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A Retrospective Cohort Mortality Study of Males Mining and Milling Attapulgite Clay

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To evaluate the possible health effects of occupational exposure to a nonasbestos mineral fiber, a cohort of 2,302 males employed for at least 1 month between 1940 and 1975 at an attapulgite (clay fiber) mining and milling facility was followed through 1975. A significant deficit of mortality (SMR = 43, 90% CI 23-76) from nonmalignant respiratory disease (NMRD) was observed for the cohort based on age-, calendar year-, and race-specific rates for U.S. males. A marked deficit of NMRD was seen regardless of presumed dust exposure level, induction-latency period, or duration employed. A statistically significant excess of mortality from lung cancer was observed among whites (SMR = 193, 90% CI 121-293), but a deficit occurred among nonwhites (SMR = 53, 90% CI 21-112). Lung cancer risk in either race was not altered substantially with presumed dust exposure level, induction-latency period, or duration employed with one exception—those employed for at least 5 years in high-exposure-level jobs.

Key words: nonasbestos fibers, carcinogenicity, fibrogenicity, clay fibers

INTRODUCTION

The carcinogenicity and fibrogenicity of asbestos are well established in both animals and humans. Substantial experimental evidence has been published and recently reviewed [Harington, 1981] supporting the theory that the carcinogenicity, and possibly the fibrogenicity, of asbestos and of other durable fibers is related to the fiber dimensions [Pott et al, 1976; Stanton et al, 1981; Wagner, 1980; Davis et al, 1978]. Stanton et al [1981] assessed the carcinogenicity in rats of a wide variety of durable mineral fibers. They found that the probability of developing pleural sarcoma correlated well with exposure to fibers that measured < 0.25 μm in diameter and $>$ 8 μm in length. High positive correlations for occurrence of pleural sarcoma also were demonstrated for exposure to fibers with diameters up to 1.5 μm and lengths $>$ 4 μm. Data from other investigators corroborate Stanton's findings at approximately the same dimensions [Harington, 1981].

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If the fiber dimension hypothesis is correct and can be extended to man, then prevention of disease resulting from exposure to man-made fibers may be possible by restriction of fiber size during production. To assess the fiber dimension hypothesis, the National Institute for Occupational Safety and Health (NIOSH) undertook cohort mortality and industrial hygiene studies of clay fiber (attapulgite) miners and millers, because most of the natural fibers associated with this industry were not in the long, thin categories hypothesized to be carcinogenic or fibrogenic [Zumwalde, 1977].

Attapulgite clay, commonly called attapulgite after its principal mineral component, is a crystalline, hydrated, magnesium-aluminum silicate with a unique chain structure that gives it unusual colloidal and sorptive properties. "Attapulgite is a commercial designation for materials that consist of asbestiform and platy palygorskite" [National Research Council, 1984]. Attapulgite is also the principal member of a group of sorptive clays including sepiolite and montmorillonite and known collectively as fuller's earth [Haden and Schwint, 1967] because of their original use in cleaning or "fulling" wool.

Attapulgite has a wide variety of industrial applications: colloidal grades are used in paint and drilling muds, and sorptive grades find use as clarifying agents, animal litter, pesticide carriers, components of no-carbon copy papers, and pharmaceutical carriers. U.S. attapulgite is mined and milled only in the Georgia—Florida area of the U.S. (900,000 tons/year). Structurally similar clays, including attapulgite from Spain and palygorskite from the U.S.S.R., consist of much longer fibers [Huggins et al, 1962]. As mined in the U.S., attapulgite clay contains: 70–80% attapulgite; 10–15% montmorillonite, sepiolite, and other clays; 4–8% quartz; and 1–5% calcite or dolomite [Haden and Schwint, 1967].

PLANT AND EXPOSURE DESCRIPTIONS

One of the major attapulgite companies in the U.S. was selected for study. Production at the plant started around 1920, and mostly granular (large particle) grades of attapulgite were produced. Although products of fine particle sizes were more common after 1940, fine materials are always produced to some degree in the processing of attapulgite clay. The clay is strip mined, hauled to the plant, then crushed to half-inch granules. Some products require extrusion as an intermediate step. All products are then rotary dried, reduced in grinding mills, size-classified by sifting, and either bagged or shipped in bulk.

In 1976, exposure levels for total and respirable personal dust samples were measured among three groups of employees during the industrial hygiene survey [Zumwalde, 1977] (Table I); group A workers were exposed to the initial process in milling the wetted clay, group B workers were exposed to the clay after it had been dried and ground, and group C workers were exposed to the clay after it had been sized for bagging or bulk shipment. In general, group A was exposed to less respirable and total dust than the other groups. Time-weighted average (TWA) free silica exposures and trace metal analyses (Cd, Co, Cr, Fe, Mn, Ni, Zn) from airborne samples were usually near the lower limits of detection. Iron was the only trace metal found in any appreciable quantity (1–2.4%). All TWA concentrations for respirable dust were below the Mine Safety and Health Administration nuisance dust standard of 5 mg/m^3.

