time period of first entry into union and by trade.
$^{**}p < 0.01$.

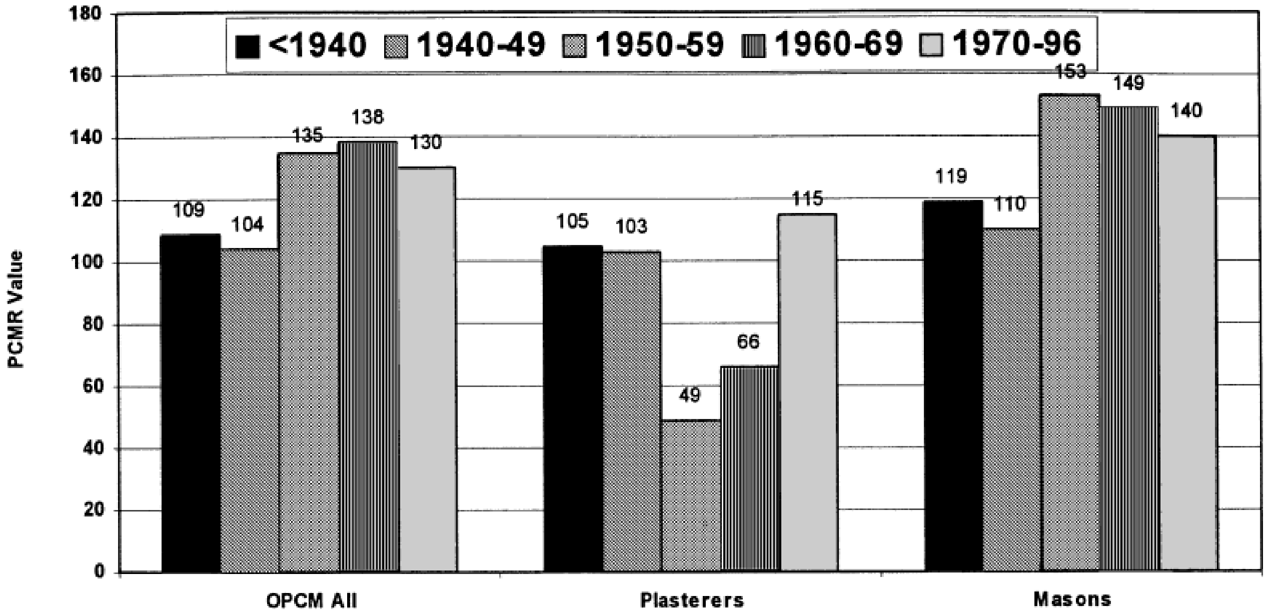

FIGURE 6. Stomach cancer PCMR values by time period of first entry into union and by trade.

Figure 6 presents the PCMRs for stomach cancer by trade and by time period entry in the union. For each time period, cement masons showed an excess stomach cancer PCMR of between 10% and 53%, but none of the results was statistically significant.

Mortality by Race

The PMRs by race are shown in Table V. Whites had significant elevations for benign neoplasms, asbestosis, other respiratory diseases, cirrhosis of the liver, and poisonings; for nonwhites, no noncancer cause of death was significantly elevated. However, the total number of deaths for nonwhites was considerably less than for whites making statistical significance harder to achieve.

Significantly elevated PCMRs were seen among white members only (Table VI); for cancers of the stomach (135 deaths, PCMR = 121, $P < 0.05$) and of the trachea, bronchus and lung (1,227 deaths, PCMR = 111, $P < 0.01$).

Mesothelioma

Due to the underascertainment of deaths related to mesothelioma (a discussion of this follows), we manually reviewed all hard copy death certificates in our possession (N = 9,449) to obtain a more accurate assessment of mesothelioma-related deaths in this cohort. We identified 40 mesothelioma deaths; even though only one-third of our cohort were plasterers, 50% of those who died of mesothelioma worked in this trade.

Multiple-Cause Mortality Analysis

A total of almost 31,000 causes of death was used in the multiple cause of death analysis. These PMR results were found to be quite similar to the underlying cause of death analysis for most of the causes of death. However, there were two noticeable exceptions. Nonmalignant diseases of the stomach had a PMR of 100 in the original analysis with 54 deaths, but was statistically significantly elevated in the multiple cause analysis with 194 deaths (PMR = 130, $P < 0.01$); and the PMR for accidental poisonings went from 35 deaths in the underlying cause of death analysis (PMR = 145, $P < 0.05$) to 123 deaths (PMR = 401, $P < 0.01$) in the multiple cause of death analysis.

