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Mortality of a Cohort of U.S. Workers Employed in the Crushed Stone Industry, 1940–1980

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The mortality of 3,246 males who had been employed 1 or more years during 1940–1980 at 20 crushed stone operations was evaluated for possible association between employment and death from lung cancer, pneumoconiosis, and other respiratory diseases. Four deaths were attributed to pneumoconiosis. Based on available work histories, at least two of these deaths were probably due to dust exposures in the crushed stone industry. Mortality attributed to pneumoconiosis and other nonmalignant respiratory diseases, including chronic obstructive lung disease, was significantly increased overall (SMR: 1.98; 95%CI: 1.21–3.05), and especially so for a subcohort of crushed stone workers that processed granite (SMR: 7.26; 95%CI: 1.97–18.59). With regard to lung cancer, overall SMRs were elevated (although not statistically significant). Analyzed by rock type, there was a significantly elevated lung cancer SMR among granite workers with at least 20 years latency (SMR: 3.35; 95%CI: 1.34–6.90). Although not definitive, results of this study are consistent with the hypothesis that exposure to respirable silica dust is a risk factor for lung cancer.

Key words: crushed stone workers, mortality, lung cancer, mesothelioma, chronic obstructive lung disease, pneumoconiosis, silicosis, granite, limestone, traprock

INTRODUCTION

Crushed stone is size-reduced rock used primarily for building and construction purposes [U. S. Dept. of the Interior, 1980]. Proportional distribution of operations in the U.S. crushed stone industry by rock type are: limestone (74%), granite (11%), traprock (9%), sandstone (3%), marble (2%), and shell (1%) [U. S. Dept. of the Interior, 1980]. A crushed stone operation is generally composed of one or more quarries and a processing facility for crushing, sorting, and cleaning the stone. Although some crushed stone is produced from underground mines, most is obtained from open pit quarries [U. S. Dept. of the Interior, 1980]. Activities consist of overburden removal, drilling and blasting, secondary breakage by drop-ball or drop-hammer cranes, stone loading, and transport to a mill in which the stone is crushed,

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screened to desired size, and loaded into trucks or railroad cars for shipping. Airborne dust is generated during most or all of these procedures.

Although nonmalignant occupational respiratory diseases had been reported in crushed stone workers [Hammond, 1944; Rice, 1983; Baucom, 1986], cancer risk among crushed stone workers had never been studied until asbestos, a known carcinogen, was discovered in a crushed stone quarry in Maryland in the mid-1970s [Carter, 1977]. This discovery, in particular, stimulated the National Institute for Occupational Safety and Health (NIOSH) to evaluate the respiratory disease mortality experience of crushed stone workers.

The NIOSH evaluation involved a cohort mortality study to report on the respiratory disease mortality of workers employed at selected crushed stone operations. In addition, an industrial hygiene survey [Kullman et al., 1995] characterized exposures to selected respiratory hazards in all but two of the 20 crushed stone operations selected for the mortality study. A report of the mortality evaluation follows.

MATERIALS AND METHODS

Cohort Selection

At the initiation of this study, crushed stone operations were selected from a Mine Safety and Health Administration (MSHA) listing of the active industry in 1978. With the exception of requiring inclusion of the traprock operation in Maryland where asbestos fibers were initially discovered, a stratified sample of operations was randomly selected by rock type (granite, limestone, traprock, or sandstone) and by geographical location (east or west of the Mississippi river, but within the continental United States).

However, because of reluctance or refusal of some companies to cooperate and because of closures of some selected operations, the studied sample was substantially different from the original sample. Although replacements were randomly selected, some replacement operations were likewise replaced due to lack of cooperation from companies. The studied sample included only 10 of the 27 randomly selected operations in the original sample (6 of 15 limestone operations, 2 of 9 granite operations, 2 of 2 traprock operations, and no sandstone operations even though one sandstone operation had been selected in the original sample).

At the time the study was planned, the granite, limestone, and traprock segments of the crushed stone industry in the continental United States included 2,586 operations employing ~ 26,475 active workers, including office workers (Table I) [U. S. Dept. of Labor, 1978]. The studied sample was composed of a total of 20 operations—nine limestone, six granite, and five traprock—which employed a total of 1,022 workers, including office workers, at the time personnel records were microfilmed for this study (Tables I, II).

Summary of Industrial Hygiene Survey

Table II includes a partial summary of data from the NIOSH industrial hygiene survey, which is reported elsewhere in detail [Kullman et al., 1995]. Although other mineral fibers (mineral particles with aspect ratios of at least 3:1) were found at several operations, asbestos fibers were found in samples from only one operation (traprock). The operation where asbestos fibers were identified was the same Maryland-based operation that had initially stimulated interest in conducting the study. At

TABLE I. Number of Active Limestone, Granite, and Traprock Crushed Stone Operations and Estimated Number of Individuals Employed at These Operations and Number of Operations and Active Employees in the Studied Sample*

	Granite				Limestone				Traprock			
	Industry		Sample		Industry		Sample		Industry		Sample	
Region[a]	No. mines	No. miners	No. mines	No. miners	No. mines	No. miners	No. mines	No. miners	No. mines	No. miners	No. mines	No. miners
West												
Sub total	39	532	1	28	887	7,392	1	186	241	1,455	1	30
% Total	19	17	17	12	43	36	11	33	74	49	20	13
East												
Sub total	167	2,566	5	197	1,167	13,017	8	385	85	1,513	4	196
% Total	81	83	83	88	57	64	89	67	26	51	80	87
Total	206	3,098	6	225	2,054	20,409	9	571	326	2,968	5	226
% of Total[b]	8	12	30	22	79	77	45	56	13	11	25	22

*Industry data from 1978 MSHA records [U.S. Depart. of Labor, 1978]; sample data from company personnel records (as of 1979–1980).