TABLE I. Time-Weighted Average Exposures to Airborne Dust by Mill Job Type

	Job type[a]	No. of samples	Geometric mean (mg/m^3)	Geometric standard deviation
Total dust	A	6	1.75	3.95
	B	5	15.90	1.85
	C	27	11.92	2.41
MSHA standard			10.0	
Respirable dust	A	11	0.45	2.95
	B	11	1.60	2.03
	C	66	1.15	2.26
MSHA standard			5.0	

[a]Job definitions: A, initial process, milling wetted clay; B, intermediate process, exposed to clay after drying and grinding; C, final process, exposed to clay after sizing.

To determine fiber size characteristics, 15 airborne samples of total dust were randomly selected from the 200 collected within the mill. Measurements made by transmission electron microscopy (TEM) at × 20,000 on approximately 460 fibers (after high-energy ultrasound dispersion) indicated attapulgite clay fiber lengths of 0.1–2.5 μm with a geometric mean of 0.52 μm, and diameters of 0.02–0.1 μm, with a geometric mean of 0.06 μm. In addition, fiber aspect ratios (length to diameter) were generally $>10:1$. It was noted during the TEM analysis that attapulgite clay fibers had an affinity to agglomerate into jagged particles ranging in diameters from 0.5 to 5.0 μm. No other types of fibrous minerals ($>3:1$ aspect ratio) were observed in either airborne or bulk mine samples based on analysis by both selected area electron diffraction and energy dispersive X-ray analysis [Zumwalde and Dement, 1977].

METHODS

The study cohort was limited to the 2,302 males who had worked either in the mine or mill of this attapulgite facility for at least 1 month any time between January 1, 1940, and December 31, 1975. The cohort members were identified from microfilm copies that we made of company personnel records. In most cases, data initially missing on race, sex, date of birth, or work history were later supplied by the company management or the Social Security Administration. Fifty males ($<2\%$ of the cohort) for whom race could not be determined were assumed to be white.

The vital status determination as of December 31, 1975, of each of the 2,302 cohort members was based on information from the Social Security Administration, Internal Revenue Service, state bureaus of motor vehicles, state vital statistics offices, and other sources. Death certificates were obtained, and the underlying cause of death was coded by a qualified nosologist in the revision of the International Classification of Diseases (ICD) in effect at the time of death and converted to 7th revision ICD codes. This method was used to maintain comparability with procedures used to determine cause-specific death rates for the U.S. male population.

Person-years at risk (PYAR) were begun for each employee upon the latter of two events; January 1, 1940, or completion of 1 month of employment at the facility.

PYAR were terminated on the date of death or, for persons alive or lost to follow-up, on December 31, 1975. This date was chosen for the persons lost to follow-up to minimize any chance of overestimating risk; consequently, we might have slightly underestimated risk. The race-, sex-, and cause-specific expected deaths, based on U.S. mortality rates, were adjusted for 5-year age- and calendar-specific periods using a modified life-table analysis program [Waxweiler et al, 1983].

Standardized mortality ratios (SMR) were calculated to represent the observed number of deaths as a percentage of the expected number. Ninety percent confidence intervals (CI) for the SMRs were based on a Poisson distribution for the observed frequency [Armitage, 1971].

Further analyses were carried out by dust exposure level. Milling jobs corresponding to groups B and C in Table I were considered to be high-exposure jobs relative to group A. Jobs in the mill, such as maintenance, that required occasional work among groups B and C were classified as intermittent-exposure jobs. Mining jobs were not sampled for dust levels but were included in the analyses with group A jobs based on our judgment of very little exposure. There were no historical industrial hygiene data available, and the dust levels undoubtedly varied over time. However, for purposes of the epidemiologic analysis, we assumed that the same jobs were always the one with high or intermittent exposures.

For these analyses, risk (PYAR) and induction-latency period began when an employee entered an exposed job, and duration of employment refers to time spent in exposed jobs. For instance, consider a person who worked first in a group A job, then in a maintenance (intermittent-exposure) job. In the first overall analysis, his PYAR and induction-latency began when he began the group A job and his duration of employment included all jobs. In the intermittent-exposure analysis, his PYAR and induction-latency began with his maintenance job, and his duration of employment included only his maintenance job.

