R. T. Vanderbilt Company, Inc.
INDUSTRIAL MINERALS AND CHEMICALS
30 WINFIELD STREET, P.O. BOX 5150, NORWALK, CONNECTICUT 06856-5150
Tel. (203) 853-1400 • Fax: (203) 853-1452 • Internet Address: www.rtvanderbilt.com

April 11, 2001

Dr. C. W. Jameson
National Toxicology Program
Report on Carcinogens
MD-EC-14
P.O. Box 12233
Research Triangle Park, NC 27709

RECEIVED
APR 13 2001

Re: 10th ROC Nominations: Public Comment
Talc (containing asbestiform fibers)

Dear Dr. Jameson:

R. T. Vanderbilt Company, Inc., clearly the focus of the captioned nomination, has previously submitted extensive documentation. The purpose of this final submission is to provide pertinent documents previously not provided, and to summarize what the record supports on this nomination.

MINERALOGY

- It is obvious from the December review by the Board of Scientific Counselors that significant confusion exists over the very meaning of this nomination. The NTP background document repeatedly confused terminology and was incomplete and outdated. In addition, there was little indication that submissions to the NTP had been read or understood by the authors of this background document. As was stated in our earlier submissions and noted by panel members, if this nomination is intended to mean talc containing asbestos, it would be redundant since asbestos is already classed as a known human carcinogen. Many references in the background document addressed asbestos, and were therefore not appropriate to this nomination. If the nomination is intended to mean non-asbestos asbestiform fibers, then upwards of 100 minerals that can be formed in the asbestiform crystal growth habit would need to be addressed. Non-asbestos asbestiform minerals show differing biological effects, which are likely linked to differing physiochemical properties, harshness, disaggregation in the body, etc. Talc fiber, fibers of mixed mineral assemblage and xonotlite (which is water soluble), for example, do not exhibit an asbestos-like risk (see further discussion under "Health" below). These minerals can not be addressed on a "category" basis.

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R.T. Vanderbilt Company, Inc.

- There is a small but observable fibrous component in Vanderbilt talc (max. 2% by weight). No more than 1% by weight of this fibrous component can correctly be described as "asbestiform". These fibers are not asbestos, and consists of talc and mixed talc/amphibole fiber. Extensive characterization supporting these percents was previously cited and submitted to the NTP by a variety of sources (i.e., Wylie, Crane, Lee, Kelse, etc.). Many mineral scientists have gone out of their way to assist the NTP in understanding this admittedly confusing subject. While these fibers have been the subject of considerable academic study and some controversy, they are essentially a mineral curiosity and do not by any measure pose a broad public exposure. Indeed, documents submitted to the NTP record suggest there may be no public exposure to them at all (i.e., CPSC and crayons, paint sanding study). The industrial grade talc at issue (Vanderbilt talc) is exclusively used in applications that incorporate this talc in product matrixes such as wax, paint blends and ceramics. Thus, addressing a nomination with very little to no public exposure does not appear to be to be within the NTP's stated criteria for nomination and review.

  Attachment I presents a breakdown by particle type, size and crystal growth habit from three air samples in high dust areas in the Vanderbilt talc mill. Data of this sort confirm again the minor contribution asbestiform fibers make in Vanderbilt talc. Though these fibers are a very minor component in Vanderbilt talc, no other talc is known that contains more of these fibers, underscoring once again the extremely limited scope of this nomination.

HEALTH

- The only relevant health study used in the NTP background document in support of this nomination is a 1980 NIOSH Technical Report that addresses Vanderbilt miners and millers. As was pointed out in public comment and noted in our earlier submissions, there are 6 other, larger and more recent mortality studies addressing these same talc miners. It is true that an overall lung cancer excess persists at the Vanderbilt talc facility. However, of all the studies, only NIOSH suggests that the excess was caused by the dust exposure. The other mortality studies (previously submitted to the NTP record) conclude that this excess is not likely caused by the dust exposure.

  Attachments II & III contain copies of two linked studies, recently revised and submitted for publication (American Journal of Industrial Medicine). One study reflects the most recent, most comprehensive mortality study of Vanderbilt talc workers (Honda, et al), while the second describes in detail the exposure assessment protocol applied in the mortality study (Oestenstad, et al). These studies confirm what the earlier tenure data suggested - the lung cancer cases were exposed to 31% less cumulative dust than the cohort as a whole. In other words - an inverse dose response has been confirmed.

R. T. Vanderbilt Company, Inc.
INDUSTRIAL MINERALS AND CHEMICALS

Attachment IV contains a study, which was apparently overlooked in prior submissions (Lamm, et al; Proceedings of the 11th International Pneumoconiosis Conference, August 1988, pp. 1576-81). This study contrasts the lung cancer and nonmalignant respiratory disease mortality between Vanderbilt talc workers and Vermont talc workers with more than one year of exposure. Although study comparisons of this type are often problematic, this comparison involves similar cohort sizes, years of study and dust level exposures. Lung cancer mortality in both talc mining groups is no different, while nonmalignant respiratory disease mortality is much higher in Vermont than in New York. Dust exposure in Vermont was not reported to contain amphibole cleavage fragments or the minor fibrous component seen in New York talc. This comparison does not support the contention that the fibrous component in New York talc presents a greater risk than talc not containing this component.

- The NIOSH study (the only one that argues for a dust etiology) is now well understood to be a seriously flawed document. This was acknowledged by several NTP panel members in December. NIOSH claimed a 40-60% exposure to asbestos in the Vanderbilt talc mine and mill, which is patently incorrect. NIOSH incorrectly characterized common, garden-variety tremolite cleavage fragments as asbestos. That error is largely responsible for much of the confusion that has surrounded Vanderbilt talc for decades. The belief by NIOSH that a major asbestos exposure exists in this talc surely influenced the Institute's "causality" thinking. This is a fatally flawed work that should not be relied on in this forum or any other (especially when it is the only work in support of this nomination).

- Vanderbilt talc was directly tested against asbestos in two animal studies (Stanton, et al, and Smith). In each study the talc (containing asbestiform fibers) produced no tumors in the test animals while asbestos samples produced prolific tumors under the same test conditions. Copies of these animal studies have been submitted to the NTP record by Vanderbilt and others. These studies were neither mentioned in the NTP background document nor discussed by the Board.

- In addition, a cell study supports the Vanderbilt position on the minor fibrous component of its talc. A concentrate of these fibers was tested against an equal weight of actual asbestos. The fibrous concentrate from the talc behaved differently than the asbestos sample. The authors (Wylie, Mossman, et al) attributed the difference in biological response to differences in physiochemical properties (morphology was similar). While this study was cited in the NTP background document, its significance to the "fibers" in question was not recognized or appropriately reviewed. A copy of this study was previously submitted.

- Even assuming that the excess lung cancer recorded among Vanderbilt talc workers is linked to the dust (and presumably to the minor real fibers in it), the excess exists only among miners - not millers. Since Vanderbilt closed the underground mine in 1995, that "exposure" no longer exists. With regard to downstream exposures, Vanderbilt talc millers are exposed to dust which most closely matches that of the finished product as handled by customers. There is no significant lung cancer seen among millers. Health studies of downstream users are limited, but those that do exist (i.e., paint manufacturers' study - earlier submitted) show no excess lung cancer.

In summary, although this nomination exists, there is no clear understanding of its meaning. Assuming the nomination means the non-asbestos asbestiform fibers in Vanderbilt talc, the NTP is addressing a mineral curiosity with little or no exposure potential to anyone (not even Vanderbilt talc workers). Even if the NTP strives to justify a dubious nomination based upon a NIOSH finding of excess lung cancer among Vanderbilt talc workers, a dust etiology is not supported by larger, stronger and more recent studies. Animal and cell studies also lend no support to a causal link.

The NTP can not ignore the scientific record before it. No matter how the data is tortured - and Vanderbilt strongly believes a major bias exists on this nomination - the evidence in no way supports this nomination.

Very truly yours,

R. T. VANDERBILT COMPANY, INC.

[redacted]

John W. Kelse
Corporate Industrial Hygienist
Manager, Corporate Risk Management

JWK/sk

RJ Lee*Group*, Inc.

350 Hochberg Road
Monroeville, PA 15146
Tel: (724) 325-1776
Fax: (724) 733-1799

The Materials Characterization Specialists

November 22, 2000

Mr. John W. Kelse
R. T. Vanderbilt Company, Inc.
30 Winfield Street
Norwalk, CT 06856-5150

RE: TEM Asbestos Analysis
RJ Lee Group Job No.: LSH006444-3

Dear Mr. Kelse:

Enclosed are the results from the transmission electron microscopy (TEM) asbestos analysis of the above referenced samples using the counting rules established by the NIOSH Method 7402, issue 2, 8/15/94. The sample and volume information were provided by R. T. Vanderbilt Company, Inc. personnel.

The analysis for asbestos fibers consisted of fiber morphology, visual selected area electron diffraction (SAED) and elemental chemical analysis by energy dispersive spectroscopy (EDS), supplemented by the measurement and interpretation of micrographs of several selected SAED patterns. The samples were analyzed at a magnification of 1,000 X. Particles meeting the definition of a fiber > 5 µm in length, > 0.25 µm in width, and having a length to width aspect ratio ≥ 3:1 were classified as chrysotile, amphibole asbestos, amphibole cleavage, or transitional fiber.

The attached table lists each sample identification number, filter area, volume, area analyzed, asbestos fiber counts (f_s), analytical sensitivity, concentration of asbestos (f/cc), total fibers counted (F_s), and asbestos fiber ratio (f_s/F_s). Copies of the count sheets are presented in Appendix A. Each sheet contains sample information pertaining to structure identification, dimensions, magnification, filter size, and type.

RJ Lee Group, Inc. is accredited by the National Voluntary Laboratory Accreditation Program (NVLAP), New York Department of Health Environmental Laboratory Approval Program (ELAP), and by the American Industrial Hygiene Association (AIHA). This report relates only to the items tested and shall not be reproduced except in full. NVLAP accreditation does not imply endorsement by NVLAP or any agency of the US government. These results are submitted pursuant to RJ Lee Group's current terms and conditions of sale, including the company's standard warranty and limitation of liability provisions. No responsibility or liability is assumed for the manner in which the results are used or interpreted. Unless notified in writing to return the samples covered in this report, RJ Lee Group will store the samples for a period of 30 days before discarding. A shipping and handling fee will be assessed for the return of any samples.

If you have any questions, please feel free to call me.

Sincerely,

[redacted]

Drew R. Van Orden, PE
Senior Scientist

Monroeville, PA • San Leandro, CA • Washington, DC • Richland, WA

www.rjlg.com

TEST REPORT

Asbestos Concentrations and Fiber Ratios

NIOSH 7402 Analysis

Project LSH006444-3

RJLG Sample Number	Client Sample Number	Filter Area (mm^2)	Volume (Liter)	Area Analyzed (mm^2)	Asbestos Fibers (f_s)	Analytical Sensitivity (f/cc)	Asbestos Concentration (f/cc)	Total Fibers (F_s)	Fiber Ratio (f_s/F_s)	Analysis Date
0114780HT	F-11	385	190.0	0.1155	1	0.0175	0.0175	103.5	0.01	11/16/00
0114781HT	F-12	385	300.0	0.0908	0	0.0141	< 0.0141	98.0	0	11/16/00
0114782HT	F-13	385	120.0	0.1485	0	0.0216	< 0.0216	101.5	0	11/20/00

F-11 Below mill crusher
F-12 Center mills 1, 2, 3
F-13 Over packer – NYTAL 300

Volumes provided by R. T. Vanderbilt Company, Inc. were used to calculate analytical results and sensitivities. Analytical sensitivity is calculated based on one structure in the area analyzed.

Appendix A

TEM Count Sheets

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114780HT	Grid Openings	14
Client Sample #	F-11 / Below mill crusher	Total Asbestos	1
Microscope	2000 FX	Total Non-Asbestos	102.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	190.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
1	0.5	11.00	2.00	Amphibole		X		X	Tremolite	Cleavage
1	1	20.00	3.00	Nonasbestos		X		X		TF
1	1	17.00	2.00	Nonasbestos		X		X		TF
1	1	40.00	1.10	Nonasbestos		X		X		TF
1	1	9.00	0.40	Nonasbestos		X		X		TF
1	1	11.00	1.00	Amphibole		291		29817	Tremolite	Cleavage
1	1	12.50	0.50	Nonasbestos		290		29815		TF
2	1	8.20	0.30	Nonasbestos				X		TF
2	0.5	17.00	0.60	Nonasbestos				X		TF
2	1	11.25	0.70	Nonasbestos				X		TF
2	1	7.00	0.80	Nonasbestos				X		TF
2	1	8.00	1.50	Amphibole		X		X	Tremolite	Cleavage
2	0.5	12.50	2.50	Nonasbestos		X				TF
2	0.5	7.50	0.30	Nonasbestos		X				TF
2	1	5.50	1.20	Amphibole		X		X	Tremolite	Cleavage
2	1	6.50	1.10	Amphibole		X		X	Tremolite	Cleavage
2	1	17.00	0.30	Nonasbestos		X				TF
3	1	8.25	0.80	Amphibole		X		X	Tremolite	Cleavage
3	1	11.00	0.35	Nonasbestos		X		X		TF
3	1	10.25	1.10	Amphibole		X		X	Tremolite	Cleavage
3	1	10.50	0.90	Amphibole		X		X	Tremolite	Cleavage
3	1	11.00	1.50	Amphibole		X		X	Tremolite	Cleavage
3	1	8.50	0.50	Nonasbestos				X		TF
3	1	5.20	0.90	Nonasbestos				X		TF
3	1	10.00	2.50	Nonasbestos				X		TF
3	1	6.75	0.80	Nonasbestos				X		TF
3	1	13.50	0.35	Nonasbestos				X		TF
3	1	9.50	0.30	Nonasbestos				X		TF
3	1	6.50	1.00	Nonasbestos		X				TF
4	0.5	12.00	1.10	Nonasbestos		X		X		TF
4	0.5	7.25	1.00	Amphibole		X		X	Tremolite	Cleavage
4	1	6.00	0.90	Amphibole		X		X	Tremolite	Cleavage
4	1	11.00	1.10	Amphibole		X		X	Tremolite	Cleavage
4	1	25.00	1.10	Nonasbestos				X		TF
4	1	6.00	0.40	Nonasbestos		X				TF
4	1	5.75	0.50	Nonasbestos				X		TF
4	1	10.50	1.75	Amphibole		X		X	Tremolite	Cleavage
4	1	8.50	2.00	Amphibole		X		X	Tremolite	Cleavage
5	1	5.20	0.40	Amphibole		X		X	Tremolite	Cleavage
5	1	8.50	0.60	Nonasbestos				X		TF

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114780HT	Grid Openings	14
Client Sample #	F-11 / Below mill crusher	Total Asbestos	1
Microscope	2000 FX	Total Non-Asbestos	102.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	190.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
5	0.5	22.00	0.50	Nonasbestos				X		TF
5	0.5	23.00	0.65	Nonasbestos				X		TF
5	1	10.00	0.40	Chrysotile				29822		
5	1	6.00	0.60	Nonasbestos		X				TF
5	1	9.50	1.40	Amphibole		X		X	Tremolite	Cleavage
5	1	12.50	1.50	Nonasbestos				X		TF
5	0.5	12.50	0.50	Nonasbestos		X		X		TF
5	1	10.00	2.00	Nonasbestos				X		TF
5	1	8.30	0.60	Nonasbestos				X		TF
5	1	5.40	1.25	Amphibole		X		X	Tremolite	Cleavage
6	1	10.00	3.00	Amphibole		X		X	Tremolite	Cleavage
6	1	7.00	0.80	Amphibole		X		X	Tremolite	Cleavage
6	1	10.00	2.50	Amphibole		X		X	Tremolite	Cleavage
6	1	7.25	0.40	Nonasbestos				X		TF
6	1	5.40	0.90	Nonasbestos				X		TF
7	1	22.00	1.50	Nonasbestos		X		X		TF
7	1	6.00	1.50	Amphibole		X		X	Tremolite	Cleavage
7	1	7.00	1.00	Amphibole		X		X	Tremolite	Cleavage
7	1	5.50	0.35	Amphibole		X		X	Tremolite	Cleavage
7	1	12.50	0.50	Nonasbestos	B			X		TF
7	1	7.50	0.40	Nonasbestos				X		TF
7	1	17.50	0.30	Nonasbestos				X		TF
7	1	7.00	0.20	Nonasbestos				X		TF
7	1	10.00	1.00	Nonasbestos		X		X		TF
8	1	6.50	1.40	Amphibole		X		X	Tremolite	Cleavage
8	1	7.50	0.70	Amphibole		X		X	Tremolite	Cleavage
8	1	10.00	0.30	Nonasbestos				X		TF
8	1	16.00	2.00	Amphibole		X		X	Tremolite	Cleavage
8	1	6.00	2.00	Amphibole		X		X	Tremolite	Cleavage
8	1	10.00	0.50	Nonasbestos				X		TF
8	1	5.50	0.80	Nonasbestos				X		TF
8	1	12.50	0.50	Nonasbestos				X		TF
8	1	5.50	1.00	Amphibole		X		X	Tremolite	Cleavage
9	0.5	6.50	0.70	Amphibole		X		X	Tremolite	Cleavage
9	0.5	7.00	2.00	Nonasbestos		X				TF
9	1	7.50	0.50	Nonasbestos		X				TF
10	0.5	24.00	1.00	Nonasbestos		X				TF
10	1	6.50	1.00	Amphibole		X		X	Tremolite	Cleavage
10	1	26.00	0.50	Nonasbestos				X		TF
10	1	6.00	1.20	Amphibole		X		X	Tremolite	Cleavage