DISCUSSION

Overall Mortality

The most notable mortality risks documented in this analysis among members from the Operative Plasterers' and Cement Masons' International Association were due to significant increases in deaths due to malignant (lung cancer) and nonmalignant diseases (asbestosis, other respiratory diseases) of the respiratory system, benign neoplasms, stomach cancers, poisonings and non-Hodgkin's lymphoma.

Plasterers

Malignancy of the lung among plasterers was found to have a statistically significant elevated PCMR, 24% higher

TABLE V. Plasterers and Cement Masons Union-Proportionate Mortality Ratios (PMRs): Selected Categories of Death,1972–1980, 1984–1996, by Race

Cause of Death (ICDA-9)	White Males			Nonwhite Males		
	OBS deaths	EXP deaths	PMR	OBS deaths	EXP deaths	PMR
All Causes (000–999)	11442	11442	100	1431	1431	100
All Cancers (140–208)	3295	2647	124**	450	356	126**
Benign neoplasms (210–239)	55	31	119**	5	4	78
Alcoholism (303)	9	27	33**	1	—	—
Diseases of the heart (390–429)	4195	4488	93**	463	680	96
Cerebrovascular diseases (430–438)	679	781	87**	110	121	91
Diseases of respiratory system (460–519)	1064	1049	101	93	106	88
Emphysema (492)	152	139	109	7	9	77
Asbestosis (501)	15	2	741**	0	—	—
Silicosis (502)	3	2	156	1	—	—
Other Pneumoconioses (500,503,505)	7	13	54	0	—	—
Other Respiratory (470–478,494–499,504,506–519)	529	477	111*	46	42	110
Diseases of digestive system (520–579)	410	410	100	39	54	73
Cirrhosis of the liver (571)	199	172	116*	15	23	66
Diseases of the genitourinary system (580–629)	141	178	79**	35	37	93
Injuries (E800–869,E880–929)	370	385	96	35	49	72*
Poisoning (E850–869)	35	21	164**	0	—	—

* $P < 0.05$; ** $P < 0.01$.

TABLE VI. Plasterers and Cement Masons Union–Proportionate Mortality Ratios (PMRs), and Proportionate Cancer Mortality Ratios (PCMRs): Selected Categories of Cancer, 1972–1980, 1984–1996, by Race

Cause of death (ICDA-9)	White males			Nonwhite males		
	Deaths	PMR	PCMR	Deaths	PMR	PCMR
All Cancers (140–208)	3295	124**	100	450	126**	100
Cancer of digestive organs (150–159)	803	121**	97	116	119	93
Cancer of esophagus (150)	74	120	96	13	82	63
Cancer of stomach (151)	135	151**	121*	26	135	106
Cancer of intestine (152–53)	297	115*	92	32	115	92
Cancer of rectum (154)	77	144**	114	9	156	121
Cancer of the respiratory system (160–165)	1280	138**	110**	166	142**	111
Cancer of larynx (161)	40	130	103	4	76	59
Cancer of trachea, bronchus, lung (162)	1227	139**	111**	159	143**	113
Cancer of other & unspecified sites(170–99)	372	123**	99	44	141*	110
Cancer of brain & nervous system(191–192)	74	132*	106	5	173	129
Cancer of bone (170)	12	199*	161	0	—	—
Cancer of lymph & hematopoietic (200–208)	282	119**	96	27	118	93
Non-Hodgkin's lymphoma (200,202)	28	132	103	2	142	101
Leukemia (204–208)	115	117	95	12	146	114

* $P < 0.05$; ** $P < 0.01$.

than that expected (Table IV). The lung cancer risk for plasterers was elevated for each time period of entry in the union (Fig. 6); however, only for the earliest time period (prior to 1940) was there a statistically significant elevated risk.

Asbestosis, a pneumoconiosis caused by inhalation of asbestos particles, was statistically significantly elevated for the entire cohort at almost seven times expected (Table I). Examining this result by trade revealed that plasterers were at a statistically significant elevated risk sixteen-and-a-half times expected (Table III). The asbestosis risk was highly significantly elevated only for those members who started prior to 1950 (Fig. 1), with only two deaths occurring in members who began in 1950 or later.