[a]East and west of Mississippi River.

[b]Total number of granite, limestone, and traprock crushed stone operations: 2,586 in the industry and 20 in the sample. Total number of granite, limestone, and traprock crushed stone employees: 26,475 in the industry and 1,022 in the sample.

the time of the environmental survey, two of the 10 personal exposures to asbestos measured by NIOSH investigators at that operation were above the NIOSH recommended exposure limit of 0.1 fiber per cc, and two were below the detection limit of 0.02 fiber per cc.

Crystalline silica in the form of alpha-quartz was found to be a component of respirable dust at essentially all of the surveyed operations, comprising 37%, 11%, and 15% by weight of the personal respirable dust samples collected at granite, limestone, and traprock, respectively (Table II) [Kullman et al., 1995]. Geometric means of measured personal respirable exposures to crystalline silica were 0.06 mg/m^3 in the granite operations and 0.04 mg/m^3 in both the traprock operations and the limestone operations [Kullman et al., 1995]. The NIOSH recommended exposure limit for crystalline silica (0.05 mg/m^3) was exceeded in 45%, 20%, and 12% of personal respirable dust samples in the granite, limestone, and traprock operations, respectively [Kullman et al., 1995].

Work Histories, Vital Status Follow-up, and Cause of Death Coding

Between July 1979 and March 1981, work histories were microfilmed from company personnel records for 5,807 of the 5,960 male workers ever employed in the 20 selected operations (Fig. 1). Work histories were missing from company records on the other 153 male workers. For each member of the cohort, each crushed stone job was coded and entered into a computer file along with start date and end date. No information on health status or on smoking habits was obtained from the company records. Statistical analyses excluded: (1) all 2,505 workers with <1 year of tenure prior to December 31, 1980, and (2) 56 others who were terminated prior to January 1, 1940 or for whom date of birth was unavailable. This left 3,246 men in the final analyzed cohort.

TABLE II. Information by Operation

Operation[a]	State	Rock type[b]	Earliest year of hire	Cohort size: Total (n)	Cohort size: >20 yr latency (n)	Quartz content of dust (%)	Geo. mean exposure[c] (mg/m^3)	Fibers[d]
1	GA	L	1955	109	5	7.2	0.03	N
2	GA	L	1976	34	0	17	0.04	N
3	TN	L	1968	55	0	8.9	0.03	N
5	PA	L	1947	28	0	15	0.05	N
6	WV	L	1917	543	377	5.5	0.04	N
9	IL	L	1909	301	95	6.6	0.04	N
13	MI	L	1919	236	159	8.0	0.03	N
14/15	IA	L	1947	382	125	8.9/18.4	0.03/0.11	N
—	MD	L	1916	186	102	—	—	—
4	GA	G	1973	42	0	42	0.09	U
7	SC	G	1917	179	44	42	0.09	N
8	NC	G	1935	163	51	39	0.05	N
10	NC	G	1959	59	1	29	0.04	N
12	MN	G	1945	34	34	29	0.04	M
—	NC	G	1942	68	7	—	—	—
11	MD	T	1955	266	28	ND	ND	A
16	CT	T	1927	33	22	ND	ND	N
17	CT	T	1940	104	39	8.2	0.04	M
18	PA	T	1908	266	188	27	0.10	M
19	CA	T	1935	158	37	12	0.04	M

[a]Codes correspond to "original operation numbers" in Table I of Kullman et al. [1994].
[b]L = limestone; G = granite; T = traprock.
[c]Respirable dust [Kullman, unpub. obs.].
[d]Results of microscopic fiber analyses [Kullman et al., 1994]: N = no fibers detected; U = fiber sampling not done; M = mineral fibers, but no asbestos fibers, detected; A = asbestos fibers detected. ND = all samples below detection limit.
— = environmental survey not done.

Follow-up of vital status covered the period January 1, 1940, through December 31, 1980. Vital status as of December 31, 1980, was ascertained for all 3,246 men—2,585 survivors and 661 deceased (Fig. 1). Determination of vital status was accomplished using standardized follow-up procedures involving searches by the U.S. Postal Service, Internal Revenue Service, Social Security Administration, and for difficult-to-locate cases, a private search organization. Death certificates were obtained for all except 46 (7%) of the deceased.

Underlying causes of death were coded according to the International Classification of Diseases, 8th rev. [U.S. Dept. of Health, Education, and Welfare, 1967] (hereafter referred to as ICD-8), by a National Center for Health Statistics nosologist who was unaware of the exposure status of decedents. These coded underlying causes of death were used for the mortality analysis.

In addition, to identify sentinel health outcomes specifically related to occupational mineral dust exposure [Rutstein et al., 1983], all death certificates were individually reviewed by the project officer (JC) to identify cases with explicit mention of "mesothelioma," "silicosis," or "pneumoconiosis." The results of this review (which identified sentinel occupational deaths recorded as contributing cause of death) were not used for the SMR analysis.