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114780HT	Grid Openings	14
Client Sample #	F-11 / Below mill crusher	Total Asbestos	1
Microscope	2000 FX	Total Non-Asbestos	102.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	190.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
10	1	8.00	1.00	Amphibole		X		X	Tremolite	Cleavage
10	1	5.50	0.30	Nonasbestos				X		TF
10	1	17.00	2.00	Nonasbestos		X				TF
11	1	6.50	0.30	Nonasbestos				X		TF
11	1	7.00	0.60	Amphibole		X		X	Tremolite	Cleavage
11	1	5.50	1.00	Nonasbestos				X		TF
11	1	7.00	0.50	Nonasbestos				X		TF
11	1	6.00	0.90	Nonasbestos				X		TF
11	1	6.00	0.70	Amphibole		X		X	Tremolite	Cleavage
11	1	7.00	0.80	Nonasbestos				X		TF
12	0.5	15.00	1.50	Nonasbestos		X		X		TF
12	1	12.00	2.50	Amphibole		X		X	Tremolite	Cleavage
12	1	7.00	0.50	Nonasbestos				X		TF
12	1	18.00	2.50	Amphibole		X		X	Tremolite	Cleavage
12	1	16.00	3.00	Nonasbestos				X		TF
12	1	17.00	1.00	Nonasbestos				X		TF
12	1	19.00	0.40	Nonasbestos				X		TF
13	0.5	6.00	0.60	Nonasbestos		X				TF
13	0.5	5.50	0.60	Nonasbestos				X		TF
13	1	8.00	0.50	Nonasbestos		X		X		TF
13	1	5.50	1.00	Nonasbestos		X		X		TF
13	1	6.00	1.00	Amphibole		X		X	Tremolite	Cleavage
13	1	7.00	1.30	Amphibole		X		X	Tremolite	Cleavage
13	1	8.00	1.75	Amphibole		X		X	Tremolite	Cleavage
13	1	5.50	1.25	Amphibole		X		X	Tremolite	Cleavage
13	1	10.00	2.50	Amphibole		X		X	Tremolite	Cleavage
14	0.5	15.50	1.10	Nonasbestos		X		X		TF
14	0.5	8.00	0.45	Amphibole	M	X		X	Tremolite	Cleavage
14	1	8.00	0.60	Nonasbestos		X		X		TF
14	1	13.00	3.20	Nonasbestos		X		X		TF
14	1	18.50	2.50	Amphibole		X		X	Tremolite	Cleavage
14	1	9.00	1.75	Amphibole		X		X	Tremolite	Cleavage

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114781HT	Grid Openings	11
Client Sample #	F-12 / Center mills 1, 2, 3	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	98
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	300.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
1	0.5	5.75	0.50	Nonasbestos		X				TF
1	1	6.50	1.00	Nonasbestos		X				TF
1	1	8.25	0.60	Nonasbestos		296		29830		TF
1	1	17.00	1.50	Nonasbestos		295		29828		TF
1	0.5	13.00	1.20	Nonasbestos		X		X		TF
1	0.5	24.00	5.25	Amphibole		X		X	Tremolite	Cleavage
2	1	13.00	2.00	Nonasbestos		X				TF
2	1	8.25	2.00	Amphibole		X			Tremolite	Cleavage
2	1	7.50	1.90	Amphibole		X			Tremolite	Cleavage
2	1	20.00	3.50	Amphibole		X			Tremolite	Cleavage
2	1	8.50	2.00	Nonasbestos		X				TF
2	1	12.00	0.50	Amphibole		X			Tremolite	Cleavage
2	1	7.00	1.50	Amphibole		X			Tremolite	Cleavage
2	1	6.00	0.70	Amphibole		X			Tremolite	Cleavage
2	1	10.00	1.75	Amphibole		297		29832	Tremolite	Cleavage
2	0.5	6.75	0.50	Nonasbestos	M					TF
3	1	6.00	0.50	Nonasbestos				X		TF
3	0.5	6.50	0.45	Nonasbestos				X		TF
3	1	8.50	1.50	Amphibole		X			Tremolite	Cleavage
3	1	6.25	1.20	Amphibole		X			Tremolite	Cleavage
3	1	9.00	1.00	Nonasbestos				X		TF
3	0.5	5.25	0.40	Nonasbestos	M			X		TF
3	0.5	19.00	0.50	Nonasbestos	M			X		TF
3	1	13.50	1.00	Nonasbestos		X				TF
4	1	8.50	2.00	Nonasbestos		X		X		TF
4	1	11.50	2.00	Nonasbestos		X		X		TF
4	1	37.00	2.00	Nonasbestos		X		X		TF
4	1	7.00	0.50	Nonasbestos		X		X		TF
4	1	7.00	1.00	Nonasbestos		X		X		TF
4	1	6.00	1.00	Amphibole		X		X	Tremolite	Cleavage
4	1	6.00	1.00	Nonasbestos		X		X		TF
4	1	8.00	1.00	Nonasbestos		X		X		TF
4	1	8.00	2.00	Amphibole		X		X	Tremolite	Cleavage
4	1	8.50	1.50	Nonasbestos		X		X		TF
4	1	7.50	0.40	Nonasbestos		X		X		TF
4	1	23.00	3.00	Nonasbestos		X		X		TF
4	1	6.50	1.20	Nonasbestos		X		X		TF
5	1	10.00	0.30	Nonasbestos		X		X		TF
5	1	10.00	2.00	Amphibole		X		X	Tremolite	Cleavage
5	1	22.00	0.90	Nonasbestos		X		X		TF

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114781HT	Grid Openings	11
Client Sample #	F-12 / Center mills 1, 2, 3	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	98
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	300.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
5	1	9.00	0.50	Amphibole		X		X	Tremolite	Cleavage
5	1	17.00	5.00	Nonasbestos		X		X		TF
5	1	9.00	0.50	Nonasbestos		X		X		TF
5	1	15.00	2.00	Amphibole		X		X	Tremolite	Cleavage
5	1	5.40	0.30	Nonasbestos		X		X		TF
5	1	47.00	2.50	Nonasbestos		X		X		TF
6	1	16.00	0.30	Nonasbestos		X		X		TF
6	1	8.50	0.50	Nonasbestos		X		X		TF
6	1	15.00	2.00	Amphibole		X		X	Tremolite	Cleavage
6	1	14.00	1.00	Nonasbestos		X		X		TF
6	1	5.50	1.00	Amphibole		X		X	Tremolite	Cleavage
6	1	21.50	1.00	Nonasbestos		X		X		TF
6	1	7.00	0.50	Amphibole		X		X	Tremolite	Cleavage
6	1	5.20	0.30	Nonasbestos		X		X		TF
7	1	11.00	1.50	Amphibole		X		X	Tremolite	Cleavage
7	1	5.50	0.60	Nonasbestos		X		X		TF
7	1	5.10	0.80	Amphibole		X		X	Tremolite	Cleavage
7	1	9.00	1.00	Amphibole		X		X	Tremolite	Cleavage
7	1	9.00	1.50	Amphibole		X		X	Tremolite	Cleavage
7	1	6.00	0.40	Nonasbestos		X		X		TF
7	1	15.00	1.30	Nonasbestos		X		X		TF
8	0.5	15.50	0.50	Nonasbestos		X		X		TF
8	1	9.50	1.00	Nonasbestos		X		X		TF
8	1	5.50	0.60	Nonasbestos		X		X		TF
8	1	8.50	2.00	Nonasbestos		X		X		TF
8	1	7.50	1.00	Nonasbestos		X		X		TF
8	1	10.00	1.50	Nonasbestos		X		X		TF
8	1	8.00	2.50	Nonasbestos		X		X		TF
8	1	15.00	3.00	Nonasbestos		X		X		TF
9	0.5	6.00	0.50	Nonasbestos		X		X		TF
9	1	10.50	1.00	Nonasbestos		X		X		TF
9	1	5.20	0.60	Nonasbestos		X		X		TF
9	1	23.50	0.50	Nonasbestos		X		X		TF
9	1	23.00	3.20	Amphibole		X		X	Tremolite	Cleavage
9	1	6.50	1.50	Nonasbestos		X		X		TF
9	1	6.00	0.40	Amphibole		X		X	Tremolite	Cleavage
9	1	8.00	0.50	Nonasbestos		X		X		TF
9	1	7.50	1.00	Nonasbestos		X		X		TF
9	1	8.00	0.90	Nonasbestos		X		X		TF
9	1	6.00	1.90	Amphibole		X		X	Tremolite	Cleavage

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114781HT	Grid Openings	11
Client Sample #	F-12 / Center mills 1, 2, 3	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	98
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	300.0 Liters
Analyst	TWS/LH	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
9	1	7.50	1.50	Amphibole		X		X	Tremolite	Cleavage
10	0.5	15.50	1.50	Nonasbestos		X		X		TF
10	1	8.00	0.30	Nonasbestos		X		X		TF
10	1	5.20	0.80	Nonasbestos		X		X		TF
10	1	9.00	0.40	Nonasbestos		X		X		TF
10	1	16.00	2.00	Nonasbestos		X		X		TF
10	1	21.00	1.00	Nonasbestos		X		X		TF
10	1	6.00	0.70	Nonasbestos		X		X		TF
10	1	7.00	0.60	Amphibole		X		X	Tremolite	Cleavage
10	1	18.00	2.50	Nonasbestos		X		X		TF
10	1	7.50	1.00	Nonasbestos		X		X		TF
11	1	6.00	0.80	Nonasbestos		X		X		TF
11	1	20.00	6.00	Nonasbestos		X		X		TF
11	1	7.50	0.60	Amphibole		X		X	Tremolite	Cleavage
11	1	5.50	1.00	Nonasbestos		X		X		TF
11	1	9.00	2.00	Amphibole		X		X	Tremolite	Cleavage
11	1	8.00	2.00	Amphibole		X		X	Tremolite	Cleavage
11	1	6.00	0.50	Nonasbestos		X		X		TF
11	1	6.50	2.00	Amphibole		X		X	Tremolite	Cleavage
11	1	6.00	1.50	Amphibole		X		X	Tremolite	Cleavage
11	1	8.20	0.30	Nonasbestos		X		X		TF
11	1	28.50	0.70	Nonasbestos		X		X		TF
11	1	5.50	0.70	Nonasbestos		X		X		TF

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114782HT	Grid Openings	18
Client Sample #	F-13 / Over packer – NYTAL 300	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	101.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	120.0 Liters
Analyst	TWS/BF	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
1	0.5	6.00	1.30	Amphibole		X		X	Tremolite	Cleavage
1	0.5	9.50	2.00	Nonasbestos		X		X		TF
1	1	10.25	2.20	Amphibole		X		X	Tremolite	Cleavage
1	0.5	9.00	1.50	Nonasbestos		X		X		TF
1	1	7.50	0.70	Nonasbestos		299		29836		TF
1	1	8.00	2.00	Amphibole		X		X	Tremolite	Cleavage
1	1	7.25	1.75	Amphibole		298		29834	Tremolite	Cleavage
1	0.5	6.25	1.10	Amphibole		X		X	Tremolite	Cleavage
1	1	9.00	0.80	Nonasbestos		X		X		TF
1	1	12.00	2.50	Nonasbestos		X		X		TF
2	0.5	7.00	0.50	Amphibole		X			Tremolite	Cleavage
2	1	10.00	2.40	Nonasbestos		X				TF
2	1	7.25	1.10	Amphibole				X	Tremolite	Cleavage
2	0.5	5.25	0.30	Nonasbestos	M			X		TF
2	1	5.50	0.60	Nonasbestos		X				TF
2	1	6.75	1.30	Amphibole		X			Tremolite	Cleavage
2	0.5	11.00	0.50	Nonasbestos		X		X		TF
2	1	14.50	3.00	Nonasbestos		X		X		TF
2	1	6.00	1.10	Amphibole		X		X	Tremolite	Cleavage
2	1	16.00	1.80	Nonasbestos		X		X		TF
3	1	8.50	1.50	Amphibole				X	Tremolite	Cleavage
3	1	8.00	2.00	Amphibole				X	Tremolite	Cleavage
3	1	8.25	1.10	Amphibole				X	Tremolite	Cleavage
3	1	11.50	0.35	Nonasbestos				X		TF
3	1	7.00	0.50	Nonasbestos				X		TF
4	1	10.50	1.30	Amphibole				X	Tremolite	Cleavage
4	1	8.00	0.80	Nonasbestos		X		X		TF
4	1	7.50	0.80	Amphibole				X	Tremolite	Cleavage
4	1	5.25	0.60	Amphibole				X	Tremolite	Cleavage
4	1	35.00	5.00	Amphibole		X			Tremolite	Cleavage
4	1	21.00	2.20	Nonasbestos		X		X		TF
4	1	8.00	0.90	Amphibole				X	Tremolite	Cleavage
5	0.5	7.00	0.90	Amphibole		X			Tremolite	Cleavage
5	1	7.00	0.80	Nonasbestos				X		TF
5	1	6.50	1.00	Nonasbestos				X		TF
5	1	11.50	0.50	Nonasbestos				X		TF
5	0.5	20.00	5.00	Amphibole		X			Tremolite	Cleavage
5	1	11.50	0.40	Nonasbestos				X		TF
6	1	11.00	1.00	Amphibole				X	Tremolite	Cleavage
6	1	12.50	3.00	Amphibole		X			Tremolite	Cleavage

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114782HT	Grid Openings	18
Client Sample #	F-13 / Over packer – NYTAL 300	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	101.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	120.0 Liters
Analyst	TWS/BF	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
6	0.5	10.25	0.40	Nonasbestos				X		TF
6	1	5.50	1.00	Nonasbestos				X		TF
6	1	7.00	1.50	Nonasbestos		X				TF
6	1	18.00	1.50	Amphibole				X	Tremolite	Cleavage
6	0.5	5.75	0.90	Nonasbestos				X		TF
6	1	7.00	0.50	Nonasbestos				X		TF
7	1	8.50	1.75	Amphibole				X	Tremolite	Cleavage
7	1	7.75	0.40	Nonasbestos				X		TF
7	1	5.75	0.50	Nonasbestos				X		TF
7	1	9.75	0.75	Nonasbestos				X		TF
8	1	7.50	0.60	Nonasbestos				X		TF
8	1	8.00	0.75	Nonasbestos				X		TF
8	1	8.50	0.40	Nonasbestos				X		TF
9	0.5	10.50	0.80	Amphibole				X	Tremolite	Cleavage
9	0.5	6.50	1.50	Amphibole				X	Tremolite	Cleavage
9	0.5	9.00	0.45	Nonasbestos				X		TF
9	1	18.00	1.00	Amphibole				X	Tremolite	Cleavage
9	1	5.75	0.90	Amphibole				X	Tremolite	Cleavage
9	1	5.50	0.90	Amphibole		X		X	Tremolite	Cleavage
9	1	6.50	0.80	Nonasbestos				X		TF
10	0.5	6.00	1.00	Nonasbestos				X		TF
10	1	5.50	1.00	Amphibole				X	Tremolite	Cleavage
10	1	9.50	1.00	Nonasbestos				X		TF
11	1	6.50	1.80	Amphibole		X		X	Tremolite	Cleavage
11	1	12.50	0.50	Nonasbestos				X		TF
12	1	6.50	0.80	Nonasbestos				X		TF
12	1	5.20	0.60	Amphibole		X		X	Tremolite	Cleavage
12	1	6.80	1.00	Amphibole		X		X	Tremolite	Cleavage
12	1	7.00	0.40	Nonasbestos		X		X		TF
12	1	20.00	3.00	Amphibole		X		X	Tremolite	Cleavage
13	0.5	19.00	2.50	Amphibole		X		X	Tremolite	Cleavage
13	1	5.20	0.50	Amphibole				X	Tremolite	Cleavage
13	1	5.50	0.80	Amphibole				X	Tremolite	Cleavage
13	1	16.00	1.50	Nonasbestos	B			X		TF
13	1	6.10	0.60	Amphibole		X		X	Tremolite	Cleavage
14	1	7.00	1.00	Nonasbestos				X		TF
14	1	7.30	0.70	Nonasbestos		X		X		TF
14	1	9.30	1.00	Amphibole				X	Tremolite	Cleavage
14	1	7.00	1.00	Nonasbestos		X		X		TF
14	1	6.00	0.80	Amphibole		X		X	Tremolite	Cleavage

RJ Lee *Group*, Inc.
Count Sheet

Client Name	R. T. Vanderbilt Company, Inc.		
Project Number	LSH006444-3	RJLG QA Number	HQ18755
RJLG Sample #	0114782HT	Grid Openings	18
Client Sample #	F-13 / Over packer – NYTAL 300	Total Asbestos	0
Microscope	2000 FX	Total Non-Asbestos	101.5
Accelerating Voltage	120 Kv	Filter	CE 385 mm^2
Magnification	1,000 X	Volume	120.0 Liters
Analyst	TWS/BF	Grid Opening Area	0.0083 mm^2
EDS Disk		Dilution Factor	1

Field	Fiber	Length µm	Width µm	Structure Type	Morph	EDS	Photo	SAED	Amphibole Type	Comment
14	1	6.50	1.00	Amphibole		X		X	Tremolite	Cleavage
14	1	9.90	2.20	Amphibole		X		X	Tremolite	Cleavage
15	1	15.00	2.00	Amphibole				X	Tremolite	Cleavage
15	1	9.50	2.30	Amphibole				X	Tremolite	Cleavage
15	1	16.00	2.00	Amphibole		X		X	Tremolite	Cleavage
15	1	6.00	0.60	Amphibole		X		X	Tremolite	Cleavage
15	1	14.00	1.60	Amphibole		X		X	Tremolite	Cleavage
15	1	5.20	1.00	Nonasbestos				X		TF
15	1	6.50	1.50	Nonasbestos		X		X		TF
15	1	8.50	1.60	Nonasbestos		X		X		TF
16	0.5	10.40	0.60	Nonasbestos				X		TF
16	0.5	7.30	1.20	Amphibole				X	Tremolite	Cleavage
16	0.5	18.50	3.50	Amphibole		X		X	Tremolite	Cleavage
16	0.5	7.00	0.40	Amphibole		X		X	Tremolite	Cleavage
16	0.5	9.00	1.40	Amphibole				X	Tremolite	Cleavage
16	1	5.20	0.50	Nonasbestos				X		TF
16	1	6.30	1.20	Amphibole				X	Tremolite	Cleavage
16	1	6.50	0.50	Amphibole				X	Tremolite	Cleavage
16	1	8.00	2.00	Nonasbestos				X		TF
17	0.5	15.00	2.00	Nonasbestos		X		X		TF
17	1	6.00	0.40	Amphibole				X	Tremolite	Cleavage
17	1	5.50	0.60	Nonasbestos		X		X		TF
17	1	7.00	0.50	Nonasbestos				X		TF
17	1	7.50	1.00	Amphibole				X	Tremolite	Cleavage
17	1	9.50	0.60	Nonasbestos		X				TF
17	1	7.00	1.00	Amphibole				X	Tremolite	Cleavage
17	1	6.00	0.26	Amphibole				X	Tremolite	Cleavage
17	1	9.00	0.60	Amphibole				X	Tremolite	Cleavage
18	0.5	6.00	0.60	Nonasbestos	B			X		TF
18	1	14.00	0.90	Amphibole				X	Tremolite	Cleavage
18	1	6.50	0.60	Amphibole				X	Tremolite	Cleavage
18	1	7.0	0.40	Nonasbestos				X		TF
18	1	14.00	1.10	Nonasbestos		X		X		TF

UAB SCHOOL OF PUBLIC HEALTH

Department of Epidemiology

March 30, 2001

Philip J. Landrigan, M.D., Editor
American Journal of Epidemiology
Post Office Box 1057
Mount Sinai School of Medicine
New York, New York 10029-6574

Dear Dr. Landrigan:

Enclosed are three copies each of two manuscripts, "Mortality among Workers at a Talc Mining and Milling Facility" and "Assessment of Historical Exposures to Talc at a Mining and Milling Facility " that we hope you will consider for publication. Although the mortality experience of these workers has been evaluated previously, the study described in the papers extends the follow-up period and incorporates several methodological improvements over prior studies. Improvements include the evaluation of mortality patterns by duration of employment and time since hire, the estimation of workers' cumulative exposure to respirable talc dust and the use of internal analyses to evaluate the relation between cumulative exposure to respirable dust and selected causes of death. We think these papers add important information to the ongoing deliberations about the classification of talc as a human carcinogen.