RESULTS

All but 140 (6%) of the cohort members were successfully traced until December 31, 1975 (Table II). Of the 315 reported to be deceased, death certificates could not be located for 19 (6%). A smaller proportion of death certificates could not be located for whites, 3%, than for nonwhites, 9%. The 2,302 cohort members (1,235

TABLE II. Vital Status of Cohort of Male Attapulgite Miners and Millers as of December 31, 1975

	All exposure levels		Intermittent-exposure		High-exposure		Intermittent high-exposure	
	Whites	Nonwhites	Whites	Nonwhites	Whites	Nonwhites	Whites	Nonwhites
Alive	1,021	826	185	231	141	599	320	756
Deceased	151	164	42	88	18	79	59	139
Missing death certificates	5	14	1	7	0	8	1	11
Lost to follow-up	63	77	11	32	8	45	19	70
Total	1,235	1,067	238	351	167	723	398	965
Person-years at risk	20,297	18,426	4,650	8,395	2,150	10,415	6,784	16,498

white and 1,067 nonwhite) contributed 38,723 PYAR to the study; an average of 17 years per member. A much smaller proportion of the whites, 398 of 1,235 (32%), than of the nonwhites, 965 of 1,067 (91%), had worked in intermittent- or high-exposure jobs. Few persons, regardless of race, had worked in both an intermittent- and a high-exposure job. For instance, only four white males (238 + 164 − 398 = 4 in Table II) were included both in the intermittent-exposure cohort and in the high-exposure cohort.

The 315 deaths observed in the study were 80% of expected (Table III). Overall mortality was less favorable among whites (151 observed and 157.8 expected) than among nonwhites (164 observed and 235.9 expected). The low overall mortality among the nonwhites was due mostly to very favorable mortality patterns for tuberculosis, all malignant neoplasms combined, circulatory system diseases, and NMRD. With the exception of all malignant neoplasms, whites experienced fewer observed than expected deaths for these same causes, but the deficits were not as great.

Among the entire cohort, statistically significant excess risk was not seen for any cause. The excess risk for liver cancer was not quite significant (SMR = 250, 90% CI 99–526). Slight, statistically insignificant excesses were found for cancer of the stomach overall (SMR = 120, 90% CI 52–237) and for cancer of the intestines among whites (SMR = 160, 90% CI 55–366). A large discrepancy was observed between the two races for mortality from lung cancer. Whites exhibited almost a twofold excess risk (SMR = 193, 90% CI 121–293), nonwhites a 40% deficit (SMR = 53, 90% CI 21–112).

For selected causes, associations between mortality and duration of employment were investigated after limiting the analysis to the period of 10 or more years since initial employment at the facility (Table IV). The excess of stomach cancer, based on six cases, was not statistically significant and showed no relationship to duration of employment. The liver cancer excess was essentially eliminated after 10 years since first employment, precluding meaningful analysis by duration of employment.

Mortality from lung cancer was strikingly different between whites and nonwhites and thus is presented separately for the two racial groups (Table IV). After 10 years induction-latency, whites exhibited nearly a twofold ratio of observed to expected deaths, which was statistically significant. However, no trend by duration of employment was evident. Among nonwhites, on the other hand, there was a substantial deficit in risk for those working < 5 years and a slight excess risk for those working longer. A statistically significantly low SMR (41; 90% CI 19–77) was seen for NMRD. The deficit in mortality from NMRD was independent of duration of employment.

Further analysis of lung cancer by calendar period of risk indicated no trends, despite the fact that no such deaths were observed prior to 1960. Furthermore, younger (<65 years) persons appeared to be at no different relative risk (17 observed vs. 14.8 expected) than older persons (4 observed vs. 2.8 expected). Similar analyses for stomach cancer and NMRD also found no trends by age or calendar period.

Analyses of employees exposed to intermittent levels of dust combined with those exposed to high levels revealed no statistically significant excesses overall; however, the numbers of expected deaths were usually quite small (Table V). Mortality from NMRD in these subcohorts was much lower than expected. Excess lung cancer deaths were limited to the whites. This anomalous finding was partially clarified by dividing the high-exposure results for lung cancer further by duration

TABLE III. Cause-Specific Mortality Among Male Attapulgite Miners and Millers 1940–1975*