Mesothelioma is a rare malignant tumor arising in the linings of the pleura (lung) or the peritoneum (stomach) with 80% of malignant mesotheliomas occurring in men exposed to asbestos in the workplace [Mossman and Gee, 1989]. The tumor may be confused with that of metastatic cancer and therefore misdiagnosis can easily occur.

It is important to note that only four of the 40 deaths were coded by our nosologist as mesothelioma, cancer of the pleura, or ICD code 163.0 [WHO, 1977]. This is because the ICD code for mesothelioma does not include all sites where mesothelioma can occur and, therefore, most mesothelioma deaths are coded into other death categories. As a result, the remainder are not counted in the national U.S. death rates for pleural or peritoneal mesothelioma and result in a large undercount of mesothelioma deaths. The latency period for mesothelioma is 35–40 years between first exposure to asbestos and clinical diagnosis with most deaths occurring after a person reaches 60 years of age. The average age at death for the 40 mesothelioma deaths in our study was 71. Even though only a third of our population were plasterers, 50% of these deaths were in this occupational grouping.

Prior occupational exposure to asbestos has been associated with cancer of the lung (and possibly cancers at other sites), asbestosis [Selikoff et al., 1968; NIOSH, 1972, 1976; Becklake, 1976; McDonald and McDonald, 1987; Mossman and Gee, 1989] and mesothelioma of the pleura and peritoneum. Various job subclassifications of plasterers have had known asbestos exposure including spray insulating, plastering, taping, and asbestos removal (during demolition of existing buildings) [Verma and Middleton, 1980; Enterline, 1985; Engholm et al., 1987; Hughes et al., 1987; Kipen et al., 1987]. In the post-World War II period, asbestos was widely sprayed as an insulating and fireproofing material in commercial construction. During the early 1970s, three million pounds of fireproofing mixture, 30% of which was chrysotile asbestos, was sprayed by plasterers on steel foundations [Fischbein et al., 1979]. Additionally, because of its low expense and physical characteristics, asbestos was often added as a filler in plastering formulations. Taping, i.e., applying paper or fiberglass tape and finishing spackle material at wallboard joints, is a task of plasterers in the construction industry. While spackling compounds are mainly plaster of Paris, it has been shown that most of these compounds, up to the late 1960s, also contained asbestos minerals added as reinforcing agents, after which time the addition of asbestos was discontinued. Spackling materials containing dry asbestos was often delivered bagged directly to the job sites (personal communication with Bill Shell, executive V.P, OPCM). Rohl et al. [1975] noted generally high airborne asbestos levels in areas where commercial and residential spackling, patching, and taping compounds were used. He showed that either chrysolite or amphibole asbestos, or both, were in spackling compounds, which could contain between 5–12% by weight of asbestos minerals. Fischbein et al. [1979] found that 13 of the 15 industrial drywall taping and spackling compounds analyzed contained asbestos. Tapers spend up to 10% of their working time sanding dried spackle and could have experienced intermittent asbestos exposure through the 1960s. Air samples taken in the breathing zone of drywall tapers during the sanding of wallboard taping showed that fiber concentrations often exceeded by several times the threshold limit value (TLV). The asbestos fiber concentrations generated by sanding were similar to those measured in the work environment of asbestos insulation workers who had a seven-fold increased risk of cancer of the lung and of the pleura [Selikoff et al., 1979]. In a clinical report of drywall construction workers, radiological evidence of pulmonary asbestosis was observed in 40.9% of the workers examined; this compared to 48.5% among insulators [Fischbein et al., 1979]. Therefore, drywall tapers may have had asbestos exposure levels similar to those of insulators.