Thank you for considering the enclosed papers. They are original works that have not been published or submitted for publication elsewhere in any part or form. If you have any questions about this study, please call me, Dr. Honda or Dr. Oestenstad any time. If the question involves a technical, rather than a substantive, issue, I am probably easier to reach than Dr. Honda. I can be reached by telephone at 205-934-7164 or by email at colleen@uab.edu.

Sincerely,

[redacted]

Colleen Beall, DrPH
Assistant Professor

Enclosures/

The University of Alabama at Birmingham
220A Ryals Building • 1665 University Boulevard
Birmingham, Alabama 35294-0022 • (205) 934-1700 • FAX (205) 975-7058

03/20/01

MORTALITY AMONG WORKERS AT A TALC MINING AND MILLING FACILITY

Yasushi Honda, M.D.[1]

Colleen Beall, Dr. P.H.[2]

Elizabeth Delzell, S.D.[2]

Kent Oestenstad, Ph.D.[3]

Ilene Brill, M.P.H.[2]

Robert Matthews, B.S.[2]

Short Running Title: MORTALITY IN TALC WORKERS

Text word count: 4,337

[1] Environmental Health Sciences Program, Institute of Health and Sport Sciences , University of Tsukuba, Japan

[2] Department of Epidemiology and International Health, University of Alabama at Birmingham School of Public Health

[3] Department of Environmental Health Sciences, University of Alabama at Birmingham School of Public Health

Address correspondence to Dr. Yasushi Honda
Environmental Health Sciences Program
Institute of Health and Sport Sciences
University of Tsukuba
Tsukuba 305-8574, Japan
Phone/Fax: +81-298-53-2627
e-mail: honda@taiiku.tsukuba.ac.jp

ABSTRACT

Background This study evaluated mortality among workers at a talc mining and milling facility.

Methods Subjects were white men actively employed between 1948 and 1989 and known to have been alive in or after 1950. Analyses assessed cancer mortality during the period 1950-1989 (809 subjects) and noncancer mortality during 1960-1989 (782 subjects).

Results Comparisons with regional general population death rates for 1960-1989 indicated that the workers had more than expected deaths from all causes combined (209 observed/160 expected, standardized mortality ratio (SMR)=131, 95% confidence interval (CI)=114-150), due mainly to increased mortality from lung cancer (31/13, SMR=232, CI=157-239) and nonmalignant respiratory disease (NMRD) (28/13, SMR=221, CI=147-320). The lung cancer excess was concentrated in miners (18/4.6, SMR=394, CI=233-622); millers had only a small increase (7/5.5, SMR=128, CI=51-263). An excess of NMRD occurred both in miners (10/4.2, SMR=241, CI=116-444) and in millers (11/4.8, SMR=227, CI=113-407). The median mg/m^3-days of estimated exposure to respirable dust was 425 for all exposed employees, 739 for mine workers and 683 for mill workers. Employees with high, compared to low, estimated exposure to dust had a rate ratio (RR) of 0.5 (CI=0.2-1.3) for lung cancer and of 11.8 (CI=3.1-44.9) for pulmonary fibrosis.

Conclusions Exposure to talc ore dust may not have been responsible for the lung cancer excess among these workers but probably contributed to the elevated rate of NMRD, particularly pulmonary fibrosis.

Keywords: TALC, OCCUPATIONAL EXPOSURE, EPIDEMIOLOGY, LUNG NEOPLAMS

INTRODUCTION

This study evaluated mortality among workers at an industrial-grade (tremolitic) talc mining and milling facility in upstate New York (NY). Four retrospective follow-up studies [Brown and Wagoner, 1978; Stille and Tabershaw, 1982; Lamm et al., 1988; Brown et al., 1990] and one nested case-control study of lung cancer [Gamble, 1993] previously assessed the mortality experience of these workers. The most recent of these included observations through 1983 [Brown et al., 1990]. The present study extended follow-up through 1989 and examined mortality in the overall group of workers and in subgroups specified on the basis of work area, years since hire, years worked and estimated cumulative exposure to respirable dust. Of particular interest were lung cancer and nonmalignant respiratory disease (NMRD) mortality patterns.

METHODS

Subjects were white men who worked for at least one day at the plant from the start of its operations in 1948 through the end of 1989, whose vital status was known in or after 1950 and who had known birth and employment dates. We restricted the study to white men because women and black men comprised only about five per cent of the workforce.

Data files from previous studies, plant personnel records and Internal Revenue Service Forms-941 identified 818 men actively employed at the facility from 1948 through 1989 and provided information on personal and employment characteristics. We later excluded nine men who were lost to follow-up before 1950, leaving 809 eligible subjects (see below). For each job held by a subject detailed work history information included the title, work location and the start and termination date. Information on specific jobs was not available for 37 short-term employees (5% of 809), who had an average work duration of only 0.14 year. We included these men in most analyses, counting them as having worked in an unknown plant location, but excluded them from cumulative exposure analyses (see below). We classified subjects' jobs into one of 12 work area groups, specified on the basis of similarity in tasks, production activities and respirable dust exposure potential [Oestenstad et al., submitted]. A long-term plant supervisor, relying on personal knowledge of operations, production records, dust control information and historical environmental reports specified time periods during which exposures in each area would have been relatively uniform and assigned for each area and time period combination exposure scores on a scale from zero to 10. The work area/time period combinations were the basis for developing a job-exposure matrix. We used this matrix and actual measurements of current respirable dust concentrations to estimate historical exposures and summed exposures across time periods to develop cumulative respirable dust exposure estimates.

Vital status information came from company records, the National Death Index and Pension Benefit Information, which maintains mortality data from the Social Security Administration death master file and other sources. We also used personal contact, credit bureau records and linkage with the NY Division of Motor Vehicles to determine the vital status of some subjects.

The company provided some death certificates,. We obtained additional death certificates from the state of death for subjects who died outside of NY. A trained nosologist classified underlying cause of death using the Eighth Revision of the International Classification of Diseases (ICD) and the coding rules in effect at the time of death. For most decedents dying in NY, the state provided cause of death data from its computerized decedent database. We converted the cause of death codes from this database, which were coded according to the ICD revision in effect at the time of death, to Eighth Revision codes.

The standardized mortality ratio (SMR) was the measure of association used to compare workers' mortality rates with the rates of the general population of the region consisting of the county in which the plant was located and five other counties that lay, at least partly, within 50 miles (presumed commuting distance) of the plant. The combined 1970 male population of the six counties was about 185,072 [U.S. Bureau of the Census, 1972]. We computed SMRs as ratio of observed to expected numbers of deaths, multiplied by 100, using the Occupational Mortality Analysis Program [Marsh et al., 1998]. We obtained expected numbers of deaths by multiplying the age- and calendar-time specific person-years (PY) of follow-up of the workers by the corresponding mortality rates of the regional white male general population and summing over the stratifying variables. We calculated 95% confidence intervals (CIs) of the SMRs assuming a Poisson distribution for the observed number of deaths.

Regional general population death rates needed for most analyses were available beginning in 1950 for cancers and in 1962 for noncancer causes of death [Marsh et al., 1998], and we used 1962-1964 rates to estimate general population rates for 1960-1964. The follow-up period for assessing cancer mortality began on the later of the hire date or on January 1, 1950, whereas the follow-up period for assessing overall and noncancer mortality began on the later of the hire date or January 1, 1960. Follow-up ended on December 31, 1989, on the death date or on the loss-to-follow-up date, whichever was earliest. Analyses of the 1950-1989 time period included 809 men. Analyses of the 1960-1989 time period included 782 men. The 27 exclusions from the 809 subjects eligible for the 1950-1989 time period were 16 men who died before 1960 and 11 who were lost to follow-up before 1960.

For certain causes of death, we analyzed subgroups specified on the basis of period of hire, years since hire, years worked, work area and cumulative dust exposure, beginning PY accumulation on the later of the first date in a particular category of each variable or on the default follow-up start date mentioned above. We combined work areas into five nonmutually exclusive groups (mills, mines, minimal exposure potential, no exposure potential, unknown). We also carried out Poisson regression analyses with an internal referent group to obtain maximum likelihood estimates of rate ratios (RRs) and their 95% CIs [Callas et al., 1990]. In the latter analyses all models included terms for age (<35, 35-54, 55-64, 65+) and either years since hire (<10, 10-19, 20+) or calendar year (1950-1959, 1960-1969, 1970-1979, 1980-1989), as well as for the other occupational variables of interest. Causes of death examined in Poisson regression analyses included: 1) lung cancer; 2) ischemic heart disease; 3) all NMRD; 4) "other NMRD," excluding pneumonia, influenza, asthma, emphysema and bronchitis and including chronic obstructive pulmonary disease (COPD) and "pulmonary fibrosis" (talcosis, pneumoconiosis, asbestosis and unspecified pulmonary fibrosis); and 5) pulmonary fibrosis alone. Other NMRD comprises a pathologically heterogeneous group of diseases but was examined because it is a standard cause of death category in SMR analyses [Marsh et al., 1998].

Poisson regression analyses incorporating exposure to respirable dust included 772 subjects for whom exposure estimates were available. For these analyses, we specified cumulative exposure categories as exposure tertiles among exposed decedents in the disease group being analyzed, combining the zero exposure group and lowest tertile to form the referent category. The death certificates of 16 decedents indicated COPD (N=8) or pulmonary fibrosis (N=8) as a contributory, rather than underlying, cause of death. We included these decedents in certain Poisson regression analyses.

RESULTS

Of the 782 men included in analyses of the 1960-1989 time period, 159 (20%) were active employees at the close of the study, and 623 (80%) were terminated or retired. Five hundred sixty-seven (73%) were classified as alive, 209 (27%) as deceased, and 6 (1%) as having an undetermined vital status. Of the 809 men included in analyses of the 1950-1989 time period, 159 (19%) were active employees at the close of the study, and 650 (81%) were terminated or retired. Five hundred sixty-seven (70%) were presumed living, 225 (28%) were deceased, and 17 (2%) had an undetermined vital status. We obtained underlying cause of death information from death certificates (N=134) or from the NY decedent file (N=86) for 220 (98%) of the 225 decedents.

For the 1960-1989 time period, subjects had a total of 15,050 PY of follow-up, an average of 19 years per man, and median values of 1962 for hire year, 27 years for age at hire and 3.0 years for duration of employment. For the 1950-1989 time period, subjects had a total of 18,048 PY of follow-up, an average of 22 years per man, and median values of 1960 for hire year, 27 years for age at hire and 2.3 years for duration of employment.

Compared to the regional general population of white men, the 782 talc workers followed up in 1960-1989 experienced a 31% increase in overall mortality, based on 209 observed/160 expected deaths (SMR=131, CI=114-150) (Table I). Excesses were largest for tuberculosis (3/0.4, SMR=788, CI=153-2303), all cancer (53/35, SMR=151, CI=113-198) and NMRD (28/13, SMR=221, CI=147-320). There were about 10% more deaths than expected from ischemic heart disease (69/63, SMR=110, CI=86-139).

The overall increase in NMRD deaths was not limited to a particular form of respiratory disease but was greatest for other NMRD (COPD and fibrosis) (17/5.9, SMR=289, CI=168-463).

This category included chronic obstructive pulmonary disease (N=10), asbestosis (N=1), pneumoconiosis (N=5) and chronic pulmonary fibrosis (N=1).

The excess of cancer deaths during the 1950-1989 follow-up period (54/37, SMR=146, CI=110-191) (Table II) was similar to that in 1960-1989 and was attributable mainly to increased mortality from respiratory cancer (34/14, SMR=239, CI=165-334), including lung cancer (31/13, SMR=232, CI=157-329) and larynx cancer (2/0.6, SMR=316, CI=38-1142). The remaining respiratory cancer decedent's death certificate indicated that he had "adenocarcinoma of the mediastinum." There were several more deaths than expected from lymphohematopoietic cancer (7/3.7, SMR=192, CI=77-395). This category included two decedents with non-Hodgkin's lymphoma, two with Hodgkin's disease, two with leukemia and one with multiple myeloma. Decedents' death certificates reported two deaths from mesothelioma. NY nosologists had coded one of these as ICD code 212 (benign neoplasm of the respiratory system) and the other as ICD code 162.9 (malignant neoplasm of bronchus and lung, unspecified), despite specific mention of mesothelioma.

Increases in mortality from all causes combined, all cancer, lung cancer and ischemic heart disease were limited to men hired before 1955 (Table III). This subgroup had 28 lung cancer deaths compared to 9.8 expected (SMR=286, CI=190-414), whereas men hired in or after 1955 had three deaths compared to 3.6 expected. For all NMRD and for other NMRD, both period of hire subgroups experienced increased mortality, but the larger increase occurred in men hired before 1955 (all NMRD: 23/9.5, SMR=242, CI=153-363; other NMRD: 15/4.2, SMR=355, CI=199-586). Men hired in or after 1955 had five NMRD deaths compared to 3.1 expected.

In analyses of mortality patterns by years since hire and years worked, data on lung cancer, all NMRD and other NMRD were sparse for the subgroups with <20 years since hire (Table IV).

In the subgroup with 20+ years since hire, there was some suggestion that SMRs and RRs increased consistently, although weakly, with increasing length of employment for NMRD. This was not seen for lung cancer or for ischemic heart disease.

Among the 782 men included in analyses of the 1960-1989 time period, 48% had worked in the talc mills for a median of 1.8 years, 40% had worked in mines for a median of 2.0 years, 23% had worked in areas involving minimal exposure for a median of 1.7 years, and 11% had worked in areas involving no exposure to talc for a median of ten months (Table V). A total of 67 subjects (9%) had spent a median of less than three months in an unknown work area.

The overall excess of lung cancer was concentrated among men ever employed in the mines (18/4.6, SMR=394, CI=233-622) (Table V). In contrast, mill workers had only a small increase in lung cancer (7/5.5, SMR=128, CI=51-263). Overall NMRD mortality was elevated both in mill workers (11/4.8, SMR=227, CI=113-407) and in miners (10/4.2, SMR=241, CI=116-444). Other NMRD deaths also were elevated in mill workers (6/2.3, SMR=266, CI=98-579) and in miners (8/1.8, SMR=434, CI=189-856). Results of Poisson regression analyses were consistent with these patterns. Some of the other work area groups had slight increases in deaths from lung cancer and/or NMRD, but these results were based on small numbers. Mutually exclusive work area analyses confirmed these patterns (data not displayed in a table). For example, the SMR for overall NMRD was 257 (11/4.8, CI=128-460) for men employed in mills but never in mines and was 277 (10/3.6, CI=133-510) for men employed in the mines but not the mills. For ischemic heart disease, there were 22% more than expected deaths among mill workers (31/26, SMR=122, CI=83-173) and 40% fewer than expected deaths in the group with minimal exposure potential (13/22, SMR=60, CI=32-102). Other work area groups had trivial differences in observed and expected numbers of deaths in this disease category.

Among 772 men with work history information adequate for estimating exposure to respirable dust, the median estimated cumulative dust level was 425 mg/m^3-days for all exposed subjects combined, 739 mg/m^3-days for men ever employed in mines and 683 mg/m^3-days for men ever employed in mills (Table VI). Among exposed decedents, the median estimated cumulative respirable dust exposure was 520 mg/m^3-days for all decedents combined, 347 mg/m^3-days for men with lung cancer, 376 mg/m^3-days for men with ischemic heart disease, 888 mg/m^3-days for men with any form of NMRD as the underlying cause of death, 1,199 mg/m^3-days for men with other NMRD as the underlying or a contributing cause and 3,759 mg/m^3-days for men with pulmonary fibrosis as the underlying or a contributing cause. These data indicate that estimated cumulative exposure was 32% lower for lung cancer decedents, and over eight times higher for decedents with pulmonary fibrosis, than for the overall group of decedents.