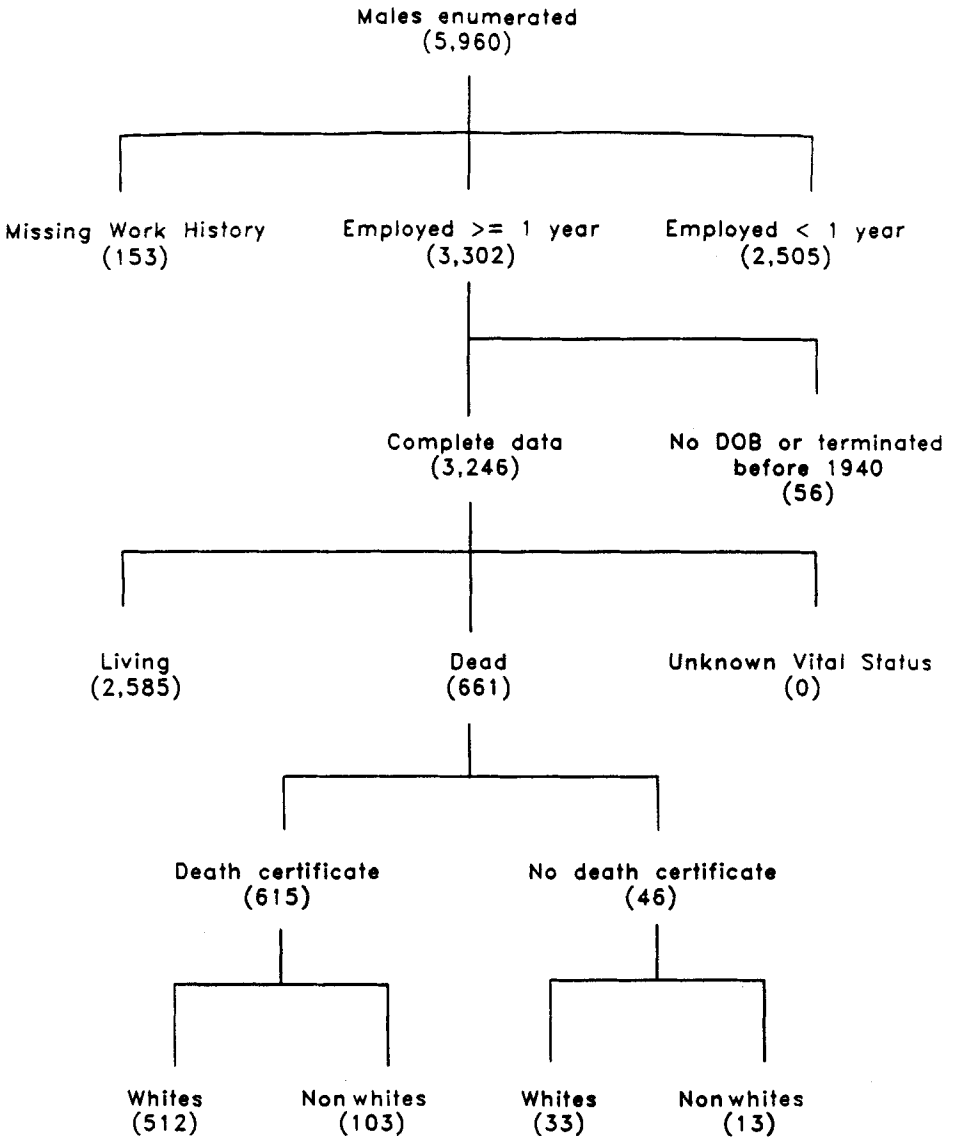

Fig. 1. Enumeration and vital status follow-up of crushed stone workers employed at 20 operations.

Mortality Analyses

Mortality from selected underlying causes was evaluated separately for whites and nonwhites, as well as for combined races. Expected numbers of deaths were estimated by employing U.S. white male rates for whites and U.S. nonwhite male rates for nonwhites. Standardized mortality ratios (SMRs), calculated by dividing the number of observed deaths by expected deaths, were computed using Version F of the NIOSH Life Table Analysis System (LTAS) [Waxweiler et al., 1983; Steenland et al., 1990], which employs a person-years analysis [Monson, 1974]. Person-years

were accumulated by 5-year intervals of age, calendar time, length of service (i.e., tenure), and time since hire (i.e., latency). For this study, person-years began to accumulate for individual members of the cohort after 1 year of employment, a criterion for study inclusion, was achieved. An assumption that the observed number of deaths was Poisson distributed was used for estimating confidence intervals (CIs) and for statistical tests of whether SMRs differed from 1.00 [Breslow and Day, 1987]. All CIs expressed in this report are 95% CIs.

With regard to this report, the term "Pneumoconioses and Other Respiratory Diseases" (PORD) refers to the combination of ICD-8 codes 500 through 519, which includes several nonmalignant respiratory diseases including silicosis and chronic obstructive lung disease (see Table III footnote). This is the most specific output relating to pneumoconiosis available from the NIOSH LTAS software [Waxweiler et al., 1983; Steenland et al., 1990]. The term "peritoneal cancer" refers to the LTAS output combination of ICD-8 code 158 ("malignant neoplasm of peritoneum and retroperitoneal tissues," including peritoneal mesothelioma) and code 159 ("malignant neoplasms of unspecified digestive organs"). Finally, the term "lung cancer" refers to ICD-8 code 162 ("malignant neoplasm of trachea, bronchus, and lung").