Wagner et al. [1960] found that the appearance of asbestosis or mesothelioma was a clear indicator of prior asbestos exposure. In one study in which 70 consecutive patients with pleural mesothelioma were examined, 69 had prior asbestos exposure, and there was some question about the seventieth [Selikoff et al., 1968]. In reviewing the hard copy death certificates in our possession ($N = 9{,}449$), we observed 40 cases of mesothelioma; 24 of which were not otherwise specified as to site, 7 pleural mesothelioma or pleural cancer (unspecified), and 9 of the lung (unspecified). Expected rates of mesothelioma deaths to which to compare our findings were not available but these deaths are considered to be quite rare in the general population. Nicholson [1982, 1986] has shown in reviewing numerous studies on asbestos exposure that there was a clear association between the number of mesothelioma deaths and the number of excess lung cancers that were attributable to asbestos exposure. The ratio of mesothelioma to excess lung cancer was found to be in the range of 1:3 to 1:10, depending on the magnitude of the exposure, the length of exposure, the time since onset of exposure (latency) and the fiber type (chrysotile, amosite or crocidolite). Based on Nicholson's findings and the 40 cases of mesothelioma in our study, we would predict an excess of between 120 and 400 lung cancer deaths due to asbestos in this population. Our data were quite consistent with Nicholson's findings. In the PCMR analysis we found the excess lung cancers to be 137 (1,386 observed—1,249 expected) and in the PMR analysis the excess lung cancers to be 390 (1386 observed—996 expected). Therefore, we would suggest that asbestos exposure may have contributed to the elevated lung cancer risk in this population.

A prior analysis of the mortality experience of the OPCM utilized 3,699 death certificates from 1972 to 1980 [London and Rinsky, 1986], which are part of the current study. Results showed a statistically significant PMR for lung cancer which was confirmed with a PCMR analysis. Examination of the mortality of plasterers separately found the PCMR for lung cancer to be statistically significant (PCMR = 134, $P < 0.01$). This was very similar to the result found in our analysis (PCMR = 124, $P < 0.01$).

Analyses of surveillance data have also shown increased risks for lung cancer among plasterers. In three PMR studies, a significantly increased risk for lung cancer was observed among plasterers. Two of these studies were conducted in the United States; one in California [Peterson and Milham, 1980] and the other in Washington State [Milham, 1983], and one was conducted in England and Wales [Registrar General, 1986]. Plasterers were also found to be at an increased risk for lung cancer in a large mortality study that analyzed data from over 876,000 death certificates from 19 states for the years 1984–1986. The results showed an elevated risk for lung cancer among those whose "usual occupation" as stated on the death certificate was construction plasterer (PMR = 127) or drywall installer (PMR = 121) [Robinson et al., 1995]. In an update of this study in which almost two million death certificates from 24 states for 1984–1988 were analyzed, a significant increased risk for lung cancer was observed among white male construction plasterers (PMR = 138) and among drywall installers (PMR = 145) [Burnett et al., 1997].

Smoking is among the greatest risk factors for a variety of diseases, especially lung cancer. In an analysis of smoking by occupation using the National Health Interview Surveys, Nelson et al., [1994] showed that the cigarette smoking prevalence among drywall installers (55%) was among the highest of all occupations included in the survey, 10 times greater than the prevalence among the lowest smoking occupational group, physicians (5.5%), and almost 50% greater than that of blue collar workers as a whole (39.2%). Unfortunately information on smoking history from OPCM members was not available to evaluate directly how much smoking might have contributed to the elevated lung cancer risk. However, if cigarette smoking was significantly elevated among our population compared to the referent group, we would have expected rates for heart and cerebrovascular diseases to be elevated, when, in fact, both were significantly lower than expected. On the other hand, the rate of death from emphysema, another smoking-related disease, was slightly elevated.

The rate of benign neoplasm deaths was also statistically significantly elevated in the entire union population (PMR = 174), among both the plasterers (PMR = 210) and cement masons (PMR = 132). A review of the 60 death certificates for benign neoplasms revealed four mesothelioma deaths, six lung tumors, nine brain tumors, and four tumors of the stomach or colon.

Cement Masons

The PMR and the PCMR for stomach cancer were statistically significantly elevated among cement masons, with a PCMR 33% higher than expected. PCMR results were also elevated for each time period of entry in the union.