Poisson regression analyses indicated that there was an inverse association between estimated cumulative respirable dust exposure and lung cancer, with an RR of 0.5 (CI=0.2-1.3) for men in the highest cumulative exposure tertile compared to men in the lowest tertile (Table VII). Ischemic heart disease was not associated with dust exposure. For the series of all NMRD coded as the underlying cause of death, the RR was elevated in the two higher tertiles of estimated cumulative respirable dust exposure as compared to the lowest exposure tertile, but the dose-response pattern was irregular. In contrast, when the decedent series was limited to other NMRD (i.e., excluding pneumonia, influenza, emphysema, asthma and bronchitis) and expanded to include those with other NMRD as a contributory cause of death, the RR increased regularly with increasing cumulative exposure and was 1.0, 1.8 (CI=0.8-4.4) and 2.1 (CI=0.9-5.1) for the lowest, middle and highest tertile, respectively. The RR also increased with increasing exposure for pulmonary fibrosis and was 1.0, 2.2 (CI=0.7-7.4) and 11.8 (3.1-44.9) for the lowest, middle and highest tertile, respectively.

DISCUSSION

Many of the mortality patterns seen in the present study were similar to patterns reported previously for workers at the same plant [Brown and Wagoner, 1978; Stille and Tabershaw, 1982; Lamm et al., 1988; Brown et al., 1990; Gamble, 1993]. Compared to the regional general population, the employees experienced increased mortality rates for most diseases, particularly for lung cancer and NMRD. Men hired in 1955 or later had mortality rates that were similar to regional population rates for disease categories other than NMRD, including all causes, all cancer and lung cancer. These results could be interpreted as indicating that any exposure at the plant related to lung cancer or conditions except NMRD was removed or controlled effectively by the mid 1950s. However, data on subjects who began working in or after 1955 were too imprecise to exclude the possibility of a continuing small excess of deaths from all causes combined or a moderate lung cancer increase. Further, subjects hired in or after 1955 have had a shorter period of time for the expression of exposure-related diseases with long induction times. Thus, additional follow-up will be required to determine if these subjects are free of excess disease.

The lung cancer excess in the overall study group was moderately strong and was concentrated in the follow-up period 20 or more years since hire, results that suggest that some aspect of employment at the plant may have been associated with lung cancer. In a nested case-control study of 22 of the 31 lung cancer decedents identified in the present study, Gamble [1993] reported that all of the lung cancer cases and 73% of employee controls had been smokers. These smoking prevalences are high, but it is unlikely that the entire moderately strong lung cancer excess, particularly the nearly fourfold increase among miners, was attributable to smoking [Blair et al., 1995]. However, several observations from the present study indicate that exposure to talc at the facility may not have been responsible for the excess.

First, although men who worked in the underground mine, a high talc dust exposure area, experienced a greater than fourfold increase in lung cancer, we found little evidence of an increase among mill workers, a group with similar exposure. In addition, the estimated dust exposure was low for lung cancer decedents compared to other workers, and internal analyses of lung cancer rates by cumulative exposure indicated an inverse relationship. These findings do not support the hypothesis that talc dust at this facility has a carcinogenic potential similar to that of asbestos, which typically produces a moderate to strong positive dose-response relationship [Seidman et al., 1986; Goodman et al., 1999]. Also, because internal analyses should be minimally subject to confounding by nonoccupational exposures, the absence of a positive dose-response pattern does not support the hypothesis that the talc ore of the plant per se is a lung carcinogen.

Studies of other occupational groups have not provided evidence that talc ore per se causes lung cancer. A study of 389 Norwegian talc miners and millers exposed to nonasbestiform talc with low quartz content indicated no excess lung cancer incidence (6 observed/6.9 expected) [Wegelund et al., 1990]. Another investigation found a lung cancer deficit among Italian talc miners and millers exposed to nonasbestiform talc (12 observed/23 expected) [Rubino et al, 1976; Rubino et al., 1979]. In that study, miners, who were exposed to silica as well as talc, experienced a threefold increase in NMRD deaths; whereas the millers, who had low silica exposure, did not experience such an excess. Straif et al. [2000] reported that German rubber industry workers with high talc exposure (8 10 years of high exposure vs. < 0.5 years of medium and high exposure levels combined) had increased lung cancer mortality (RR=1.9, CI=1.1-3.1); however, the results were not controlled for exposure to asbestos, which was present in that industry, or for other agents to which the workers were likely to have been exposed.

Because of the high nonasbestiform amphibole content of the ore and dust at the facility investigated in the present study, research on other workers exposed to amphiboles is particularly relevant to our findings [Wylie et al., 1985; Kelse and Thompson, 1989; Wylie et al., 1993; U.S. Department of Labor, 2000]. Retrospective follow-up studies of workers exposed to taconite, which contains the nonasbestiform amphibole, cummingtonite-grunerite, reported no association with lung cancer or NMRD [Higgins et al., 1983; Cooper et al., 1992]. Investigations of gold miners exposed to silica, in addition to cummingtonite-grunerite and small amounts of tremolite-actinolite, found an increase in NMRD deaths but no excess of lung cancer [McDonald et al., 1978; Brown et al., 1986].

In addition, several animal studies have evaluated the carcinogenicity of nonasbestiform amphiboles, talc per se and individual components of the talc ore found at the study facility [Wagner and Berry, 1969; Pott et al., 1974; Smith et al., 1979; Stanton et al., 1981; McConnell et al., 1983; Davis et al., 1991]. Results of these studies indicated that nonasbestiform amphibole minerals in general and talc ore in particular did not increase the incidence of tumors, whereas asbestos was carcinogenic under the same experimental conditions [Wagner and Berry, 1969; Pott et al., 1974; Smith et al., 1979]. Both talc and asbestos are cytotoxic in cell culture; however asbestos, but not talc, has demonstrated proliferative potential in some cells [Wylie et al., 1997].

NMRD mortality patterns differed from those seen for lung cancer in several respects. NMRD was elevated both among subjects hired before 1955 and among subjects hired in 1955 or later, although the increase in the latter group was based on small numbers. NMRD was increased both among miners and among mill workers and was positively associated with increasing duration of employment. Moreover, NMRD decedents with pneumoconiosis or interstitial lung disease, the group most likely to include dust-related disease, had a median cumulative dust exposure that was eight times higher than the corresponding values for the overall study group. Internal comparisons indicated a positive relation between estimated cumulative dust levels and this category of NMRD.

As with lung cancer, patterns of smoking and occupational exposures in jobs before and after those held at the study facility may explain some of the overall excess of NMRD seen in our analyses. The observation of elevated SMRs among short-term workers is consistent with this interpretation. In addition, pre-employment records were available for 25 of the 28 men who had NMRD as the underlying cause of death. Of these, 20 had worked in other mining operations before starting work at the talc facility under study. Exposures sustained in these other mining operations are likely to have contributed to the development of respiratory disease. However, the impact of potential confounding by such factors should have been reduced in internal analyses. Similarly, any observation bias due to selective reporting of NMRD on the death certificates of deceased talc workers should have been lower in the internal analyses than in the external analyses. On balance, the positive associations seen in these analyses support a causal association between exposure to the talc ore dust at this plant and NMRD. The fact that we observed excess NMRD mortality when our exposure estimates suggested concentrations of respirable talc dust lower than the current threshold limit value of 2 mg/m^3 is of concern [Oestenstad et al., submitted]. However, because workers may have sustained exposures in other jobs that contributed to the etiology of NMRD, doubt remains about the hazard associated with talc dust levels below 2 mg/m^3.

Comparisons of the employees with the regional general population indicated a slight increase in ischemic heart disease deaths. Ischemic heart disease rates were not, however, associated consistently with employment duration, time since hire or cumulative exposure to respirable dust. Other results included a small increase in deaths from lymphopoietic cancer, based on seven observed and 3.5 expected deaths. The latter deaths were not limited to any particular subtype of lymphopoietic cancer, and it is likely that the increase was due to chance or to confounding by an unidentified factor.

The occurrence of two deaths from mesothelioma is difficult to interpret. Of the two men with mesothelioma, one worked at the talc facility for 15 years, had a relatively high cumulative exposure and died 15 years after starting work at the talc facility. His previous employment history (obtained by querying next-of-kin) included 16 years as a carpenter and millwright, eight years as a lead miner and five years as a repairman in a milk plant. The other decedent with mesothelioma worked only briefly at the facility as a draftsman during mill construction in 1948-1949. His job would have entailed minimal exposure to talc dust. He previously had worked for several years on the construction of another talc mine, and he subsequently installed and repaired oil burning heating systems and delivered fuel oil. Although medical records that we obtained for this subject reported no history of asbestos exposure, he may have been exposed from the insulating materials in his fuel oil business. Experimental animal studies of the talc ore of the study facility have not observed pleural tumors [Stanton et al., 1981]. For this reason, and because of the short amount of time between first exposure and death of the first case and the low exposure of the second case, it is unlikely that either of the two mesotheliomas is due to talc ore dust.

Compared to previous investigations of the same workers, the present study had several advantages. These included longer follow-up, larger size, analyses by work area and estimated cumulative exposure to respirable dust, comparisons of subjects' mortality rates with regional general population rates and use of an internal referent group in some analyses.

Limitations of our study included the exclusion from analyses of work areas and cumulative dust exposure of a small proportion (6%) of subjects because work histories were unavailable. Most of these subjects were short-term workers whose cumulative exposure would have been low. Potential misclassification of subjects by cumulative exposure was inherent in the exposure estimation approach used for the study [Oestenstad et al., submitted]. However, because we developed the work area/time period exposure estimates using procedures that did not involve any reference to disease outcome, misclassification errors should have been nondifferential, blunting any true dose-response relations. Another limitation of exposure estimation was our lack of information on subjects' peak exposure intensities and exposure to respirable fibers, either of which might be more biologically relevant than cumulative exposure.

We also lacked comprehensive information on potential confounders such as cigarette smoking and other occupational exposures. Because data on the smoking habits of subjects were unavailable, we cannot rule out the possibility that the lung cancer and NMRD patterns observed were due, at least in part, to heavier and/or more prevalent smoking by the subjects than by the comparison population. Similarly, work in other mining operations and construction jobs may have contributed to the respiratory disease mortality patterns observed.

Some misclassification of NMRD, resulting both from difficulties with the clinical diagnosis of various respiratory diseases and with the possible overlap between NMRD and cardiovascular disease, may have occurred. In addition, NMRD that was present at death may not be mentioned on the death certificate. If the tendency to list NMRD as a cause of death is greater for talc worker decedents than for decedents in the general population who died with the same set of medical conditions, observation bias would elevate SMRs for NMRD.

In summary, the reason for the increased lung cancer mortality among plant workers compared to the general population remains unclear. The association may be due, in part, to confounding by smoking and by other unidentified risk factors. It is unlikely to be related to respirable talc ore dust per se. An unidentified constituent of the ore or of the underground mine environment, exposure to which is poorly correlated with total respirable dust exposure, may have been responsible for some of the excess lung cancer. We have no information, apart from the disease patterns seen in this study, to substantiate or refute this speculation. The study found an increased rate of NMRD among workers that probably is related to exposure to the talc ore dust at the facility, as well as to dust exposures encountered in other work environments and to smoking. Other causes of death among the plant workers did not appear to be related to the occupational factors.

REFERENCES

1. Blair A, Stewart WF, Stewart PA, Sandler DP, Axelson O, Vineis P, Checkoway H, Savitz D, Pearce N, Rice C. 1995. A philosophy for dealing with hypothesized uncontrolled confounding in epidemiological investigations. Med Lav 86:106-110.
2. Brown DP, Kaplan SD, Zumwalde RD, Kaplowitz M, Archer VE. 1986. A retrospective study of underground gold mine workers. In: Goldsmith DF, Winn DM, Shy CM, editors. Silica, silicosis and cancer: controversy in occupational medicine. Philadelphia: Praeger Press, p 335-350.
3. Brown DP, Sanderson W, Fine LJ. 1990. NIOSH health hazard evaluation report no. 90-390-2065.
4. Brown DP, Wagoner JK. 1978. Occupational exposure to talc containing asbestos: III. Retrospective cohort study of mortality. U.S. DHEW (NIOSH) Pub. 80-115:23-33.
5. Callas PW, Pastides H, Hosmer DW. 1990. Empirical comparisons of proportional hazards, Poisson, and logistic regression modeling of occupational cohort data. Am J Ind Med 33:33-47.
6. Cooper WC, Wong O, Trent LS, Harris F. 1992. An updated study of taconite miners and millers exposed to silica and non-asbestiform amphiboles. J Occup Med 34:1173-1180.
7. Davis JMG, Addison J, McIntosh G, Miller G, Niven K. 1991. Variations in the carcinogenicity of tremolite dust samples of differing morphology. In: Landrigan PJ, Kazemi H, editors. The third wave of asbestos disease. New York: The New York Academy of Sciences, 643:473-490.
8. Gamble JF. 1993. A nested case-control study of lung cancer among New York talc workers. Int Arch Occup Environ Health 64:449-456.

9. Goodman M, Morgan RW, Ray R, Malloy CD, Zhao K. 1999. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. Cancer Causes Control 10:453-465.

10. Higgins ITT, Glassman JH, Ohs MS, Cornell RG. 1983. Mortality of Reserve Mining Company employees in relation to taconite dust exposure. Am J Epidemiol 118:710-719.

11. Kelse JW, Thompson CS. 1985. The regulatory and mineralogical definitions of asbestos and their impact on amphibole dust analysis. Am Ind Hyg Assoc J 50:613-622.

12. Lamm SH, Levine MS, Starr JA, Tirey SL. 1988. Analysis of excess lung cancer risk in short-term employees. Am J Epidemiol 127:1202-1209.

13. Marsh G, Youk AO, Stone RA, Sefcik S, Alcorn C. 1998. OCMAP-Plus: A program for the comprehensive analysis of occupational cohort data. J Occup Environ Med 40:351-362.

14. McConnell EE, Rutter HA, Ulland BM, Moore JA. 1983. Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. Environ Health Perspect 53:27-44.

15. McDonald JC, Gibbs GW, Liddell FDK, McDonald AD. 1978. Mortality after long exposure to cummingtonite-grunerite. Am Rev Respir Dis 118:271-277.

16. Oestenstad K, Honda Y, Delzell E, Brill I. Assessment of historical exposures to talc at a mining and milling facility. Am J Ind Med Submitted, 2001.

17. Pott F, Huth F, Friedrichs KF. 1974. Tumorigenic effect of fibrous dusts in experimental animals. Environ Health Perspect 9:313-315.

18. Rubino GF, Scansetti G, Piolatto G, Romano C. 1976. Mortality study of talc miners and millers. J Occup Med 18:186-193.

19. Rubino GF, Scansetti G, Piolatto G, Gay G. 1979. Mortality and morbidity among talc miners and millers in Italy. In: Lemen R, Dement JM, editors. Dust and disease. Park Forest South, IL: Pathotox Publishers, p 379-388.

20. Seidman H, Selikoff IJ, Gelb SK. 1986. Mortality experience of amosite asbestos factory workers: dose-response relationship 5 to 40 years after onset and short-term work exposure. Am J Ind Med 10:479-514.

21. Smith WE, Hubert DD, Sobel HJ, Marquet E. 1979. Biologic tests of tremolite in hamsters. In: Lemen R, Dement JM, editors. Dust and disease. Park Forest South, IL: Pathotox Publishers, p 335-339.

22. Stanton MF, Layard M, Tegeris A, Miller E, May M, Morgan E, Smith A. 1981. Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. J Natl Cancer Inst 67:965-975.

23. Stille WT, Tabershaw IR. 1982. The mortality experience of upstate New York talc workers. J Occup Med 24:480-484.

24. Straif K, Keil U, Taeger D, Holthenrich D, Sun Y, Bungers M, Weiland SK. 2000. Exposure to nitrosamines, carbon black, asbestos, and talc and mortality from stomach, lung, and laryngeal cancer in a cohort of rubber workers. Am J Epidemiol 152:297-306.

25. U. S. Bureau of the Census. 1972. County and city data book, 1972. Washington, DC: U. S. Government Printing Office.

26. U. S. Department of Labor. 2000. Background information regarding the analysis of industrial talcs. Salt Lake City, UT: Occupational Safety and Health Administration Salt Lake Technical Center.

27. Wagner JC, Berry G. 1969. Mesothelioma in rats following inoculation with asbestos. Br J Cancer 23:567-581.

28. Wergelund E, Anderson A, Baerheim A. 1990. Morbidity and mortality in talc-exposed workers. Am J Ind Med 17:505-13.

29. Wylie AG, Bailey KF, Kelse JW, Lee RJ. 1993. The importance of width in asbestos fiber carcinogenicity and its implications for public policy. Am Ind Hyg Aoosc J 54:239-252.

30. Wylie AG, Virta RL, Russek E. 1985. Characterizing and discriminating airborne amphibole cleavage fragments and amosite fibers: implications for the NIOSH method. Am Ind Hyg Assoc J 46:197-201.

31. Wylie AG, Skinner HCW, Marsh J, Snyder H, Garzione C, Hodkinson D, Winters R, Mossman BT. 1997. Mineralogical features associated with cytotoxic and proliferative effects of fibrous talc and asbestos on rodent tracheal epithelial and pleural mesothelial cells. Toxicol Appl Pharmacol 147:143-150.

TABLE I. Observed and expected numbers of deaths, SMR and 95% CI by cause of death, 1960-1989, 782 subjects, 15,050 person-years

	Obs/Exp	SMR	95% CI
All causes	209/160	131	114-150
Tuberculosis	3/0.4	788	163-2303
All cancer	53/35	151	113-198
Cerebrovascular disease	6/8.8	68	25-148
Rheumatic heart disease	2/1.1	175	21-633
Ischemic heart disease	69/63	110	86-139
Hypertension	2/0.9	224	27-807
Other heart disease	5/4.6	108	35-252
Nonmalignant respiratory disease (NMRD)	28/13	221	147-320
Influenza & pneumonia	7/3.8	185	74-381
Bronchitis, emphysema, asthma	4/3.3	122	33-312
Other NMRD*	17/5.7	297	173-475
Cirrhosis of liver	5/3.7	137	44-319
External causes of death	16/15	104	60-169
Other known causes	16/18	89	51-144
Unknown causes	4		

* Observed number includes chronic obstructive pulmonary disease (N=10) and pulmonary fibrosis (N=7).