RESULTS

Overall Mortality

Overall, all causes mortality rates for whites, for nonwhites, or for combined races were not significantly increased (Table III). An apparent "healthy worker effect" was evident in the low cardiovascular disease mortality rates—significant for whites (SMR: 0.85 CI: 0.73–0.99) and for combined races (SMR: 0.84; CI 0.75–0.94).

Pneumoconiosis Deaths

Based on review of all death certificates, four crushed stone workers in the analyzed cohort had "silicosis" or "pneumoconiosis" as an underlying or contributing cause of death. To be specific, one worker died with "silicosis" as the underlying cause, two workers died with "silicosis" as a contributing cause, and a fourth worker died with "pneumoconiosis" as a contributing cause. One of these workers had been hired in 1937, worked at a limestone operation for between 3 and 4 years in an undetermined capacity, and died in 1968 at the age of 73. A second had been hired in 1913, worked for 47 years as a foreman at a traprock operation, and died in 1976 at age 81. A third, an employee of the same limestone operation as the first case, had been initially hired in 1943, worked as a driller helper for nearly 1 year, was rehired in 1946, worked again as a driller helper for nearly 1 year, and died in 1978 at age 62. A fourth had been hired in 1957, worked as a driller helper for 20 years and as a truck driver for 1 year at a limestone operation, and died in 1979 at age 43 with silicosis listed as the underlying cause of death on his death certificate.

Pneumoconiosis and Other Respiratory Disease (PORD) Mortality

Twenty crushed stone workers in this cohort had an underlying cause of death classified as PORD. Specifically, these underlying causes of death were distributed as follows: chronic obstructive lung disease—15 deaths; chronic interstitial pneumonia—2 deaths; silicosis—1 death; lung abscess—1 death; and empyema—1 death.

TABLE III. Observed (O) and Expected (E) Number of Deaths and Standardized Mortality Ratios (SMRs) by Cause of Death and Race

		Whites			Nonwhites			Combined		
Cause of death	ICD code[a]	O	E	SMR (95% CI)	O	E	SMR (95% CI)	O	E	SMR (95% CI)
All causes	0–999	545	564.3	0.97 (0.89–1.05)	116	126.3	0.92 (0.76–1.11)	661	690.6	0.96 (0.89–1.04)
All malignant neoplasms	140–209	104	108.7	0.96 (0.79–1.17)	21	21.3	0.99 (0.61–1.51)	125	129.9	0.96 (0.80–1.15)
Cancer—peritoneum	158–159	5	0.5	9.74 (3.16–22.69)	0	0.1	— —	5	0.6	7.88 (2.55–18.36)
Cancer—lung	162	40	33.6	1.19 (0.85–1.62)	11	5.9	1.85 (0.92–3.31)	51	39.5	1.29 (0.96–1.70)
Diseases of circulatory system	390–458	249	293.1	0.85 (0.75–0.82)	45	58.2	0.77 (0.56–1.03)	294	351.3	0.84 (0.75–0.94)
Arteriosclerotic heart disease	410–413	172	201.8	0.85 (0.73–0.99)	26	28.3	0.92 (0.60–1.35)	198	230.1	0.86 (0.75–0.99)
Nonmalignant respiratory disease	460–519	30	32.9	0.91 (0.61–1.30)	9	7.3	1.23 (0.56–2.34)	39	40.3	0.97 (0.69–1.32)
Emphysema	492	3	7.5	0.40 (0.08–1.17)	2	0.7	2.88 (0.35–10.40)	5	8.2	0.61 (0.20–1.42)
Pneumoconiosis and other respiratory diseases (PORD)	500–519	16	8.7	1.85 (1.06–3.00)	4	1.4	2.79 (0.76–7.14)	20	10.1	1.98 (1.21–3.05)
All accidents	800–949	48	38.9	1.23 (0.91–1.64)	8	8.5	0.94 (0.41–1.85)	56	47.4	1.18 (0.90–1.55)
Total No. Person-years			48,892.3			7,054.3			55,946.6	
Total No. at Risk			2,867			379			3,246	

[a]8th Revision International Classification of Diseases [U.S. Dept. of HEW, 1967]. Codes include: 162—"malignant neoplasm of trachea, bronchus, and lung"; 158—"malignant neoplasm of peritoneum and retroperitoneal tissue"; 159—"malignant neoplasm of unspecified disgestive organs"; 500—"hypertrophy of tonsils and adenoids"; 501—"peritonsillar abscess"; 502 — "chronic pharyngitis and nasopharyngitis"; 503—"chronic sinusitis"; 504—"deflected nasal septum"; 505—"nasal polyp"; 506—"chronic laryngitis"; 507—"hay fever"; 508 — "other diseases of the upper respiratory tract," including vocal cord paralysis, vocal cord polyp, laryngeal edema, retropharyngeal abscess, etc.; 510—"empyema"; 511— "pleurisy"; 512—"spontaneous pneumothorax"; 513— "abscess of the lung"; 514—"pulmonary congestion and hypostasis"; 515—"pneumoconiosis due to silica and silicates"; 516—"other pneumoconioses and related diseases"; 517—"other chronic interstitial pneumonia"; 518 — "bronchiectatsis"; 519—"other diseases of respiratory system," including pulmonary collapse, mediastinitis, etc.

As seen in Table III, PORD SMRs were elevated for all race categories—statistically significant for whites (SMR: 1.85; CI: 1.06–3.00) and combined races (SMR: 1.98; CI: 1.21–3.05), but not for nonwhites (SMR: 2.79; CI: 0.76–7.14).