In 1930, cancer of the stomach was the leading cause of death due to cancer among men in the United States. The risk of stomach cancer has declined remarkably since then [Devesa et al., 1987) but it still has one of the poorest 5-year survival rates of all site-specific cancers with only 19% survival over the period 1986–1991 [ACS, 1996]. Although dietary factors as well as lower socioeconomic status have been associated with increased risk of stomach cancer, occupational exposures to cement dust and asbestos have also been associated with stomach cancer. A NIOSH study [Robinson et al., 1995] of occupational-coded death certificates from 19 U.S. states found that those in cement-related occupations had a elevated risk for stomach cancer: brick and stone masons (PMR = 208, $P < 0.01$) and cement and terrazzo finishers (PMR = 179, $P > 0.05$). A study of workers in cement production also showed a statistically significant excess mortality rate for stomach cancer (SMR = 175) which was considered to be related to cement dust exposure [McDowall, 1984]. Another study, evaluating the hypothesis that exposure to cement dust is associated with an increased risk of stomach cancer, examined a sample of workers employed in 23 United States non-asbestos cement plants for at least 5 years. Stomach cancer was increased overall (SMR = 135), but was not statistically significant and was not significantly associated with tenure, latency, or year of birth [Amandus, 1986]. A matched case-referent study examining all gastrointestinal cancer deaths from 1922 to 1988 from two parishes surrounding a cement plant found an elevated risk for cement masons among the grouping of "esophageal or stomach cancer," odds ratio (OR) = 3.2; $P = 0.06$ [Jakobsson et al., 1993]; however, many of these deaths were among short-term workers.

Asbestos exposure has also been linked with stomach cancer. As early as 1979, it had been reported that workers in the construction industry exposed to asbestos (including cement masons) had a fourfold excess risk of stomach cancer [Selikoff et al., 1979].

It should also be noted that although nonmalignant diseases of the stomach had a PMR of 100 with 54 deaths in the overall analysis, in the multiple-cause analysis, examining contributory causes of death, the risk increased to a statistically significant PMR of 130 with 194 deaths.

The risk among cement masons for death by poisoning was statistically significantly elevated among both white and nonwhite members of the union. When the death certificates of the 35 deaths due to poisoning were reviewed, none of the deaths appeared to be associated with occupational exposure. All of the deaths were identified as either drug or alcohol intoxication or as deaths due to carbon monoxide poisoning which occurred away from the work site. The average age at death was 41.6 years. Factors extrinsic to the workplace appear to be the cause of the excess number of poisonings among cement masons which has increased dramatically since 1960.

Cement masons may also be exposed to silica dust which has been shown to cause silicosis. Linch et al. [1998] found that some of the highest respirable silica dust concentrations occurred in construction, especially masonry. The study also noted that workers in standard industrial classification 174—masonry, stonework, tile setting, and plastering—were exposed to at least 10 times the NIOSH recommended exposure limit. Although most of the time cement masons lay concrete free-handed or in pre-cast forms, there are times when cement masons may have to grind or rub the concrete by machine, possibly producing respirable silica dust. We observed three deaths due to silicosis among cement masons with a PMR of 236 and, while the number of observed deaths was small, the PMR was high. Silicosis deaths could also have been misdiagnosed or placed into the broader category of pneumoconiosis and other respiratory diseases which was slightly elevated among cement masons.

Shophands

There were 676 deaths among shophands in our cohort; statistically significant excess risks were observed for deaths due to poisonings ($\mathrm{PMR} = 304$, $P < 0.05$) and for non-Hodgkin's lymphoma ($\mathrm{PCMR} = 366$, $P < 0.01$). Again, deaths due to poisonings did not appear to be occupationally related. Scherr et al. [1992] in a case-control study of new cases of non-Hodgkin's lymphoma diagnosed from January 1980–May 1982 in Boston found an increased risks for plasterers (relative risk $= 6.0$) as well as for brick and stone masons (relative risk $= 12.0$). These two occupations had the highest risk, other than farmers, among all occupations reviewed. The risk appeared to be from exposures to dust and fibers.

Limitations and Strengths

The primary advantage of a PMR study based on union records, as this one is, is that the occupational information on union records may be more accurate than that listed on death certificates. Union records contain the occupation or skill classification reported by the member during his working life versus death certificates which only report the worker's usual or lifetime occupation reported by a survivor of the deceased worker and may reflect recall bias. In addition, Beaumont et al. [1981] reported that PMR studies based on a data set that resulted from a financial incentive for survivors to report deaths among inactive and retired workers provide the most reliable estimates of the death rates or standardized mortality ratios (SMRs).