TABLE II. Observed and expected numbers of cancer deaths, SMR and 95% CI, 1950-1989, 809 subjects, 18,048 person-years

	Obs/Exp	SMR	95% CI
All cancer	54/37	146	110-191
Digestive organs & peritoneum	10/9.9	102	49-187
Stomach	2/1.4	144	17-520
Colon & rectum	2/4.8	42	5-150
Respiratory system	34/14	239	165-334
Larynx	2/0.6	316	38-1142
Lung	31/13	232	157-329
Lymphatic & hematopoietic tissue	7/3.7	192	77-395
Other cancer	3/9.3	32	7-95

TABLE III. Observed and expected numbers of deaths, SMR and 95% CI for selected causes of death by year of hire, 1960-1989 for causes other than cancer, 1950-1989 for cancer

	Hired < 1955			Hired 8 1955		
	Obs/Exp	SMR	95% CI	Obs/Exp	SMR	95% CI
All causes	169/113	150	128-174	40/47	85	61-116
All cancer	46/27	172	126-229	8/10	79	34-155
Cancer of the lung	28/9.8	286	190-414	3/3.6	83	17-242
Ischemic heart disease	56/46	122	92-158	13/17	77	41-132
Nonmalignant respiratory disease (NMRD)	23/9.5	242	153-363	5/3.1	160	52-373
Other NMRD	15/4.2	355	199-586	2/1.5	133	16-479

TABLE IV. Observed and expected numbers of deaths, SMR, RR* and 95% CI for selected causes of deaths by years since hire and years worked

Years since hire	Years worked		Lung cancer	Ischemic heart disease	Nonmalignant respiratory disease (NMRD)	Other NMRD
< 20	< 5	Obs/Exp	3/2.4	11/13.2	5/2.2	2/0.7
		SMR (CI)	126 (26-370)	83 (42-149)	224 (73-524)	294 (36-1062)
		RR (CI)	1.0	1.0	1.0	1.0
< 20	5+	Obs/Exp	2/1.6	9/8.8	2/1.4	2/0.5
		SMR (CI)	126 (15-454)	103 (47-195)	140 (17-506)	417 (51-1507)
		RR (CI)	1.0 (0.2-5.9)	1.3 (0.5-3.1)	0.6 (0.1-3.1)	1.4 (0.2-10.2)
20+	<5	Obs/Exp	19/5.8	30/23.6	11/5.0	7/2.6
		SMR (CI)	331 (199-516)	127 (86-181)	222 (111-397)	271 (109-558)
		RR (CI)	3.0 (0.7-11.8)	2.0 (0.9-4.5)	1.2 (0.3-4.1)	1.4 (0.2-8.8)
20+	5+	Obs/Exp	7/3.7	19/17.2	10/4.0	6/2.0
		SMR (CI)	190 (76-392)	111 (67-173)	248 (119-456)	302 (111-657)
		RR (CI)	1.8 (0.4-8.1)	2.0 (0.8-4.7)	1.6 (0.4-5.9)	1.8 (0.3-11.8)

* RR for each category of years since hire and years worked adjusting for age (<35, 35-54, 55-64, 865) and calendar year (1960-1969, 1970-1979, 1980-1989).

Referent category for the RR for each category of years since hire and years worked computed using the subgroup with < 20 years since hire and < 5 years worked as the referent.

TABLE V. Observed and expected numbers of deaths, SMR, RR and 95% CI for selected causes of deaths by non-mutually exclusive work area, 1960-1989

Work area (N of subjects)		Lung cancer	Ischemic heart disease	Nonmalignant respiratory disease (NMRD)	Other NMRD
Mills (N=379)	Obs/Exp	7/5.5	31/26	11/4.8	6/2.3
	SMR (CI)	128 (51-263)	122 (83-173)	227 (113-407)	266 (98-579)
	RR* (CI)	0.6 (0.2-1.8)	1.2 (0.6-2.3)	0.9 (0.3-2.5)	1.2 (0.3-4.4)
Mines (N=311)	Obs/Exp	18/4.6	23/22	10/4.2	8/1.8
	SMR (CI)	394 (233-622)	104 (66-156)	241 (116-444)	434 (189-856)
	RR (CI)	2.1 (0.8-5.5)	1.1 (0.6-2.1)	1.0 (0.4-2.7)	2.0 (0.6-7.2)
Minimal exposure (N=182)	Obs/Exp	3/3.9	13/22	9/4.6	5/2.0
	SMR (CI)	77 (15-224)	60 (32-102)	194 (89-369)	255 (83-596)
No exposure (N=87)	Obs/Exp	3/1.1	4/6.6	2/1.3	1/0.5
	SMR (CI)	281 (58-821)	60 (16-154)	158 (19-572)	217 (5-1207)
Unknown area (N=67)	Obs/Exp	2/1.3	7/6.4	3/1.2	3/0.6
	SMR (CI)	151 (18-547)	109 (44-225)	242 (50-708)	546 (113-1597)

* RR for mill workers compared to all other employees, adjusting for age, calendar period and employment in mines.
RR for mine workers compared to all other employees, adjusting for age, calendar period and employment in mills.

TABLE VI. Median estimated cumulative exposure to respirable dust among selected subgroups of 772 subjects with adequate work history data

Group (median years worked)	Number	% exposed	Median cumulative exposure among exposed (mg/m^3-days)
All subjects (2.6)	772	95	425
Mill workers, ever (3.0)	389	100	683
Mine workers, ever (3.6)	331	100	739
All decedents (2.3)	213	94	520
Ischemic heart disease (2.4)	70	94	376
Lung cancer decedents (1.0)	29	90	347
All nonmalignant respiratory disease (NMRD) decedents* (2.3)	27	96	888
Other NMRD decedents (8.3)	30	100	1,199
Pulmonary fibrosis decedents (11.8)	17	100	3,759

* Nonmalignant respiratory disease as the underlying cause of death.
Nonmalignant respiratory disease as the underlying or a contributing cause of death.

Table VII. RR and 95% CI for selected causes of death by tertile* of cumulative respirable dust exposure, adjusted for age and years since hire

Cause of death and cumulative exposure (mg/m^3-days)	Deaths	Person-years	RR	95% CI
Lung cancer				
0-<95.1	11	2,625	1.0	-‡
95.1-<987.0	9	2,660	0.8	0.3-1.9
987.0+	9	3,796	0.5	0.2-1.3
			df=12, D=11.6	
Ischemic heart disease				
0-<131.8	25	3,931	1.0	-
131.8-<2456.8	23	5,083	0.7	0.4-1.2
2456.8+	22	2,927	1.0	0.6-1.8
			df=19, D=17.7	
All nonmalignant respiratory disease (NMRD) (underlying)				
0-<488.8	9	6,023	1.0	-
488.8-<2554.7	9	3,058	2.2	0.9-5.6
2554.7+	9	2,860	1.6	0.6-4.1
			df=19, D=14.0	
Other NMRD (underlying/contributory)				
0-<519.7	10	6,124	1.0	-
519.7-<4110.5	10	3,948	1.8	0.8-4.4
4110.5+	10	1,869	2.1	0.9-5.1
			df=19, D=17.5	
Pulmonary fibrosis (underlying/contributory)				
0-<863.3	5	6,990	1.0	-
863.3-<7529.6	6	4,437	2.2	0.7-7.4
7529.6+	6	514	11.8	3.1-44.9
			df==16, D=14.0	

* Tertiles are based on the cumulative exposure distribution of cases in the cause of death category. Age categories were 35-54, 55-64, 65+ years for all disease groups evaluated. Years since hire categories were <10, 10-19 and 20+ for ischemic heart disease, all NMRD, other NMRD and pulmonary fibrosis and were 10-19 and 20+ for lung cancer.

‡ Dash indicates referent category for the RR.

df, model degrees of freedom; D, model deviance.

UAB SCHOOL OF PUBLIC HEALTH

Department of Epidemiology

March 30, 2001

Philip J. Landrigan, M.D., Editor
American Journal of Epidemiology
Post Office Box 1057
Mount Sinai School of Medicine
New York, New York 10029-6574

Dear Dr. Landrigan:

Enclosed are three copies each of two manuscripts, "Mortality among Workers at a Talc Mining and Milling Facility" and "Assessment of Historical Exposures to Talc at a Mining and Milling Facility" that we hope you will consider for publication. Although the mortality experience of these workers has been evaluated previously, the study described in the papers extends the follow-up period and incorporates several methodological improvements over prior studies. Improvements include the evaluation of mortality patterns by duration of employment and time since hire, the estimation of workers' cumulative exposure to respirable talc dust and the use of internal analyses to evaluate the relation between cumulative exposure to respirable dust and selected causes of death. We think these papers add important information to the ongoing deliberations about the classification of talc as a human carcinogen.

Thank you for considering the enclosed papers. They are original works that have not been published or submitted for publication elsewhere in any part or form. If you have any questions about this study, please call me, Dr. Honda or Dr. Oestenstad any time. If the question involves a technical, rather than a substantive, issue, I am probably easier to reach than Dr. Honda. I can be reached by telephone at 205-934-7164 or by email at colleen@uab.edu.

Sincerely,

[redacted]

Colleen Beall, DrPH
Assistant Professor

Enclosures/

The University of Alabama at Birmingham
220A Ryals Building • 1665 University Boulevard
Birmingham, Alabama 35294-0022 • (205) 934-1200 • FAX (205) 975-7058

ASSESSMENT OF HISTORICAL EXPOSURES TO
TALC AT A MINING AND MILLING FACILITY

Kent Oestenstad, Ph.D. [1]

Yasushi Honda, M.D.[2]

Elizabeth Delzell, S.D. [3]

Ilene Brill, M.P.H. [3]

Short Running Title: HISTORICAL TALC EXPOSURES

Text word count: 4073

[1] Department of Environmental Health Sciences, University of Alabama at Birmingham School of Public Health

[2] Environmental Health Sciences Program, Institute of Health and Sport Sciences , University of Tsukuba, Japan

[3] Department of Epidemiology and International Health, University of Alabama at Birmingham School of Public Health

Address correspondence to: Kent Oestenstad
Department of Environmental Health Sciences
School of Public Health
University of Alabama at Birmingham
RPHB 120, 1530 3rd Ave. S.
Birmingham, AL 35294-0022
Phone: 205-934-6208
Fax: 205-975-5484
e-mail: oestensk@uab.edu

ABSTRACT

The purpose of this study was to develop an estimate of exposure to respirable dust for all job categories and all years in a retrospective follow-up study of worker mortality in a talc mining and milling facility. All jobs were assigned to work areas that were considered to have similar exposure profiles. Uniform exposure time periods during which non-random, deterministic variables were thought to be constant were identified, and an experienced rater assigned categorical exposure scores to each work area/time period. These scores and measured baseline respirable dust concentrations were used to calculate the estimated job area/year concentrations for each work area/time period. Estimates were validated by comparison to available historical measurements. The estimated exposures ranged from 1.7 mg/m^3 to 0.1 mg/m^3, and displayed a decreasing trend over time. When compared to measured exposures, the estimated exposures had a correlation coefficient of 0.73 with an average bias of –0.01 mg/m^3, and a range of 0.17 to –0.13 mg/m^3. The estimates were considered to be acceptable for determining relative ranking of subjects according to cumulative exposure.

Keywords: TALC, OCCUPATIONAL EXPOSURE, EXPOSURE ESTIMATION

BACKGROUND

The purpose of this investigation was to develop a job-exposure matrix for respirable dust, covering all work areas in an industrial-grade (tremolitic) talc mining and milling facility in upstate New York (NY) as part of a retrospective follow-up study of mortality among workers at this facility [Honda et al., 2001]. The facility started operating in 1948 with the opening of an underground mine (mine 1) and a mill. An open pit mine (mine 2) opened in 1974. Talc from the facility was used predominantly for paint and ceramic tile.

The job-exposure matrix consisted of an estimate of the average respirable dust concentration in each work area and each calendar year from 1948 through 1989. The estimated dust concentrations were derived from exposure scores, ranging from 1 (low) to 10 (high), for each work area and year and from reference dust concentrations measured in surveys in 1991 to determine average concentrations under current operating conditions. The matrix was linked with employees work histories to estimate the cumulative exposure to respirable dust of each subject in the companion retrospective follow-up study [Honda et al., 2001].

METHODS

Overview of exposure estimation procedures

As with most retrospective follow-up studies, the utility of historical exposure data that were available for the retrospective follow-up study was limited [Stewart et al., 1996]. A total of 1322 exposure measurements over the 38 years of the study were identified. The utility of these data was limited because a variety of methods was used over the study period to measure exposures to talc. The methods included dust counts (n=428), fiber counts (n=442), respirable dust measurements (n=206), and total dust measurements (n=246). There were questions about the validity of much of the data because of uncertainty regarding the source, type or location of the samples [Stewart, 1999]. Also, measurements were not available for many of the work area/year combinations in the study period.

Because of the sparseness and uncertain quality of the historical data, cumulative respirable dust exposure estimation for individual subjects could not be based exclusively on existing dust measurements. Rather, a job-exposure matrix consisting of estimates of respirable dust concentrations for various work area and calendar year combinations was developed. The process of developing this matrix included: 1) specifying work areas and associated jobs, 2) defining time periods during which exposure levels could be considered uniform within the work areas, and assigning an exposure score to each work area/time period, 3) conducting baseline surveys to determine current exposure conditions in the work areas and to develop a factor to convert historical dust count data to respirable dust concentrations, 4) estimating historical respirable dust concentrations for each work area/calendar year category, and 5) validating the job-exposure matrix estimates by comparing available historical measurements with estimated concentrations. Respirable talc dust, rather than dust count, data were used as the basis of cumulative exposure estimates because of the better precision of respirable mass sampling and analytical methods compared to the other sampling methods employed [Ayer, 1969], and the necessity of pooling historical data collected by several agencies [Seixas et al., 1990; Ulfvarson, 1983]. In addition, respirable mass is a more appropriate measure of exposure because of the relevance of physiological region of respiratory deposition of that size fraction to occupational respiratory diseases, and to have a comparable measure to the TLV for talc.

Work area specification

A previous report by the National Institute of Occupational Safety and Health (NIOSH) further describes most of the operations and jobs at the study facility [Dement et al.,1980]. Each job title included in the work histories of employees was assigned to a work area, defined as a group of jobs having a similar exposure profile based on similar processes, tasks, engineering controls (e.g., ventilation), and exposures to airborne talc [Corn et al., 1979; Stewart et al., 1996]. The areas and each area's component jobs were specified by the one of the authors (KO), a certified industrial hygienist, by observing current operations, consulting long-term supervisory personnel familiar with operating conditions at the facility and reviewing historical exposure measurements [Dement et al.,1980]. Jobs comprising a given work area were assumed to be reasonably homogeneous with respect to exposure within the specified time periods.

Table I shows the final 13 work areas and typical job activities within each area. The "mill average" work area consists of laborers who worked in unspecified areas within the mills, the "no exposure" work area consists of office employees and outside laborers, and the "unknown" work area was assigned to work history entries for which activities were not specified and for which exposure estimates could not be developed.

Specification of uniform exposure time periods

Work area-specific uniform exposure time periods were defined as calendar periods during which non-random, deterministic variables, such as operating processes and control technology, were constant, and during which the average exposure level probably did not change over time [Yu RC et al., 1990]. These periods were specified by a panel of three knowledgeable supervisory personnel (the "expert panel") assembled for the previous NIOSH study, using production records, dust control information and past environmental reports. The expert panel included three salaried, long-term employees: Rater 1, hired in 1953 and familiar with both the mines and the mills; Rater 2, hired in 1948 and familiar with the mills; and Rater 3, hired in 1971 and familiar with the mines. One of us (KO) reviewed the periods identified by the panel and judged their specifications to be adequate.

Development of exposure scores

The expert panel and five additional employees were asked to assign an exposure score, ranging from 0 for no exposure, to 10 for highest exposure, within each time period to the most commonly held jobs comprising a given work area. The additional employees included: Rater 4, hired in 1951 and familiar with the mills; Rater 5, hired in 1954 and familiar with the mills; Rater 6, hired in 1957 and familiar with the mills; Rater 7, hired in 1950 and familiar with the mines; and Rater 8, hired in 1959 and familiar with the mines. Raters 4 through 8 had all been hourly paid employees.

Rater 1 assigned scores to all jobs and years. Because of their different hire dates and different work experiences, all the other raters assigned scores only to selected jobs within selected work areas and locations (e.g., mill, mine 1, mine 2) and only for selected years. Rater 6 provided extremely incomplete information, and his scores were discarded.

Several types of average scores were developed. First, all job/year-specific exposure scores were averaged over jobs comprising a particular work area to obtain a "work area/year" score. Second, work area/year-specific scores were averaged over all work areas within a location and over all years within a time period to obtain a mean "location/time period score." Time periods were specified on the basis of the number of raters providing scores. For example, in some time periods, only two raters provided scores for a given location, whereas in other time periods three or four raters provided scores. Finally, we computed an "adjusted" summary scores for each work area/year category, averaging across all raters who provided relevant information for the specific category and adjusting for the constant difference among raters' scores. To obtain the average "adjusted" scores, the scores of Rater 1 were designated as the "standard" set of scores. Rater 1 was chosen as the standard because he had extensive experience in both the mines and the mills and was judged to be the most knowledgeable of all the raters. Next, we computed for each of the other raters an adjusted score for a given location/work area/year combination as his actual score, minus the difference between his and the standard rater's mean location/time period-specific scores. The average adjusted score for each work area/year-specific category was determined by summing the adjusted scores of all raters contributing data to that category and dividing by the number of raters. Two sets of exposure estimates were developed, one based only on the scores of Rater 1, and the other based on the average adjusted scores of all raters.