PORD SMRs are shown by tenure and latency in Table IV. For nonwhites, there were no deaths among those with < 10 years tenure or among those with < 20 years latency. However, the PORD SMR was substantially elevated and statistically sig-

TABLE IV. Pneumoconiosis and Other Respiratory Diseases (PORD)*: Observed (O) Number of Deaths and Standardized Mortality Ratios (SMRs) by Race, Tenure, and Latency for Males With at Least 1 Year Tenure

		Years tenure					
		<10		10+		Total	
Race	Years latency	O	SMR	O	SMR	O	SMR
Whites	<20	4	1.77 (0.48–4.53)	1	1.09 (0.03–6.07)	5	1.57 (0.51–3.66)
	20+	6	2.79 (1.02–6.08)	5	1.50 (0.49–3.50)	11	2.01 (1.00–3.60)
	Total	10	2.27 (1.09–4.18)	6	1.41 (0.52–3.07)	16	1.85 (1.06–3.00)
Nonwhites	<20	0	—	0	—	0	—
	20+	0	—	4	8.89 (2.42–22.76)	4	4.60 (1.25–11.78)
	Total	0	—	4	6.67 (1.81–17.08)	4	2.79 (0.76–7.14)
Combined	<20	4	1.49 (0.41–3.81)	1	0.93 (0.02–5.18)	5	1.33 (0.43–3.10)
	20+	6	2.33 (0.86–5.08)	9	2.38 (1.09–4.52)	15	2.36 (1.32–3.89)
	Total	10	1.90 (0.91–3.50)	10	2.06 (0.99–3.79)	20	1.98 (1.21–3.05)

*ICD-8 codes 500–519. Numbers in parentheses = 95% confidence interval.

nificant for nonwhites with 10 or more years tenure and 20 or more years latency (SMR: 8.89; CI: 2.42–22.76). Significantly elevated SMRs were also observed for all nonwhites with at least 10 years tenure (SMR: 6.67; CI: 1.81–17.08) and for all nonwhites with at least 20 years latency (SMR: 4.60; CI: 1.25–11.78).

In contrast, PORD SMRs for whites were significantly elevated for the subgroup with < 10 years tenure and at least 20 years latency (SMR: 2.79; CI: 1.02–6.08). Elevated SMRs were also observed for all whites with < 10 years tenure (SMR: 2.27; CI: 1.09–4.18) and for all whites with at least 20 years latency (SMR: 2.01; CI: 1.00–3.60). The PORD SMR was also elevated for all whites with 10 or more years tenure (SMR: 1.41; CI: 0.52–3.07).

In the combined races analysis, PORD SMRs were highest and statistically significant for the subgroup with 10 or more years tenure and 20 or more years latency (SMR: 2.38; CI: 1.09–4.52). In addition, a significant elevated SMR was observed for all workers with at least 20 years latency (SMR: 2.36; CI: 1.32–3.89).

Table V shows PORD SMRs by tenure, race, and rock type for workers with at least 20 years; 95%CI: 0.93 years latency. Overall, the SMRs for limestone workers (SMR: 2.04; CI: 0.93–3.88) and traprock workers (SMR: 1.44; CI: 0.17–5.20) were elevated, but not statistically significant. In contrast, PORD SMRs for granite workers were substantially elevated and significant for nonwhites with 10 or more years tenure (SMR: 14.33; CI: 2.95–41.84), for nonwhites overall (SMR: 14.04; CI: 2.89–41.00), and for combined races overall (SMR: 7.26; CI: 1.97–18.59). PORD

TABLE V. Pneumoconiosis and Other Respiratory Diseases (PORD)*: Observed (O) Number of Deaths and Standardized Mortality Ratios (SMRs) by Rock Type, Race, and Tenure for Males With 20 or More Years Latency

	Years tenure					
	<10		10+		Total	
Race	O	SMR	O	SMR	O	SMR
			Limestone			
Whites	5	2.83 (0.92–6.59)	3	1.40 (0.29–4.09)	8	2.04 (0.88–4.02)
Nonwhites	0	—	1	4.72 (0.12–26.29)	1	2.02 (0.05–11.25)
Combined	5	2.44 (0.79–5.68)	4	1.70 (0.46–4.35)	9	2.04 (0.93–3.88)
			Granite			
Whites	1	26.81 (0.68–149.3)	0	—	1	2.97 (0.08–16.54)
Nonwhites	0	—	3	14.33 (2.95–41.84)	3	14.04 (2.89–41.00)
Combined	1	23.81 (0.60–132.6)	3	5.90 (1.22–17.23)	4	7.26 (1.97–18.59)
			Traprock			
Whites	0	—	2	2.26 (0.27–8.16)	2	1.63 (0.20–5.88)
Nonwhites	0	—	0	—	0	—
Combined	0	—	2	2.19 (0.26–7.91)	2	1.44 (0.17–5.20)

*ICD-8 codes 500–519. Numbers in parentheses = 95% confidence interval.

SMRs were also significantly elevated for all granite workers with at least 10 years tenure and 20 years latency (SMR: 5.90; CI: 1.22–17.23).

Lung Cancer Mortality

As seen in Table III, lung cancer SMRs were elevated, although not statistically significant, for whites (SMR: 1.19; CI: 0.85–1.61), nonwhites (SMR: 1.85; CI: 0.92–3.31), or combined races (SMR: 1.29; CI: 0.96–1.70).