A potential limitation in the PMR methodology is that the magnitude of the PMR for each cause of death is dependent on the magnitude of the PMRs for other causes of death. This can be especially important if a specific common cause has a relatively high or low mortality. If the PMR for the common cause of death is high, the PMRs for other causes will be low. Typically in a working population, the risk for heart disease is lower than expected due to the so called "healthy worker effect" and, therefore, in a PMR study the other causes would be disproportionally elevated. The deficit for heart and cerebrovascular disease (424 deaths) was not balanced by an excess in another noncancer cause of death in our study. Therefore, PCMR analyses were conducted to adjust for this potential bias.

We would normally have conducted additional analyses to identify excess risks based on total duration of employment and on local union membership. OPCM records do not show enough detail to distinguish between periods of active employment and active union membership. Many members of this cohort were still on the active union membership rolls many years after their last day of work. Therefore, we conducted the previously mentioned analyses by time period of entering the union within trade.

Another possible limitation of the study was the lack of deaths from 1981 to 1983. When we examined the 5-year calendar period of deaths from 1980 to 1984, we observed that both the observed and expected deaths were approximately two-fifths of what would have been expected. We realize there is a potential for biased results; however, we have no reason to believe that the deaths between 1981 and 1983 would be substantially different than those which occurred in 1980 and 1984. Therefore, we compared the deaths for those years to the expected deaths in the general population.

Other limitations include not having individual work histories, work processes and work materials which could delineate which sub-group of members was at greatest risk in the union and the reason; the loss from the study of short-term workers; and the inaccuracies in cause of death on death certificates.

SUMMARY

In summary, our study of almost 13,000 deceased members of the Operative Plasterers' and Cement Masons' International Association observed statistically significant elevated risk among plasterers due to lung cancer, asbestosis, other non-malignant respiratory diseases, and benign neoplasms; among cement masons due to stomach cancer, benign neoplasms and poisoning; and among shophands due to poisonings and non-Hodgkin's lymphoma. Asbestos, cement dust, silica, fibers and possible other exposures may have contributed to some of these excess risks. Except for poisonings, which do not appear to be occupationally related, all of the statistically significant excess risks were among members who entered the union prior to 1950. However, the risk for lung cancer among plasterers was still elevated among those entering the union after 1970 as was the risk for stomach cancer among cement masons who entered the union after 1950. Therefore, union members still living should be screened for asbestos-related diseases and educated about the future risks for lung cancer, stomach cancer, mesothelioma, etc. In addition, further research could be conducted by: (1) performing a case-control designed study which might better clarify which types of jobs are at greatest risk and (2) a continued surveillance of this group of workers through death certificate review. These recommendations were presented to the union. The extent of cigarette smoking in this cohort was not known and could have contributed to the elevated levels of respiratory cancers if smoking was higher in this group than in the referent population. However, heart and cerebrovascular disease mortality, which are strongly associated with excess cigarette smoking, were found to be significantly lower than expected.

APPENDIX A: Operative Plasterers' and Cement Masons' International Association Classifications for Plasterers and Cement Masons

Plasterer's Classification

Acoustical Tile Applicator
Artificial Marble Worker
Bond Acoustical Applicator
Certified File Applicator
Cork Specialist
Epoxy Applicator
Exterior/Interior Insulation Applicator
Fireproofer
Gyproc Filler
Kenetex Applicator
Machine Applicator Acoustical Worker
Nail On Wood
Perma Stone Worker
Plasterboard Applicator
Gun Plasterer Machine Operator
Plastic Coating Applicator
Sculptor
Speculative Housing
Spray Applicator
Stone Applicator
Swimming Pool
Taper, Joiner, Glaze Coater
Trainee
Wallboard Taper and Joiner/Drywall Applicator

Cement Mason's Classification

Concrete Paver
Concrete Production Worker
Concrete Rubber (Sacker)
Concrete Saw Operator
Coring and Storing Machine operator
Epoxy Floors
Floor Hardeners, Sealers and Curing Applicator
Form Setter
Grinder
Gunite Operator
Gypsum Roof, Zonolite, Etc.
Heavy and Highway
Laser Screed Operator
Magnesite Composition Seamless Floors (Asst)
Marblecrete
Nozzleman
Restoration Steeplejack
Sand Blaster and Bush Hammering
Sidewalk
Speculative Housing
Spray Applicator Urethane
Steeplejack
Swimming Pool
Trainee
Waterproofer