Baseline exposure surveys

Two one-week exposure surveys were conducted by one of us (KO) to measure current respirable dust concentrations during warm- (July) and cold-weather (December) months and to develop a factor to convert historical dust count data to respirable dust concentrations. Personal air samples were collected and analyzed to determine eight-hour, time-weighted average respirable dust concentrations according to NIOSH Analytical Method 0600 - Nuisance Dust, Respirable [National Institute for Occupational Safety and Health, 1984]. Impinger samples for dust counts were collected and analyzed according to the US Public Health Service Impinger Sampling Technique [Roach, 1973]. Area samples for respirable dust were collected using a high volume cyclone [Hering, 1989]. These samples were also analyzed by NIOSH Method 0600 [National Institute for Occupational Safety and Health, 1984]. The use of the high volume cyclone allowed identical sampling times for the impinger and respirable dust samples.

The arithmetic means of baseline survey and NIOSH respirable dust concentrations were used to define baseline exposures in each area, and consequently, to calculate estimated exposures for the job-exposure matrix. These values were used because the accumulated uptake of the contaminant by the human body is proportional to the arithmetic mean of the period under observation [Skinner et al., 1988; Ulfvarson, 1983]. The value for mill 1-average was the arithmetic mean of all observations in work areas 2 through 5. The value for the minimal exposure group was taken as the exponent of the 5^{th} percentile of natural logarithms of exposures used to define baseline values for all work areas. In some work areas, the baseline exposures are based on very few samples ($n \leq 4$), so the true average could be within a relatively large range.

Coincident respirable dust and dust count samples were used to generate a factor for converting historical dust counts to respirable dust concentrations. When available, paired respirable dust and dust count data collected during the NIOSH survey of 1975 [Dement et al.,1980] were included in the data set used to develop the conversion factor. A weighted regression equation of the natural logarithms of the respirable dust and the dust count concentrations was used to convert historical dust count data to respirable dust concentrations. Based on corresponding sample descriptive information, the historical dust concentration data were classified into the previously described work area/year matrix. The average of the historical measurements was then calculated from the data available for each work area/year category for use in the validation procedure.

Estimation of work area/time period-specific dust levels

Quantitative dust concentration estimates were developed for each work area-time period combination as follows. First, the "baseline" arithmetic mean respirable dust concentration for each work area was derived from data collected in the two exposure surveys conducted by us and from data collected in the NIOSH survey [Dement et al.,1980]. Baseline concentrations were intended to represent exposure conditions in 1985-1989. The NIOSH data were included in calculating the mean baseline concentration of a work area if there was not a significant difference between the data collected in this study and the NIOSH survey. Based on this criterion, the NIOSH data were included in the baseline mean concentration estimates for three work areas. The purpose of incorporating the NIOSH data was to reduce the confidence interval for the estimate of the mean concentration.

Next, for each time period, the estimated average respirable dust concentration for the work area was computed as the product of the baseline mean concentration and the ratio of the time period-specific exposure score to the baseline exposure score. This computation is illustrated in the following conceptual equation:

$$\text{Estimated Dust Conc} = \text{Baseline Dust Conc X } \frac{\text{Time Period - Specific Exposure Score}}{\text{Baseline Exposure Score}}$$

Validation of exposure estimation procedures

Exposure estimation was validated by comparing work area/year-specific exposure estimates with the mean of the historical dust measurements, available for selected work areas and years. The use of the latter data was complicated by the fact that dust samples were collected by several agencies, including the employer, environmental consultants and/or insurance carriers, state and Federal safety and health regulatory agencies, and NIOSH using diverse methods. The use of pooled data collected by different agencies could produce information bias [Ulfvarson, 1983; Olsen et al., 1991]. Particularly, regulatory agencies tend to overestimate the average dust level by conducting compliance or "worst case" sampling [Seixas et al., 1990]. This bias may also be present in data collected by insurance carriers, and even in some data collected by company hygienists. Also, the precision of the historical data was limited because most of the data were converted from dust counts to respirable mass concentrations by a regression equation with a moderate coefficient of determination.

RESULTS

Exposure scores

Tables II and III display the exposure scores of Rater 1 and the adjusted exposure scores based on all seven raters' judgments, respectively. Differences between the two sets of scores tended to be small, and only those based on Rater 1 are considered in the remainder of this report. The scores indicated a gradual decrease in exposure over the study period in all work areas.

Baseline dust concentrations

The range of all respirable dust concentrations observed in the two baseline exposure surveys was from 0.01 to 2.67 milligrams per cubic meter (mg/m^3), with an arithmetic mean of 0.47 mg/m^3, a geometric mean of 0.28 mg/m^3, and a geometric standard deviation of 3.05. The geometric mean of the measurements made during the summer survey was 0.59 mg/m^3, whereas that for the winter survey was 0.41 mg/m^3. These values were not significantly different. Therefore, no adjustment was made for seasonal differences in the subsequent data analysis or exposure estimation.

The work area arithmetic and geometric mean baseline dust concentrations developed from the baseline survey data and, for work areas 4 through 6, from combined baseline survey and NIOSH data are shown in Table IV. The use of baseline survey data and NIOSH surveys for work areas 4 through 6 indicates that exposure levels in these areas did not significantly change between 1975 and 1991. Exposure levels were relatively high in mine 2-crushing (0.83 mg/m^3) and in mine 1-underground (0.73 mg/m^3); intermediate in mill 1 (0.35-0.53 mg/m^3) and mine 2-equipment operating (0.22 mg/m^3); and low in all other areas (0.06-0.14 mg/m^3).

Conversion of dust counts

Historical dust counts were converted to respirable mass concentrations by linear regression analysis of 50 paired dust count and respirable mass samples. Previous studies have reported an average ratio for this type of conversion [Ayer, 1969; Jacobson and Tomb, 1967]. However, the set of ratios in this study was found to be log-normally distributed, so a regression equation using the natural logarithms of measured dust counts and respirable mass concentrations was thought to be a more appropriate method of conversion. The weighted regression equation, shown below, yielded a correlation coefficient of 0.78:

$$\ln(\text{mg/m}^3) = \ln(\text{mppcf}) * 0.3255 - 0.8529$$

where mppcf = million particles per cubic foot.

The natural logarithms of the 50 coincident dust count and respirable mass samples with the regression line are shown in Figure 1.

Work area/year-specific dust concentration estimates

Table V presents work area/calendar year-specific estimates of average respirable dust concentrations, computed from the exposure scores of Rater 1 (Table II) and the baseline respirable dust concentrations (Table IV). Exposure concentrations were estimated to be slightly higher in milling than in underground mining until the early 1970s, and were estimated to be similar in the two locations or slightly higher in underground mining than in milling thereafter.

Validation of historical dust concentration estimates

The years and work areas (n=45) for which measured and converted, historical respirable dust measurements were available and the corresponding predicted exposures from the estimation procedure described above are shown in Table VI. The data also are plotted in Figure

2. The correlation coefficient for the relation between the measured and the predicted concentrations was 0.73. The average bias for the predicted concentrations was -0.01 mg/m^3 [Seixas et al., 1990]: that is, on average, the predicted concentrations were 0.01 mg/m^3 higher than the historical measured concentrations. Bias within the work areas ranged from 0.17 mg/m^3 in mine 1-surface crushing to -0.32 mg/m^3 in mine 2-crushing.

DISCUSSION

A job-exposure matrix based on work area and time period was developed to estimate historical quantitative respirable dust exposures of employees at the talc mining and milling facility under study. The procedures used measured baseline work area-specific exposure concentrations along with categorical exposure scores assigned by a qualified rater to estimate year-specific respirable dust exposure concentrations for the years 1948 through 1989. This approach was thought to be the most effective method of estimating exposures given the limited quantity and quality of available historic exposure data. This method is more sensitive than ordinal classification of exposures, and it avoids the uncertainties of exposure prediction models [Hornung, 1991].

Initially, it had been proposed to use dust count data as a parallel estimate of exposure to talc dust. However, it was decided to use respirable dust concentrations because these data were considered to be more precise than dust count data [Edwards et al., 1966; Ayer, 1969] and less biased than some of the historical data [Olsen et al., 1991; Ulfvarson, 1983]. The use of baseline and NIOSH respirable dust concentration data reduced imprecision that would have resulted from converting these data to dust counts by a regression equation that had a correlation coefficient of 0.78. The effect of the imprecision of the conversion was limited to those respirable dust concentrations that were converted from historical dust counts and used to validate the exposure estimates.

No attempt was made to use available fiber count data because of inconsistencies between the regulatory and mineralogic definitions of fibers and the mineralogic composition of talc dust at this facility. According to the NIOSH analytical method for asbestos, a fiber is defined as any particle with a length-to-width aspect ratio of at least 3:1 and a length of 5 •m or more observed under phase contrast microscopy [Leidel et al.,1979]. However, this definition has been criticized by mineralogists as being nonspecific for true asbestiform fibers [Skinner et al., 1988]. Kelse and Thompson [1989] have demonstrated that airborne cleavage fragments of nonasbestiform tremolitic talc dust collected at this facility would be incorrectly classified as fibers under the 3:1 aspect ratio rule. This misclassification resulted in an overestimation of fiber counts in air samples collected at that facility.

The development of the job-exposure matrix involved assumptions and uncertainties. However, we refined previously developed job/work area classifications by evaluating the operational characteristics of the work areas and by analyzing respirable dust data obtained during our baseline surveys and from NIOSH and of by analyzing exposure scores assigned to jobs in the areas. We also validated our exposure estimates, making use of historical data available for eight of the 11 work areas where exposure was judged to be more than minimal. Validation was, however, limited to 45 (10.8%) of the 418 cells of the job-exposure matrix because historical measures tended to be clustered in specific years, and in some cases, the number of historical exposure measures in a cell was small.

The observed correlation coefficient of measured and estimated exposures was considered to be good given the following characteristics of the data: 1) the inherent variability of the dust count method [Ayer, 1969; Edwards et al., 1966] 2) the relatively low correlation coefficient for the conversion of dust counts to respirable dust exposures; 3) the use of pooled data collected by several agencies using different methods [Ulfvarson, 1983]; and 4) the use of averages of a small number of observations to represent exposures which are known to exhibit considerable inter- and intra-day variation [Seixas et al., 1988]. Given the above characteristics of the data, an average bias of only -0.01 mg/m^3 was considered to be remarkable. The wide range of average bias among the work areas is probably an indication of the instability of this number. A detailed statistical validation of the predicted exposures was not conducted because of the relatively small number of cells. However, the average bias for each work area is well within a factor of one of the mean value for that area.

The estimated exposures in this study do not take into account other factors that affect the uptake of contaminants. These factors could include: 1) the effective use of respiratory protection, 2) part-time exposures, 3) personnel rotation not recorded in administrative work histories, 4) unfavorable distribution of exposure periods over time, and 5) unusually hard work increasing the ventilation of the exposed individuals. Of these, the use of respiratory protection would be the most likely uptake modifier among these workers. During the baseline surveys it was observed that there was current wide-spread usage of respirators, but it is not known when the use of this equipment was initiated systematically, or how conscientiously and effectively respirators were used.

The highest average estimated exposure was 1.7 mg/m^3, which is below the Threshold Limit Value (TLV) of 2mg/m^3 for nonsabestiform talc [American Conference of Governmental Industrial Hygienists, 2000]. The retrospective follow-up study of workers at the facility under study found an increased rate of nonmalignant respiratory disease [Honda, et al., 2001]. Thus, our results might indicate the TLV for talc is too high to prevent increased risk of these diseases. However, such an interpretation may not be correct because we did not know employees' occupational exposures outside the study facility.

In summary, it is expected that the concentrations in the job-exposure matrix over-estimate the actual exposures experienced by these employees. This is based on a slight average negative bias of estimated exposures when compared to historical data that are thought to represent worst-case conditions [Seixas et al., 1990]. Also, the estimated exposures do not take into account the diminishing effect of respiratory protection. For these reasons, the absolute values of cumulative exposure estimated for subjects in the retrospective follow-up study may not be accurate. However, cumulative exposure estimates based on these values should be useful for obtaining a relative ranking of subjects according to exposure for use in an epidemiological dose-response analysis.

REFERENCES

1. Ayer, HE. 1969. The Proposed ACGIH Mass Limits for Quartz: Review and Evaluation. Am Ind Hyg Assoc J 30:117-125.

2. Corn M, Esman NA. 1979. Workplace exposure zones for classification of employee exposures to physical agents. Am Ind Hyg Assoc J 40:47-57.

3. Dement JM, Zummwalde RD, Gamble JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK. 1980. Occupational exposure to talc containing asbestos: environmental studies of mines and millers. U.S. Department of Health, Education and Welfare (NIOSH) Pub. No. 80-115.

4. Edwards RG, Powell CH, Kendrick MA. 1966. Dust counting variability. Am Ind Hyg Assoc J 27:546-554.

5. Hering SV. 1989. Inertial and gravitational collectors. In: Air sampling instruments for evaluation of atmospheric contaminants. 7th ed. Cohen BS, editor. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, pp 337-386.

6. Honda Y, Beall C, Delzell E, Brill I, Mathews R. A retrospective follow-up study of workers at a talc mining and milling facility. Am J Med Submitted, 2001.

7. Hornung RW. 1991. Statistical evaluation of exposure assessment strategies. Appl Occup Environ Hyg 6:516-520.17.

8. Jacobson M, Tomb TF. 1967. Relationship between gravimetric respirable dust and midget impinger number concentration. Am Ind Hyg Assoc J 28:554-556.

9. Kelse JW, Thompson CS. 1989. The regulatory and mineralogical definitions of asbestos and their impact on amphibole dust analysis. Am Ind Hyg Assoc J 50:613-622.

10. Leidel NA, Bayer SG, Zumwalde RD, Busch KA. 1979. USPHS/NIOSH membrane filter method for evaluating airborne asbestos fibers (Technical Report No. 79-127). Cincinnati, OH: National Institute for Occupational Safety and Health, 15 p.

11. National Institute for Occupational Safety and Health. 1984. NIOSH manual of analytical method 0600: nuisance dust, respirable (DHHS (NIOSH) Publication Number 84-100). 3rd ed. Cincinnati, OH: National Institute for Occupational Safety and Health, p 3.

12. Olsen E, Laursen B, Vinzents PS. 1991. Bias and random errors in historical data of exposure to organic solvents. Am Ind Hyg Assoc J 52:204-211.

13. Roach, SA. 1973. The industrial environment: its evaluation and control (S/N 017-001-00396-4). In: Sampling for Air Particulates. Cincinnati, OH: National Institute for Occupational Safety and Health, pp 139-165.

14. Seixas NS, Robins TG, Moulton LH. 1988. The use of geometric mean and arithmetic mean exposures in occupational epidemiology. Am J Ind Med 14:465-477.

15. Seixas NS, Robins TG, Rice CH, Moulton LH. 1990. Assessment of potential bias in the application of MSHA respirable coal mine dust data to an epidemiologic study. Am Ind Hyg Assoc J 51:534-540.

16. Skinner HCW, Ross M, Frondel C. 1988. Asbestos and other fibrous materials: mineralogy, crystal chemistry and health effects. New York: Oxford University Press, 204 p.

17. Stewart PA, Lees PSJ, Francis M. 1996. Quantification of historical exposures in occupational cohort studies. Scand J Work Environ Health. 22:405-414.

18. Stewart P. 1999. Challenges to retrospective exposure assessment. Scand J Work Environ Health. 25:505-510.

19. Ulfvarson U. 1983. Limitations to the use of employee exposure data on air contaminants in epidemiological studies. Int Arch Occup Environ Health 52:285-300.11.

20. Yu RC, Tan WY, Mathew RM, Andjelkovich DA, Levine RJ. 1990. A deterministic mathematical model for quantitative estimation of historical exposure. Am Ind Hyg Assoc J 51:194-201.