Lung cancer SMRs are shown by tenure and latency in Table VI. Among whites, the lung cancer SMR was not elevated for those with < 10 years tenure and < 20 years latency (SMR: 0.97; CI: 0.47–1.78). However, for all other subgroups with longer latency and/or tenure, lung cancer SMRs were elevated, although not statistically significant, the highest SMR occurring for the subgroup with the longest tenure and latency (SMR: 1.35; CI: 0.76–2.23).

Similarly, the lung cancer SMR among nonwhites was not elevated for those with < 10 years tenure and < 20 years latency (SMR: 0.64; CI: 0.02–3.56). However, for all other subgroups with longer latency and/or tenure, lung cancer SMRs were elevated, although not statistically significant, the highest SMR occurring

TABLE VI. Lung Cancer*: Observed (O) Number of Deaths and Standardized Mortality Ratios (SMRs) by Race, Tenure, and Latency for Males With at Least 1 Year Tenure

Race	Years latency	Years tenure <10 O	<10 SMR	10+ O	10+ SMR	Total O	Total SMR
Whites	<20	10	0.97 (0.47–1.78)	6	1.29 (0.47–2.81)	16	1.07 (0.60–1.73)
	20+	9	1.20 (0.55–2.28)	15	1.35 (0.76–2.23)	24	1.29 (0.83–1.91)
	Total	19	1.07 (0.64–1.67)	21	1.34 (0.83–2.05)	40	1.19 (0.85–1.61)
Nonwhites	<20	1	0.64 (0.02–3.56)	1	1.43 (0.04–7.97)	2	0.88 (0.11–3.18)
	20+	4	2.20 (0.60–5.63)	5	2.69 (0.87–6.27)	9	2.45 (1.12–4.66)
	Total	5	1.47 (0.48–3.43)	6	2.34 (0.86–5.10)	11	1.85 (0.92–3.31)
Combined	<20	11	0.92 (0.46–1.65)	7	1.31 (0.53–2.70)	18	1.04 (0.62–1.64)
	20+	13	1.40 (0.74–2.39)	20	1.55 (0.95–2.39)	33	1.48 (1.02–2.09)
	Total	24	1.13 (0.72–1.67)	27	1.48 (0.98–2.16)	51	1.29 (0.96–1.70)

*ICD-8 code 162. Numbers in parentheses = 95% confidence interval.

among the subgroup with the longest tenure and latency (SMR: 2.69; CI: 0.87–6.27). The lung cancer SMR for all those with at least 20 years latency was significantly elevated (SMR: 2.45; CI: 1.12–4.66).

In the combined races analysis, the lung cancer SMR was not elevated for those with < 10 years tenure and < 20 years latency (SMR: 0.92; CI: 0.46–1.65). However, for all other subgroups with longer latency and/or tenure, lung cancer SMRs were elevated, although not statistically significant, the highest SMR occurring among the subgroup with the longest tenure and latency (SMR: 1.55; CI: 0.95–2.39). The lung cancer SMR for all those with at least 20 years latency was significantly elevated (SMR: 1.48; CI: 1.02–2.09).

Table VII shows lung cancer SMRs by tenure, race, and rock type for workers with at least 20 years latency. Overall, the SMR for traprock workers was not elevated (SMR: 0.63; CI: 0.13–1.84). The overall lung cancer SMR for limestone workers (SMR: 1.50; CI: 0.95–2.25) was elevated, although not statistically significant. In contrast, the overall lung cancer SMR for granite workers (SMR: 3.35; CI: 1.34–6.90) was elevated and significant, with race-specific SMRs of similar magnitude for both whites and nonwhites. All of the lung cancer deaths in granite workers with > 20 years latency occurred among those with at least 10 years tenure. Thus the lung cancer SMR was elevated and significant for granite workers with at least 10 years tenure (SMR: 3.54; CI: 1.42–7.29), with race-specific SMRs of similar magnitude for both whites and nonwhites.

TABLE VII. Lung Cancer*: Observed (O) Number of Deaths and Standardized Mortality Ratios (SMRs) by Rock Type, Race, and Tenure for Males With 20 or More Years Latency

	Years tenure					
	<10		10+		Total	
Race	O	SMR	O	SMR	O	SMR
			Limestone			
Whites	8	1.34 (0.58–2.64)	10	1.37 (0.66–2.52)	18	1.36 (0.81–2.15)
Nonwhites	3	2.42 (0.50–7.07)	2	2.33 (0.28–8.41)	5	2.38 (0.77–5.55)
Combined	11	1.52 (0.76–2.72)	12	1.56 (0.81–2.73)	23	1.50 (0.95–2.25)
			Granite			
Whites	0	—	4	3.57 (0.97–9.14)	4	3.32 (0.90–8.50)
Nonwhites	0	—	3	3.45 (0.71–10.07)	3	3.37 (0.69–9.84)
Combined	0	—	7	3.54 (1.42–7.29)	7	3.35 (1.34–6.90)
			Traprock			
Whites	1	0.71 (0.02–3.95)	1	0.35 (0.01–1.92)	2	0.49 (0.06–1.77)
Nonwhites	1	1.79 (0.05–9.97)	0	—	1	1.45 (0.04–8.08)
Combined	2	1.02 (0.12–3.68)	1	0.36 (0.01–2.01)	3	0.63 (0.13–1.84)

*ICD-8 code 162. Numbers in parentheses = 95% confidence interval.