APPENDIX B: Selected Physical Exposure Agents and Ergonomic Hazards for Plasterers and Cement Masons Surveyed in 1982–1983 in Industrial Workplaces*

Plasterers

Physical Agents
Plaster of Paris
Cement Dust
1,1,1- Trichloroethylene
Sand
Mica

Quartz
Silica
Attapulgite
Talc
Fiberglass

Ergonomic Hazards
Lifting Postures
Arm Transport
Shoulder Transport

Cement Masons

Physical Agents
Cement Dust
1,1,1- Trichloroethylene
Quartz
Sand
Silica
Asphalt
Brick Clay
Talc
Carbon Tetrachloride
Dioxane
Tetrachloroethylene

Ergonomic Hazards
Lifting Postures
Hand/Wrist Manipulations
Shoulder Transport
Arm Transport
Whole Body Vibration

*From the National Occupational Exposure Survey. [Sieber, et al., 1991].

REFERENCES

Amandus H. 1986. Mortality from stomach cancer in United States cement plant and quarry workers, 1950–80. Br J Ind Med 43:526–528.

American Cancer Society (ACS) 1996. Cancer Facts and Figures—1996. American Cancer Society, Inc. 96-300M-No. 5008.96, Atlanta, GA.

Beaumont J, Leet TL, Okun AH.1981. Occupational data sets appropriate for proportionate mortality ratio analysis. Banbury Report 9: "Quantification of Occupational Cancer," p. 391–411.

Becklake M. 1976. Asbestos related diseases of the lung and other organs. Their epidemiology and implications for clinical practices. Am Rev Resp Dis 114:187–227.

Burnett C, Maurer J, Rosenberg H, Dosemeci M. 1997. Mortality by occupation, industry, and cause of death: 24 reporting states, 1984–1988. DHHS (NIOSH) Pub. No. 97–114.

The Center to Protect Workers' Rights (CPWR). 1997. The construction chart book. The U.S. construction industry and its workers. Washington, D.C.

Devesa SS, Silverman DT, Young JJ, Pollack ES, Brown CC, Horm JW, Percy CL, Myers MH, McKay FW, Fraumeni JF. 1987. Cancer incidence and mortality trends among whites in the United States, 1947–1984. J Natl Cancer Inst 79:701–770.

Engholm G, Englund A, Fletcher AC, Hallin N.1987. Respiratory cancer incidence in Swedish construction workers exposed to man-made mineral fibers and asbestos. Ann Occup Hyg 31: 663–675.

Enterline PE. 1985. Asbestos and cancer. Proceedings of a symposium on Epidemiology and Risk Assessment, Columbia, Maryland, 14–16 May 1985, Centers for Disease Control/ NIOSH, p 17.

Fischbein A, Rohl A, Selikoff I, Nicholson W. 1979. Drywall construction and asbestos exposure. Am Ind Hyg Assoc J 40:402–407.

Fowler D. 1978. Industrial hygiene survey of Frederick Meiswinkel, Inc. in San Francisco, California. SRI International, Project No. 5415, Contract No. 210-76-0120, NIOSH, Cincinnati, OH.

Gunter B. 1979. Health hazard evaluation determination, Report no. HHE-79–130–645, Craig Power Plant, Craig, Colorado. DHHS Cincinnati, OH.

Hughes JM, Weill H, Hammad YY. 1987. Mortality of workers exposed in two cement manufacturing plants. Br J Ind Med 44:161–174.

Jakobsson K, Horstmann V, Welinder H.1993. Mortality and cancer morbidity among cement workers. Br J Ind Med 50:264–272.

Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. 1987. Pulmonary fibrosis in asbestos insulation workers with lung cancer: A radiological and histopathological evaluation. Br J Ind Med, 44:96–100.

Linch K, Miller W, Althouse R, Groce D, Hale J. 1998. Surveillance of respirable crystalline silica dust using OSHA compliance data (1979–1995). Am J Ind Med 31:547–58.

London M, Rinsky R. 1986. Health hazard evaluation report no. HETA-81-025-1668. Operative Plasterers and Cement Masons International Assoc., DHHS, Cincinnati, OH.

McDonald J, McDonald A. 1987. Epidemiology of asbestos-related lung cancer. In: Antman K, Aisner J, editors. Asbestos-Related Malignancy. Orlando, FL: Grune & Straton; p 57–79.