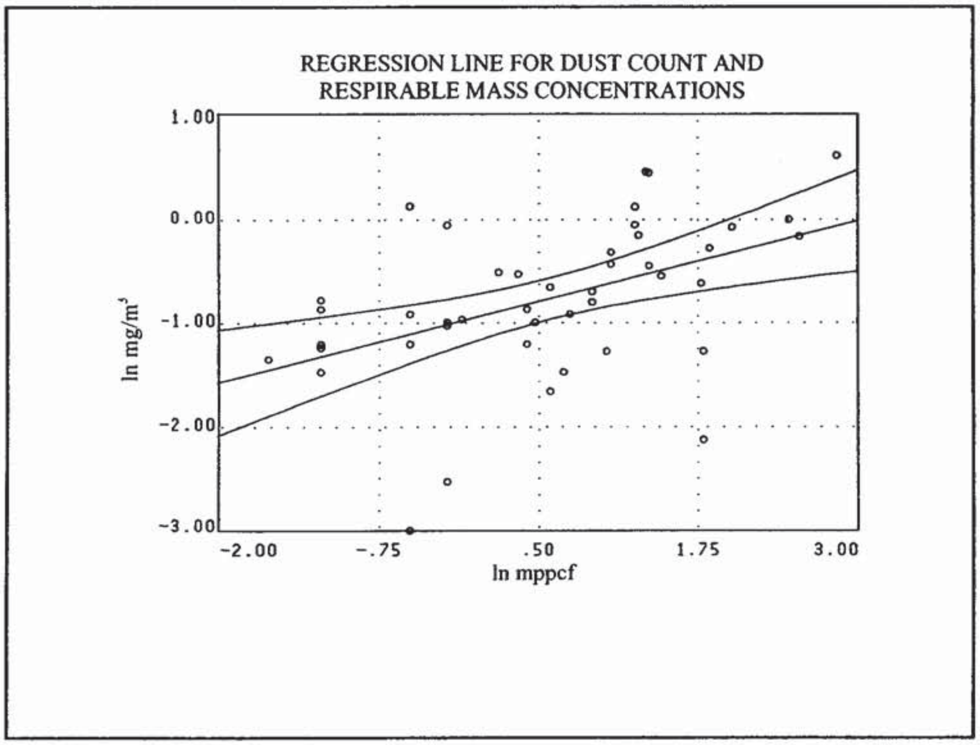

Figure 1 – Regression of Dust Count and Respirable Mass Concentrations

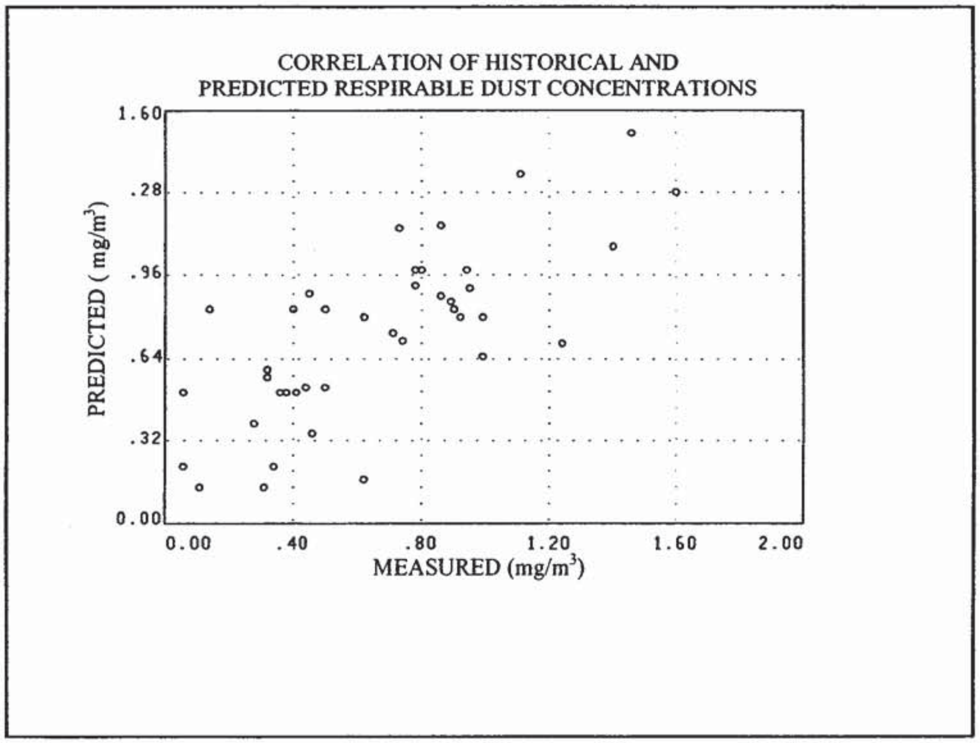

Figure 2 - Correlation of Historical and Measured Respirable Dust Concentrations

Table I. Work Areas and Typical Job Activities

1. Mill – average
    - Mill laborer, unspecified
2. Mill – milling
    - Crusher/dryer operators
    - Wheeler operators
    - Hardinge operators
    - Air process operators
    - Cal process operators
    - Foremen/supervisors/managers
3. Mill - palletizing/packing
    - Packers
    - Palletizers
4. Mill - packhouse support
    - Utility men/pumpmen/laborers
    - Fork lift operators
    - Bulk loaders
    - Foremen/supervisors
    - Car liners
5. Mill – maintenance
    - Millwrights
    - Machinists/oilers
    - Electricians
    - Sheet-metal workers/welders
    - Laborer, maint.
    - Instrument repairmen
6. Mine 1 – Underground
    - Driller helpers
    - Slushers/scrapers
    - Trammers
    - Muckers
    - Eimco operators
    - UG crusher operators
    - Pocket cagemen/hoistmen
    - Repairmen
    - Repairman helpers
    - Mechanic
    - Laborer
    - Mine maintenance
    - Blacksmiths/welders
    - Supervisors
7. Mine 1 - Surface crushing
    - Surface crusher operators
8. Mine 2 - Surface - Equipment operators
    - Truck drivers
    - Loader operators
    - Drillers
    - Tractor operators
9. Mine 2 - Crusher
    - Crusher operators
10. Maintenance
    - Mobile mechanics
    - Maintenance workers
    - Supervisors
11. General - minimal exposure
    - Lab workers
    - Mine managers
    - Construction workers
    - Engineers
    - Janitor
    - Masons
    - Powerline workers
    - Quality control
    - Stock clerks
    - Store keepers
    - Surveyors
    - Warehousemen
    - Watchmen
12. No Exposure
    - Inventory contr. supervisors.
    - Mine 4 workers
    - Purchasing agents
    - Office clerks & managers
    - Laborers, outside

99. Unknown

Table II. - Work Area / Year – Specific Exposure Scores by Rater 1

	Mill					Mine 1		Mine 2			General
Year	1	2	3	4	5	6	7	8	9	10	11
1948	8.5	7.8	10	9.5	6.7	4.4	6.0	-	-	-	4.0
1949	8.5	7.8	10	9.5	6.7	4.4	6.0	-	-	-	4.0
1950	8.5	7.8	10	9.5	6.7	4.4	6.0	-	-	-	4.0
1951	8.5	7.8	10	9.5	6.7	4.4	6.0	-	-	-	4.0
1952	7.2	7.2	8.0	7.5	6.0	4.4	6.0	-	-	-	4.0
1953	7.1	7.0	8.0	7.5	6.0	4.4	6.0	-	-	-	4.0
1954	7.1	7.0	8.0	7.5	6.0	4.4	6.0	-	-	-	4.0
1955	6.8	6.5	8.0	7.5	5.3	4.4	6.0	-	-	-	4.0
1956	6.8	6.5	8.0	7.3	5.3	4.5	6.0	-	-	-	4.0
1957	6.8	6.5	8.0	7.3	5.3	4.5	6.0	-	-	-	4.0
1958	6.8	6.5	8.0	7.3	5.3	4.5	6.0	-	-	-	2.5
1959	6.7	6.0	8.0	7.3	5.3	4.5	6.0	-	-	-	2.5
1960	6.2	5.2	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1961	6.1	4.8	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1962	6.1	4.8	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1963	6.1	4.8	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1964	6.1	4.8	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1965	6.1	4.8	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1966	6.1	4.7	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1967	6.0	4.3	8.0	7.3	4.3	4.5	6.0	-	-	-	2.5
1968	5.1	4.3	6.0	5.7	4.3	3.9	3.0	-	-	-	2.5
1969	4.5	4.3	6.0	4.0	3.7	3.9	3.0	-	-	-	2.0
1970	4.6	4.8	6.0	4.0	3.7	3.9	3.0	-	-	-	2.0
1971	4.6	4.8	6.0	4.0	3.7	3.9	3.0	-	-	-	2.0
1972	4.3	3.8	6.0	4.0	3.3	3.4	3.0	-	-	-	2.0
1973	4.2	3.4	6.0	4.0	3.3	3.4	3.0	-	-	-	2.0
1974	4.2	3.4	6.0	4.0	3.3	3.4	3.0	2.3	4.0	1.5	2.0
1975	4.1	3.2	6.0	4.0	3.3	3.4	3.0	2.3	4.0	1.5	2.0
1976	4.1	3.2	6.0	4.0	3.3	3.3	3.0	2.3	4.0	1.5	2.0
1977	4.1	3.2	6.0	4.0	3.3	3.3	3.0	2.3	4.0	1.5	2.0
1978	3.6	2.8	6.0	2.3	3.3	3.3	3.0	2.0	4.0	1.5	2.0
1979	3.1	2.8	4.0	2.3	3.3	3.3	3.0	2.0	4.0	1.5	2.0
1980	3.0	2.8	4.0	2.3	2.7	3.3	2.0	2.0	2.0	1.5	1.5
1981	2.8	2.3	4.0	2.3	2.3	3.3	2.0	2.0	2.0	1.5	1.5
1982	2.8	2.3	4.0	2.3	2.3	3.3	2.0	1.5	2.0	1.5	1.5
1983	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1984	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1985	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1986	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1987	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1988	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5
1989	2.8	2.3	4.0	2.3	2.3	3.4	2.0	1.5	2.0	1.5	1.5

Table III. - Average Adjusted Work Area / Year – Specific Exposure Scores by All Raters

Year	Mill					Mine 1		Mine 2			General
	1	2	3	4	5	6	7	8	9	10	11
1948	7.8	6.9	10	8.4	5.7	4.0	5.2	-	-	-	3.5
1949	7.8	6.9	10	8.4	5.7	4.0	5.2	-	-	-	3.5
1950	7.8	6.9	10	8.4	5.7	4.0	5.2	-	-	-	3.5
1951	7.6	6.7	10	8.4	5.4	4.0	5.2	-	-	-	3.5
1952	6.9	6.6	9.1	6.9	5.1	4.0	5.2	-	-	-	3.5
1953	6.6	6.5	8.2	6.7	5.1	4.1	5.2	-	-	-	3.5
1954	6.6	6.0	8.6	6.4	5.4	4.1	5.2	-	-	-	3.4
1955	6.4	5.8	8.3	6.4	5.2	4.1	5.2	-	-	-	3.4
1956	6.4	5.8	8.3	6.4	5.2	4.3	5.2	-	-	-	3.4
1957	6.5	5.9	8.3	6.4	5.2	4.3	5.2	-	-	-	3.4
1958	6.1	5.6	7.7	6.1	5.1	4.3	5.2	-	-	-	2.4
1959	6.1	5.5	7.7	6.1	5.1	4.0	6.1	-	-	-	2.0
1960	5.9	5.1	7.7	6.1	4.6	4.0	6.1	-	-	-	2.0
1961	5.8	4.7	7.7	6.1	4.5	3.9	6.1	-	-	-	2.0
1962	5.8	4.7	7.7	6.1	4.5	4.0	6.1	-	-	-	2.0
1963	5.7	4.7	7.7	5.8	4.5	4.0	6.1	-	-	-	2.0
1964	5.6	4.7	7.5	5.6	4.4	3.8	6.1	-	-	-	2.0
1965	5.6	4.7	7.5	5.6	4.4	3.8	6.1	-	-	-	2.0
1966	5.5	4.6	7.5	5.5	4.2	3.8	6.1	-	-	-	2.0
1967	5.3	4.4	7.1	5.5	4.0	3.8	6.1	-	-	-	2.0
1968	5.0	4.3	6.4	5.1	4.0	3.7	5.2	-	-	-	1.9
1969	4.6	4.2	5.9	4.4	3.8	3.7	2.2	-	-	-	1.5
1970	4.6	4.3	5.9	4.4	3.6	3.7	2.2	-	-	-	1.5
1971	4.4	4.1	5.9	4.2	3.5	3.8	2.9	-	-	-	1.2
1972	4.4	3.9	5.9	4.2	3.4	3.6	2.9	-	-	-	1.2
1973	4.2	3.5	5.9	4.1	3.3	3.6	2.9	-	-	-	1.2
1974	4.0	3.5	5.5	3.9	3.1	3.6	2.9	2.5	3.8	1.1	1.1
1975	3.9	3.2	5.5	3.9	3.1	3.6	2.9	2.5	3.8	1.1	1.1
1976	3.8	3.2	5.2	3.8	2.9	3.6	2.9	2.5	3.8	1.1	1.0
1977	3.6	3.1	4.8	3.6	2.8	3.6	2.9	2.5	3.8	1.1	1.0
1978	3.3	2.9	4.3	3.1	2.7	3.4	2.0	2.4	3.8	1.1	1.0
1979	3.0	2.8	3.6	3.0	2.6	3.4	2.0	2.4	2.0	1.1	1.0
1980	2.7	2.6	3.3	2.7	2.3	3.4	1.8	2.4	1.4	1.1	0.9
1981	2.6	2.5	3.3	2.7	2.0	3.3	1.8	2.4	1.4	1.1	0.9
1982	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1983	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1984	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1985	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1986	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1987	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1988	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9
1989	2.6	2.5	3.2	2.7	2.0	3.2	1.8	2.0	1.4	1.1	0.9

Table IV – Baseline Respirable Dust Exposures

			Work Area Baseline Exposures			
			Arithmetic		Geometric	
Number	Work Area	n	Mean (mg/m^3)	Std. Dev.	Mean (mg/m^3)	Std. Dev.
1	Mill 1 - Average		0.46[1]			
2	Mill 1 - Milling	29	0.51	0.59	0.26	3.62
3	Mill 1 - Palletizing/Packing	26	0.53	0.27	0.46	1.70
4	Mill 1 - Packhouse Support	26	0.35	0.23	0.28	2.18
5	Mill 1 - Maintenance	23	0.45	0.27	0.36	2.18
6	Mine 1 – Underground	24	0.73	0.54	0.54	2.27
7	Mine 1 - Surface Crushing	1	0.14			
8	Mine 2 - Equipment Op.	11	0.22	0.25	0.11	3.36
9	Mine 2 - Crusher	4	0.83	0.55	0.70	1.96
10	Mine 2 - Maintenance	3	0.06	0.06	0.04	3.47
11	Minimal Exposure		0.09[2]			

[1]Average of work areas 2 through 5.

[2]This concentration represents the 5th percentile of all exposure data used to determine baseline exposures.

Table V – Work Area / Calendar Year-Specific Estimated Average Respirable Dust Concentrations (mg/m^3) Based on Exposure Scores of Rater 1

	Mill 1					Mine 1		Mine 2			General
Year	1	2	3	4	5	6	7	8	9	10	11
1948	1.4	1.7	1.3	1.4	1.3	0.9	0.4	-	-	-	0.2
1949	1.4	1.7	1.3	1.4	1.3	0.9	0.4	-	-	-	0.2
1950	1.4	1.7	1.3	1.4	1.3	0.9	0.4	-	-	-	0.2
1951	1.4	1.6	1.3	1.4	1.2	0.9	0.4	-	-	-	0.2
1952	1.2	1.6	1.1	1.1	1.2	0.9	0.4	-	-	-	0.2
1953	1.2	1.5	1.1	1.1	1.2	0.9	0.4	-	-	-	0.2
1954	1.2	1.5	1.1	1.1	1.2	0.9	0.4	-	-	-	0.2
1955	1.1	1.4	1.1	1.1	1.0	0.9	0.4	-	-	-	0.2
1956	1.1	1.4	1.1	1.1	1.0	1.0	0.4	-	-	-	0.2
1957	1.1	1.4	1.1	1.1	1.0	1.0	0.4	-	-	-	0.2
1958	1.1	1.4	1.1	1.1	1.0	1.0	0.4	-	-	-	0.2
1959	1.1	1.3	1.1	1.1	1.0	1.0	0.4	-	-	-	0.2
1960	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1961	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1962	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1963	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1964	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1965	1.0	1.1	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1966	1.0	1.0	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1967	1.0	0.9	1.1	1.1	0.8	1.0	0.4	-	-	-	0.2
1968	0.9	0.9	0.8	0.9	0.8	0.8	0.2	-	-	-	0.2
1969	0.8	0.9	0.8	0.6	0.7	0.8	0.2	-	-	-	0.1
1970	0.8	1.0	0.8	0.6	0.7	0.8	0.2	-	-	-	0.1
1971	0.8	1.0	0.8	0.6	0.7	0.8	0.2	-	-	-	0.1
1972	0.7	0.8	0.8	0.6	0.6	0.7	0.2	-	-	-	0.1
1973	0.7	0.7	0.8	0.6	0.6	0.7	0.2	-	-	-	0.1
1974	0.7	0.7	0.8	0.6	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1975	0.7	0.7	0.8	0.6	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1976	0.7	0.7	0.8	0.6	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1977	0.7	0.7	0.8	0.6	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1978	0.6	0.6	0.8	0.4	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1979	0.5	0.6	0.5	0.4	0.6	0.7	0.2	0.3	1.7	0.1	0.1
1980	0.5	0.6	0.5	0.4	0.5	0.7	0.1	0.3	0.8	0.1	0.1
1981	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.3	0.8	0.1	0.1
1982	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1983	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1984	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1985	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1986	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1987	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1988	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1
1989	0.4	0.5	0.5	0.4	0.5	0.7	0.1	0.2	0.8	0.1	0.1

Table VI – Measured and Predicted Respirable Dust Concentrations By Year and Work Area

Year	n	Work Area	Measured Average (mg/m³)	Predicted exposure (mg/m³)
1985	8	2	0.84	0.51
1984	13	2	0.36	0.51
1983	15	2	0.38	0.51
1982	4	2	0.06	0.51
1979	4	2	0.32	0.62
1975	12	2	0.99	0.69
1973	2	2	0.74	0.74
1969	5	2	0.89	0.95
1958	3	2	0.73	1.42
1952	3	2	1.11	1.57
1985	5	3	0.73	0.53
1984	6	3	0.44	0.53
1983	4	3	0.50	0.53
1979	2	3	0.32	0.53
1975	6	3	0.95	0.80
1973	2	3	0.80	0.80
1969	6	3	0.78	0.80
1958	2	3	1.60	0.80
1952	1	3	1.46	1.06
1983	1	4	0.46	0.35
1979	2	4	0.28	0.35
1975	10	4	0.41	0.60
1952	3	4	0.45	1.12
1975	10	5	1.24	0.64
1973	2	5	0.71	1.64
1952	1	5	0.86	1.16
1985	7	6	0.61	0.73
1984	9	6	0.62	0.73
1983	10	6	0.92	0.73
1982	2	6	0.99	0.72
1979	12	6	0.40	0.72
1975	14	6	0.86	0.73
1969	10	6	0.78	0.84
1958	8	6	1.40	0.96
1952	8	6	0.94	0.96
1985	1	7	0.27	0.14
1984	2	7	0.11	0.14
1983	3	7	0.31	0.14
1969	1	7	0.62	0.21
1985	5	8	0.40	0.22
1984	2	8	0.06	0.22
1983	3	8	0.34	0.83
1984	3	9	0.90	0.83
1983	1	9	0.50	0.83
1982	2	9	0.14	0.80

'Measured respirable mass data may be converted from historical particle count data by method outlined under Conversion of Dust Counts

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SIMILARITIES IN LUNG CANCER AND RESPIRATORY DISEASE MORTALITY OF VERMONT AND NEW YORK STATE TALC WORKERS

S.H. LAMM, M.D. • J.A. Starr, MSc
Consultants in Epidemiology and Occupational Health, Inc., Washington, DC 20007

ABSTRACT

The risks from malignant and non-malignant respiratory deaths of New York State and Vermont State talc workers with at least one year of employment have been compared for both miners and millers. The mortality patterns are similar. In both areas, the talc miners have a 4.5 fold risk of lung cancer, and the talc miners have no increased risk of lung cancer. In both areas, all workers appear to have an increased risk of non-infectious, non-neoplastic respiratory disease (NNRD) mortality, although only the Vermont millers show a statistically significantly elevated risk (7.9 fold). Thus, although the New York talc has been described as asbestiform talc and the Vermont talc as non-asbestiform talc, the mortality patterns of the workers appear to be inconsistent with that classification in that their lung cancer mortality rates are no different and only the Vermont talc millers show a significantly increased NNRD mortality.