Pleural and Peritoneal Cancer Mortality

There was one death in this cohort coded as ICD-8 code 163 ("malignant neoplasm of other and unspecified respiratory organs"), which includes malignant neoplasm of the pleura. However, the underlying cause of death for this decedent was listed on his death certificate as "adenocarcinoma of the mediastinum," not mesothelioma. One other death was certified as having "malignant mesothelioma of the lung" as the only cause of death, but the death was coded as lung cancer (ICD-8 code 162.1). As seen in Table III, peritoneal cancer SMRs were significantly elevated for whites (SMR: 9.74; CI: 3.16–22.69) and combined races (SMR: 7.88; CI: 2.55–18.36). No deaths from peritoneal cancer were observed in nonwhites, where only 0.1 were expected. Only two of the five deaths coded as peritoneal cancer had specific mention of mesothelioma on death certificate. For the three deaths with death certificate mention of mesothelioma, age at hire, years tenure, and years latency were: 58, 4, 5; 43, 3, 16; and 31, 23, 23, respectively.

DISCUSSION

After the initial discovery of asbestos in a Maryland traprock operation, others confirmed that some crushed stone deposits are contaminated with asbestos and/or

other mineral fibers [Carter, 1977; Rohl et al., 1977; U. S. Environmental Protection Agency, 1979, 1981; Kullman et al., 1995]. Although a concern over occupational exposure to asbestos fibers in the crushed stone industry stimulated NIOSH to conduct a study of mortality among crushed stone workers, asbestos fibers were found in only one of the operations which were environmentally surveyed—the Maryland traprock operation at which the initial discovery of asbestos had been made [Kullman et al., 1995].

At the Maryland traprock operation where asbestos was identified, measured concentrations of airborne asbestos fibers in two out of 10 personal samples were above the NIOSH recommended exposure limit of 0.1 f/cc at the time of the environmental survey [Kullman et al., 1995]. Based on epidemiological results from other industries, the estimated percent increase in lung cancer mortality risk due to an average asbestos exposure of 0.1 f/cc over a 50-year working life would amount to 3% (SMR: 1.03) for tremolite [Amandus and Wheeler, 1987] and 0.7% (SMR: 1.007) for chrysotile [McDonald et al., 1980]. If the measured fiber concentrations at the Maryland traprock operation accurately reflect past exposures to asbestos, this study would not have the power to detect a statistically significant elevated SMR of that magnitude, even if all workers in the present cohort were similarly exposed.

The substantially elevated and statistically significant mortality from peritoneal cancer in this crushed stone worker cohort, and the three deaths with specific mention of mesothelioma as a cause of death, should raise concern with regard to the well-known asbestos fiber etiology of mesothelioma. However, given the ages at which these workers were first hired by the studied crushed stone operations, all three were likely to have worked in other jobs prior to their crushed stone employment. Also, an etiology related to occupational exposures associated with employment in the selected crushed stone operations is virtually excluded by the very short 5-year latency in one of the individuals. In the absence of further information, it seems unlikely that these three deaths were caused by occupational exposures associated with the studied crushed stone operations.

Although clearly inadequate to fully assess possible risk associated with asbestos exposure in the crushed stone industry, the collected occupational history and mortality data were analyzed to evaluate lung cancer mortality, as well as pneumoconiosis and other nonmalignant respiratory disease mortality, in this selected cohort of crushed stone workers. Of special concern, crystalline silica, a common air contaminant in crushed stone operations [Hammond, 1944; Baucom, 1986; Kullman et al., 1995], is known to cause silicosis. Chronic obstructive lung disease is associated with occupational exposure to dust in general [Becklake, 1989]. Biological evidence and plausibility for this association is strongest for mineral dusts retained in the lung (e.g., crystalline silica). Such dusts are associated with mineral dust airways disease characterized by functionally important tissue reactions in the small airways of the lung [Churg et al., 1985]. In addition, evidence associating silica or silicosis with lung cancer has recently generated substantial concern [Goldsmith et al., 1982; Heppelston, 1985; International Agency of Research on Cancer, 1987; McDonald, 1989; Simonato et al., 1990].

Review of death certificates for this cohort of crushed stone workers revealed four deaths with specific mention of silicosis or pneumoconiosis as an underlying ($n = 1$) or contributing ($n = 3$) cause of death. Of these decedents, one died at age 63 and one at 72. Each had < 4 years of tenure in a crushed stone operation located in

West Virginia, suggesting the possiblity that these workers may have been employed for substantial portions of their working years in other mining industries that would put them at risk for pneumoconiosis. Lacking further occupational history on these two workers, it is uncertain whether their disease was associated with crushed stone employment. In contrast, it seems reasonable to conclude that the worker who died of silicosis at age 43 after working as a driller helper for > 20 years in a crushed stone operation is likely to have developed silicosis as a result of dust exposure experienced in his crushed stone work. Likewise, another worker who died with silicosis did so after working 47 years (a full working lifetime) at a crushed stone operation, so it seems likely that his disease was also caused by exposures experienced in the course of that work.

With regard to PORD mortality, the overall SMR was significantly elevated (SMR: 1.98; CI: 1.21–3.05). By race, although the overall PORD SMR was not statistically significant for blacks (SMR: 2.79; CI: 0.76–7.14), it was significantly elevated for whites (SMR: 1.85; CI: 1.06–3.00). Furthermore, whereas the PORD SMR among whites was significantly elevated only for those with < 10 years tenure, PORD SMRs were substantially increased and statistically significant for the subgroup of nonwhites with high tenure and long latency. These apparently inconsistent observations may be plausibly explained by a past tendency (affirmed by statements made to the study project officer by some company officials) for nonwhites to more often be placed in and to remain for longer tenures in dustier crushed stone jobs.