McDowall M. 1984. A mortality study of cement masons. Br J Ind Med 41:179–182.

Milham S. 1983. Occupational mortality in Washington State 1950–1979. NIOSH Publications No. 83–116. DHHS, Cincinnati, OH.

Mossman B, Gee B. 1989. Asbestos-related diseases. N Engl J Med 320:1721–1730.

Nelson D, Emont S, Brackbill R, Cameron L, Peddicord J, Fiore M. 1994. Cigarette smoking prevalence by occupation in the United States. J Occup Med 36:516–525.

National Institute for Occupational Safety and Health (NIOSH). 1972. Criteria for a recommended standard . . . occupational exposure to asbestos. US DHEW, Cincinnati, OH.

National Institute for Occupational Safety and Health (NIOSH). 1976. Revised recommended asbestos standard. US. DHEW, Cincinnati, OH.

National Institute for Occupational Safety and Health (NIOSH). 1988, 1990a,b. National Occupational Exposure Survey. DHHS (NIOSH) Publication Nos. 88–106, 89–102, 89–103. Washington, DC.

Nicholson WJ, Perkel G, Selikoff IJ. 1982. Occupational exposure to asbestos: Population at risk and projected mortality—1980–2030. Am J Ind Med 3:259–311.

Nicholson WJ. 1986. Airborne asbestos health assessment update. Prepared for the United States Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-84/003F, Washington, D.C.

Peterson GR, Milham S. 1980. Occupational mortality in the State of California 1959–1961. DHEW (NIOSH) Publication No. 80–104, Cincinnati, OH.

Registrar General's decennial supplement for England and Wales, 1979–1980, 1982–1983 occupational mortality. 1986. Office of Population Censuses and Surveys. London: Her Majesty's Stationary Office.

Robinson C, Stern F, Halperin W, Venerable H, Petersen M, Frazier T, Burnett C, Lalich N, Salg J, Sestito J, Fingerhut M. 1995. Assessment of mortality in the construction industry of the United States, 1984–1986. Am J Ind Med 28:49–70.

Rohl A, Langer A, Selikoff I, Nicholson W. 1975. Exposure to asbestos in the use of spackling, patching, and taping compounds. Science 189:551–553.

Rothman K, Boice J.1979. Epidemiologic analysis with a programmable calculator. Washington, DC. Deptartment of Health and Human Services, NIH pub. 79-1649: p 30–31.

Sanderson W, Davidson C. 1982. Industrial hygiene survey of Northwestern States—Cement, Mason City, Iowa. Cement workers morbidity study. NIOSH, Morgantown, WV.

Scherr P, Hutchison G, Nieman R. 1992. Non-Hodgkin's lymphoma and occupational exposure. Cancer Res 52:5303–5309.

Selikoff I, Hammond EC, Seidman H. 1979. Mortality experience of insulation workers in the United States and Canada, 1943–1976. Ann NY Acad Sci 330:91–116.

Selikoff I, Hammond E, Churg J. 1968. Asbestos exposure, smoking and neoplasia. J Am Med Assoc 204:104–110.

Sieber K, Sundin D, Frazier T, Robinson C. 1991. Development, use, and availability of a job exposure matrix based on National Occupational Exposure Survey data. Am J Ind Med 20:163–174.

Steenland K, Beaumont J, Spaeth S, Brown D, Okun A, Jurcenko L, Ryan B, Phillips S, Roscoe R, Stayner L, Morris J. 1990. New developments in the life table analysis system of the National Institute for Occupational Safety and Health. J Occup Med 32:1091–1098.

Steenland K, Nowlin S, Ryan B, Adams S. 1992. Use of multiple-cause mortality data in epidemiologic analyses: US rate and proportion files developed by the National Institute for Occupational Safety and Health and the National Cancer Institute. Am J Epidemiol 136:855–862.

Verma D, Middleton C. 1980. Occupational exposure to asbestos in the drywall taping process. Am Ind Hyg Assoc 41:264–269.

Wagner JC, Sleggs CA, Marchand P. 1960. Diffuse pleural mesothelioma and asbestos exposure in North Western Cape Province. Br J Ind Med 17:260–271.

WHO. 1977. International classification of diseases, 9th revision. Geneva: World Health Organization.