INTRODUCTION

Studies of talc miners and millers in the New York and Vermont talc industry include analyses of mortality, morbidity, industrial hygiene, and mineralogy. Mineralogical differences between the two talcs have been highlighted. The upstate New York talc contains an elongated particulate not found in the Vermont talc that is considered by scientists at the National Institute for Occupational Safety and Health (NIOSH) as tremolitic asbestos and by scientists at the Bureau of Mines and at the company that owns the plant as true talc particulates and as prismatic non-asbestiform tremolite. NIOSH has called the New York State talc asbestiform talc and the Vermont talc non-asbestiform talc. Leaving the question of the mineralogical label of these particulates to the mineralogists, we have elected to examine the respiratory health outcomes of the employees at these two talc industries.

MATERIALS

The initial shaft of the New York State talc plant was sunk in 1947. Mining and milling operations started in 1948. The mortality experience (1947 through 1978) of all persons hired at the plant between 1947 and 1977 has been reported.[1] Mortality analysis was restricted to the 705 male employees (all caucasian). None of the 36 women employees had died of a respiratory condition. Sixty percent of the men worked at the plant for at least one year; twenty percent for two months to one year; and twenty percent for less than two months. Mortality analysis was reported separately for the 280 white male employees employed at the talc plant for less than one year and for the 425 white male employees employed for at least one year. That report[1] suggested that prior employment jobs accounted for the lung cancer rate. In-plant job records and prior employment histories on the job applications were analyzed. Employees were classified from the inplant job records as miners (187 worked exclusively in the mine), millers (152 worked exclusively in the mill), and others (34 worked in both the mine and the mill, 11 worked neither in the mine or the mill, and 41 had uninformative records).

The cohort of white male employees of the Vermont talc industry was developed from the records of the Vermont State Health Department's annual radiographic survey of employees of the dusty trades, begun in 1937. Selevan et al. of the National Institute for Occupational Safety and Health (NIOSH) defined the Vermont talc study cohort[2] as all white males in the Vermont talc industry on or after January 1, 1940 with at least one year of talc employment prior to January 1, 1970. Individuals who had at least two radiographs in the file and who had worked for any of five talc companies in three geographic areas of Vermont were eligible for the study. Mortality follow-up was continued through 1975 of the 392 men determined to belong to the cohort.

Health Department and company records were scrutinized to determine their job assignments, and each cohort member was classified as a miner after having had one year of exposure in the mine and/or as a miller after having had one year of exposure in the mill. 225 workers were classified as miners; 163 workers were classified as millers (of whom 47 had also been classified as miners); and 51 were not classifiable.

METHODS

This report compares standardized mortality ratios (SMRs) for malignant and non-malignant respiratory causes of death for miners and millers with at least one year of experience in the Upstate New York talc (said to be asbestiform) industry with those in the Vermont State talc (said to be non-asbestiform) industry. Comparison is reasonable, despite the differences in classification variables between the two studies.

RESULTS

The risks of lung cancer and of non-infectious, non-neoplastic respiratory disease (NNRD) for employees with at least one year in the mines or mills of New York State or Vermont State talc industries are presented, analyzed, and discussed below.

Respiratory Mortality of New York and Vermont Talc Workers

	Observed/Expected Ratios		Standardized Mortality Ratios	
	New York	Vermont	New York	Vermont
Lung Cancer				
Millers	1/1.41	2/1.96	071	102
Miners	5/1.15	5/1.09	460*	435*
Others	0/0.55	0/0.81	---	---
Total	6/3.11	7/3.66	1.92	1.91
NNRD				
Millers	2/0.74	7/0.89	270	787*
Miners	2/0.49	2/0.56	408	357
Others	2/0.38	2/0.34	526	588
Total	6/1.61	11/1.79	373*	615*

* – $p<0.05$, two-tailed Poisson test

The risk of malignant disease of the lung (lung/respiratory cancer) is not increased for millers but is significantly increased (4.5 fold) in talc miners both in New York (4.60) and in Vermont (4.35). No difference in risk is seen between miners and millers of New York and of Vermont (Figure 1). These data are sufficiently strong to rule out with eighty percent confidence an underlying relative risk for New York miners vs. Vermont miners of 1.7 and with about ninety five percent certainty an underlying risk of greater than 2.0.

The risk of non-malignant respiratory disease (excluding pneumonia and influenza), i.e., NNRD has a significantly increased risk (almost eight-fold) for Vermont talc millers but not for New York talc millers (risk of 2.7, not significant). The risks for NNRD for miners are calculated to be 4.1 and 3.6 (both non-significant) for those from New York and Vermont, respectively (Figure 2).

As for other respiratory system deaths, influenza or pneumonia caused the death of one New York State talc worker (0.9 expected) but no Vermont talc miner (0.7 expected) or miller (0.8 expected). Mesothelioma caused the death of one New York State talc man (15 years after hire which followed 28 years in mining and construction) and of one Vermont talc man.

DISCUSSION

We have attempted to assemble similarly defined cohorts of New York State and Vermont State talc workers in order to compare the respiratory mortality risks of their miners and millers. The exposures of millers generally exceed that of miners by a factor of two to six. Nonetheless, both groups demonstrate a similar excess lung cancer risk only for their millers and not for their miners. The similar lung cancer risks of the two groups of talc workers exposed to the differently described talcs suggest that the elongated particulates seen in the New York State talc have not introduced an increased lung cancer risk. We further observe that the risk of non-infectious, non-neoplastic respiratory death, while apparently increased in all groups, is significantly elevated only among the Vermont millers.

Standardized mortality ratios (SMRs) were calculated for each group based on age-specific, calendar time-specific, cause-specific mortality rates for white males. The New York State study SMRs had been calculated using U.S. rates with death certificates coded according to the eighth revision of the International Classification of Diseases (ICD). The Vermont State study SMRs were first calculated using U.S. rates and then recalculated by its authors using Vermont State rates for non-malignant respiratory disease and respiratory cancer

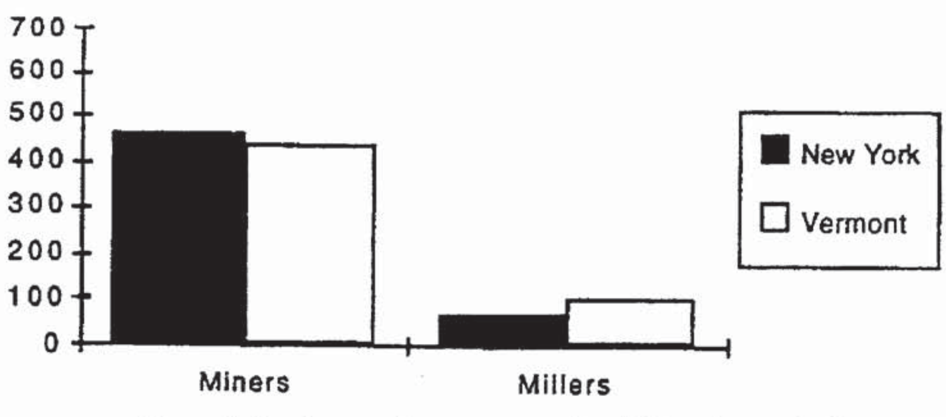

Figure 1. Respiratory or lung cancer mortality risk for miners and millers of New York State and Vermont State talc.

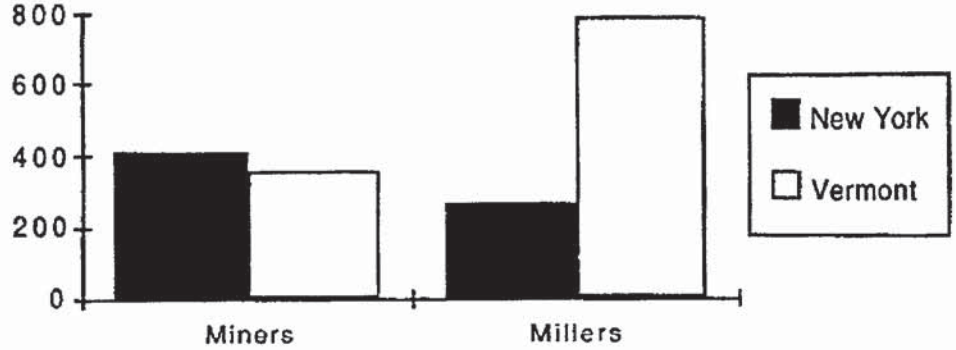

Figure 2. Non-infectious, non-malignant respiratory disease (NNRD) mortality risks for miners and millers of New York State and Vermont State talc.

COMPARATIVE LUNG MORTALITY RISKS of VERMONT and NEW YORK STATE TALC WORKERS

	with at least one year experience at Talc Plant				Ever employed		< One Year	
	Vermont		New York		New York		New York	
All Emp >1 yr.	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR
All Causes	44/37.15	118	64/49.83	128	118/83.58	141*	54/33.75	160*
All Cancers			15/9.55	157	26/15.7	165*	9/6.15	146
Lung Cancer	6/3.61	163	6/3.11	193	12/5.01	240*	6/1.90	316*
NNRD	11/1.79	615*	6/1.61	372*	6/2.64	227	0/1.03	---
Millers								
All Causes			20/21.74	92	35/30.97	113	15/9.23	163
All Cancers			3/4.23	71	6/5.94	101	3/1.71	175
Lung Cancer	2/1.96	102	1/1.41	71	1/1.92	52	0/.51	---
NNRD	7/.89	787*	2/0.74	270	2/1.02	196	0/.28	---
Miners								
All Causes			31/16.76	185*	50/26.32	190*	19/9.56	199
All Cancers			10/3.23	310*	15/5.00	300*	5/1.77	282
Lung Cancer	5/1.15	435*	5/1.09	460*	9/1.66	543*	4/0.57	701*
NNRD	2/0.56	357	2/0.49	408	2/0.77	260	0/0.28	---
Others								
All Causes			13/11.33	115	33/26.29	126	20/14.96	134
All Cancers			2/2.09	96	5/4.76	105	3/2.67	112
Lung Cancer	0/0.55	---	0/0.61	---	2/1.43	140	2/0.82	244
NNRD	2/0.34	588.0	2/0.38	526	2/0.85	235	0/0.47	---

COHORT DEFINITION

Cohort Variable	NEW YORK	VERMONT
Gender	Male	Male
Race	White	White
Employment Dates	1947-1977	1940-1969
Employment Duration	One Year +	One Year +
Mortality Dates	1947-1978	1940-1975

Cohort Numbers

Miners	152	163
Millers	187	225

LUNG CANCER

	NEW YORK	VERMONT
Observed/Expected		
Millers	1/1.41	2/1.96
Miners	5/1.15	5/1.09
Others	0/0.55	0/0.61
SMR		
Millers	71	102
Miners	460	435
Others	---	---

4/7/88

COMPARATIVE LUNG MORTALITY RISKS of
VERMONT and NEW YORK STATE TALC WORKERS
with at least one year experience at Talc Plant

All Emp >1 yr.	Vermont O/E	Vermont SMR	New York O/E	New York SMR	New York (Ever employed) O/E	New York (Ever employed) SMR	New York (< one Year) O/E	New York (< one Year) SMR
LATENCY (Years)								
0-4			0/0.27	---	0/0.42	---	0/0.15	---
5-9			0/0.31	---	0/0.49	---	0/0.18	---
10-14			1/0.45	224	1/0.69	145.0	0/0.24	---
15-19			2/0.60	331	2/0.98	205.0	0/0.38	---
20-24			3/0.79	378	8/1.29	623*	5/0.50	1000
25-29			0/0.65	---	1/1.09	92.0	1/0.44	227
30+			0/0.04	---	0/0.05	---	0/0.01	---
Total			6/3.11	193	12/5.01	240*	6/1.90	316*
0-9			0/0.58	---	0/0.91	---	0/0.33	---
10-19			3/1.05	285	3/1.67	180	0/0.62	---
20-29			3/1.44	208	9/2.38	378*	6/0.94	638*
30+			0/0.04	---	0/0.05	---	0/0.01	---
Total			6/3.11	193	12/5.01	240*	6/1.90	316*
0-4			0/0.27	---	0/0.42	---	0/0.15	---
5-14			1/0.76	132	1/1.18	85	0/0.42	---
15-24			5/1.39	360*	10/2.27	441*	5/0.88	568*
25+			0/0.69	---	1/1.14	87	1/0.45	222
Total			6/3.11	193	12/5.01	240*	6/1.90	316*

4/7/88

COMPARATIVE LUNG MORTALITY RISKS of
VERMONT and NEW YORK STATE TALC WORKERS
with at least one year experience at Talc Plant

		Vermont		New York	
		O/E	SMR	O/E	SMR
All Causes	Emp >1	44/37.15	118.0	64/49.83	128
All Cancers	Emp >1			15/9.55	157
Lung Cancer	Emp >1	6/3.61	163	6/3.11	193
NNRD	Emp >1	11/1.79	615	6/1.61	372
Pneumonia/Influ	Emp >1	0/1.89	000	1/0.9	109
All Causes	Millers			20/21.74	92
All Causes	Miners			31/16.76	185
All Causes	Others			13/11.33	115
All Cancers	Millers			3/4.23	71
All Cancers	Miners			10/3.23	310
All Cancers	Others			2/2.09	96
Lung Cancer	Millers	2/1.96	102	1/1.41	71
Lung Cancer	Miners	5/1.15	435	5/1.09	460
Lung Cancer	Others	0/0.55	---	0/0.61	---
NNRD	Millers	7/.89	787	2/0.74	270
NNRD	Miners	2/0.56	357	2/0.49	408
NNRD	Others	2/0.34	508	2/0.38	526

4/7/88 **Bold** = $p < 0.05$

Pneumonia/Influ	Millers	0/.83	000
Pneumonia/Influ	Miners	0/.67	000
Pneumonia/Influ	Others	0/.39	000

Standardized Mortality Ratios

	Vermont	New York
Lung Cancer		
Millers	102	71
Miners	435	460
Others	---	---
NNRD		
Millers	787	270
Miners	357	408
Others	588	526

NON-INFECTIOUS, NON-MALIGNANT RESPIRATORY DISEASE

NEW YORK

VERMONT

Observed/Expected

Miller	2/0.74	7/0.89
Miners	2/0.49	2/0.56
Others	2/0.38	2/0.34

SMR

Millers	270	787
Miners	408	357
Others	526	588

with death certificates coded according to the seventh revision of the ICD. This report bases the SMRs on the U.S. rates.

The New York State study reports lung cancer as their measure of malignant respiratory disease and NNRD (non-infectious, non-neoplastic respiratory disease) as their measure of non-malignant respiratory disease. The Vermont State study reports respiratory cancer as their measure of malignant respiratory disease and ONMRD (other non-malignant respiratory disease) as their measure of non-malignant respiratory disease. Both NNRD and ONMRD are terms for total non-malignant respiratory disease, excluding influenza and pneumonia. We have used the labels of lung cancer and NNRD to represent the malignant and non-malignant respiratory disease measures.

Twelve of the thirteen respiratory cancers among the New York State talc workers were lung cancers. The thirteenth case was a man whose five years at the plant included three months as a laborer/oiler in the talc mill and ended with death from mediastinal cancer. Re-analysis of the New York State data as respiratory cancer rather than lung cancer would have reduced the SMR estimates by about 5% but not have altered the comparison between the miners and millers. Both the New York and the Vermont data are compared against U.S. mortality rates.

The Vermont data included persons with experience in both the mine and the mill in each category; the New York data separated them out. There were only 34 such New York workers with experience in both the mine and the mill. Less than 0.1 lung cancer and less than 0.1 NNRD deaths were expected among them, and none were observed. Including this group among the miners and the millers of New York State would not have affected the results.

Studies of both cohorts lack full information on smoking history. Each indicates that most of the lung cancer cases were known to be cigarette smokers, but data on smoking appears to be inadequate for both cohorts. There is no evidence that miners and millers differ in their smoking habits. Thus, it is unlikely that the differences observed in these comparisons could be due to differences in smoking between groups.

The mortality of the experienced employees of the New York and Vermont cohort who worked other than in the mine or the mill for a year were also examined. There were no lung cancer deaths. Each group had two NNRD deaths, yielding non-significant risks of 5.9 for those from Vermont and 5.3 for those from New York.

While the NNRD mortality may be due to dust exposures at the talc plants, the etiology of the lung cancer is less clear. The NIOSH authors[2] concluded that talc dust was unlikely to be the cause of the respiratory cancer, since the risk was seen only in the miners and not seen among the millers, a group with probable higher dust exposure. Radon daughter measurements in the New York mine do not explain the finding. The presence of a particulate in New York dust and not in Vermont talc dust cannot explain the difference.

The CEOH study[1] had supported the hypothesis of risk from prior employments as the explanation for the lung cancer risk of the New York State talc workers, however, that hypothesis has not been examined for the Vermont talc workers. Further study of both cohorts should be undertaken to explain the mortality patterns seen. The small number of cases in either group will probably be a hindrance to a full and clear explanation. Both cohorts should probably be extended to include later employees and the period of follow-up should be brought more current by at least a decade. A four-fold risk of lung cancer seen in two different studies of talc miners (but not millers) cries for an explanation.

REFERENCES

1. Lamm, S.H., Levine, M.S., Starr, J.A., Tirey, S.L.: Analysis of Excess Lung Cancer Risk in Short-Term Employees. *Am. J. Epi.* 127(6):1202-1209 (1988).
2. Selevan, S.G., Dement, J.M., Waggoner, J.K., Froines, J.R.: Mortality Patterns among Miners and Millers of Non-Asbestiform Talc: Preliminary Report. *J. Environ. Path. and Tox.* 2:273-284, 1979.