By rock type, a key finding is that, among crushed stone workers with at least 20 years latency, the subgroup employed in granite operations experienced substantially elevated and significant PORD SMRs. Traprock and limestone workers also had overall elevated PORD SMRs, but these elevations were of a much lower magnitude and were not statistically significant. These observations parallel the generally higher observed airborne concentrations of respirable silica dust at the crushed stone operations processing granite [Kullman et al., 1995].

With regard to lung cancer, overall lung cancer SMRs were elevated, but not statistically significant. Among workers with at least 20 years latency, however, elevated lung cancer SMRs were statistically significant for nonwhites and for combined races overall. Considering all tenure/latency analyses, the low tenure and low latency subgroups consistently had lung cancer SMRs that were all < 1.00. Within latency strata, point estimates for lung cancer SMRs were all higher for the higher tenure subgroups. Within tenure strata, point estimates for lung cancer SMRs were all higher for the longer latency subgroups. This pattern is consistent with an occupational etiology associated with employment in the studied crushed stone operations.

By rock type, among those with 20 or more years latency, the lung cancer SMR was significantly elevated only for granite workers (SMR: 3.35; CI: 1.34–6.90). This finding is notable with regard to the generally greater occupational exposure to respirable crystalline silica at the granite operations [Kullman et al., 1995]. Together, these observations are consistent with the hypothesis that exposure to respirable silica dust is a risk factor for lung cancer. This hypothesis remains under active investigation by occupational epidemiologists in a variety of work settings. Some relevant studies, e.g., Carta et al. [1994], have presented negative findings. Others, including a study of diatomaceous earth workers by Checkoway et al. [1993], have presented clearly positive findings.

Several published studies that have addressed the issue of lung cancer among

granite workers are most relevant to our finding of significantly increased lung cancer mortality among the granite subcohort of crushed stone workers. In a proportionate mortality study of Vermont granite workers who had participated in a medical surveillance program between 1952 and 1978, Davis et al. [1983] observed a small, although nonsignificant, lung cancer excess but no evidence of an association of lung cancer mortality with either tenure or estimated level of dust exposure.

Based on a proportionate mortality ratio (PMR) analysis of records of the Granite Cutters Union in the United States, Steenland et al. [1986] reported a significantly increased lung cancer mortality among the cohort after exclusion of those who died of silicosis. A slight increase of lung cancer for the whole cohort was not significant and did not appear to be related to duration of exposure, although lung cancer was significantly associated with silicosis listed as a contributing cause of death.

Using a standardized mortality ratio (SMR) analysis, Costello and Graham [1988] found significantly increased lung cancer mortality among Vermont granite shed workers. More specifically, significant lung cancer excesses occurred among long-tenured workers with long latency who had been employed prior to 1930, when silica dust levels were notably high. For workers employed in later years, when dust levels were lower, this excess of lung cancer was not evident.

Koskela et al. [1987] conducted a cohort study of Finnish granite workers and produced evidence for a slight excess of lung cancer mortality overall, with clearly significant excesses among those with $>$ 15 years latency. Indirect evidence suggested that smoking could not account for the observed lung cancer excess.

In a mortality study of 159 Chinese granite workers registered as having silicosis, Chia et al. [1991] observed a twofold, although nonsignificant, lung cancer excess, which was not entirely explained by smoking. These authors observed a trend of increasing lung cancer associated with both increasing duration of exposure, as well as with increasing severity of silicosis.

In aggregate, the findings of these studies have not consistently supported an increased risk of lung cancer among granite workers that might be attributable to silica exposure, although the more recently published studies tend to provide more evidence for such an association. Although contributing to this evidence, the findings of our study of crushed stone workers cannot be considered conclusive.

CONCLUSIONS

Results of this study may not be generalizable to the crushed stone industry as a whole because studied operations did not represent a random sample of all operations in the crushed stone industry. However, this study identified important sentinel occupational mortality events and significant excesses of mortality that may be due to occupational exposure to dust generated during the processing of crushed stone.

Pneumoconiosis, a nonmalignant lung disease caused by the inhalation of mineral dust and nearly always related to occupational exposure, contributed to at least four deaths within this cohort according to death certificate documentation. Based on available work histories, it is highly likely that at least two of these four decedents incurred their exposures as a result of employment in crushed stone operations.

Mortality attributed to pneumoconiosis and selected other nonmalignant respiratory diseases was significantly increased overall (SMR: 1.98; CI: 1.21–3.05), and

especially so for a subcohort of crushed stone workers that processed granite (SMR: 7.26; CI: 1.97–18.59). Although not definitive, these and related observations are consistent with an occupational etiology related to employment in the studied crushed stone operations, particularly in light of findings from the environmental survey of these operations.

With regard to lung cancer, overall SMRs were elevated (although not statistically significant) for whites, nonwhites, and combined races (SMR: 1.29; CI: 0.96–1.70). By tenure and latency, mortality patterns were consistent with an occupational etiology related to employment in the studied crushed stone operations. Also, by rock type, there was a significantly elevated lung cancer SMR for granite workers with at least 20 years latency (SMR: 3.35; CI: 1.34–6.90). Although not definitive, these findings, which seem unlikely to be explained by potential confounding by either smoking or diesel exhaust exposure, are otherwise consistent with the hypothesis that exposure to respirable silica dust may be carcinogenic for humans.

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