Cause of death	ICD (7th Rev)	White		Nonwhite		Total		SMR
		Obs	Exp	Obs	Exp	Obs	Exp	
All causes		151	157.8	164	235.9	315	393.7	80
Tuberculosis	(1–19)	1	1.8	2	8.6	3	10.4	29
All malignant neoplasms (MN)	(140–205)	31	28.0	18	33.5	49	61.5	80
Buccal cavity and pharynx	(140–148)	0	0.9	0	1.4	0	2.3	
Digestive System	(150–159)	9	8.2	8	11.6	17	19.8	86
Esophagus	(150)	1	0.6	0	2.1	1	2.7	
Stomach	(151)	2	1.7	4	3.3	6	5.0	120
Intestines	(152,153)	4	2.5	1	2.1	5	4.6	109
Rectum	(154)	0	1.0	0	0.9	0	1.9	
Liver and biliary tract	(155,156A)	2	0.7	3	1.3	5	2.0	250
Pancreas	(157)	0	1.5	0	1.8	0	3.3	
Other digestive system	(158,159)	0	0.1	0	0.2	0	0.3	
Lung	(162,163)	16	8.3	5	9.4	21	17.7	119
Larynx	(161)	0	0.4	1	0.6	1	1.0	
Lymphatic and hematopoietic tissue	(200–205)	2	3.0	2	2.5	4	5.5	73
Other malignancies		4	7.2	2	8.0	6	15.2	39
Vascular lesions of CNS	(330–334)	11	10.3	21	21.4	32	31.7	101
Circulatory system diseases	(400–468)	62	66.9	47	75.6	109	142.5	76
Nonmalignant respiratory system disease	(470–527)	4	7.8	5	13.0	9	20.8	43
Acute upper respiratory infection	(470–475)	0	0.0	0	0.1	0	0.1	
Influenza and pneumonia	(480–483, 490–493)	1	3.7	3	9.1	4	12.8	31
Bronchitis	(500–502)	0	0.5	0	0.4	0	0.9	
Other nonmalignant respiratory disease	(510–527)	3	3.6	2	3.4	5	7.0	71
Accidents	(800–962)	17	16.1	20	22.9	37	39.0	95
All other causes		17	25.0	32	52.8	49	77.8	63
Unknown cause	(780–783, 795)	3	1.9	5	7.8	8	9.7	83
Missing death certificates		5		14		19		

*Obs, observed; Exp, expected; SMR = 100 (obs/exp).

TABLE IV. Cause-Specific Mortality by Duration of Employment Among Attapulgite Miners and Millers After 10 Years Since First Employment by Duration of Employment*

	Duration of Employment						
	1 Month–4.9 Years		⩾ 5 Years		Total		
Cause of Death	Obs	Exp	Obs	Exp	Obs	Exp	SMR
All causes	166	186.8	99	121.6	265	308.4	86
Stomach cancer	3	2.4	3	1.9	6	4.2	143
Intestinal cancer	2	2.3	2	1.6	4	3.9	103
Liver cancer	2	1.0	0	0.7	2	1.7	
Lung cancer	12	9.9	9	5.9	21	15.8	133
Whites	10	4.3	6	2.9	16	7.2	222
Nonwhites	2	5.6	3	3.0	5	8.6	58
Nonmalignant respiratory system disease	4	10.2	3	6.8	7	17.0	41
Missing and unknown cause	17	4.4	6	2.9	23	7.4	311
Person-years at risk	14,193		7,174		21,366		

*Obs, observed deaths; Exp, expected deaths; SMR = 100 (obs/exp).

TABLE V. Mortality From Selected Causes Among Attapulgite Miners and Millers Exposed to Intermittent and High Levels of Respirable Dust*

	Intermittent exposure				High exposure			
	White		Nonwhite		White		Nonwhite	
Cause of death	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp
All causes	42	49.1	88	120.3	18	18.4	79	120.6
Stomach cancer	1	0.6	3	1.8	1	0.2	1	1.6
Lung cancer	4	2.5	3	4.9	3	1.0	3	4.7
Circulatory system diseases	19	22.1	27	39.9	8	7.9	19	37.4
Nonmalignant respiratory system disease	1	2.6	1	6.8	1	0.9	3	6.4

*Obs, observed deaths; Exp, expected deaths.

worked. Among those with >5 years of employment in high-exposure jobs, there were five observed deaths from lung cancer versus 1.62 expected (SMR = 309, 90% CI 122–649). Both whites (two observed, 0.45 expected) and nonwhites (three observed, 1.18 expected) contributed to this excess.

Because of particular a priori interest in lung cancer and digestive system cancer and the slight excesses of observed deaths for lung and stomach cancer in Tables IV and V, further analyses of these sites were performed. Experience in both intermittent- and high-exposure jobs was considered simultaneously, and risk was examined by periods of duration worked in such jobs and induction-latency period (Table VI). The only statistically significant excess in this analysis was for lung cancer among whites after 20 years induction-latency (six observed, two expected; SMR = 300, 90% CI 131–592). The comparable cell for nonwhites had a decreased risk for lung cancer. An examination of risk of mortality from stomach cancer among those who had worked in either intermittent- or high-exposure jobs uncovered no statistically significant excesses (Table VII).

TABLE VI. Observed and Expected Deaths From Lung Cancer by Induction-Latency Period and Duration Worked in High- and Intermittent-Exposure Jobs*

Induction latency (years)		Duration					
		1 Month–4.9 years		⩾5 years		Total	
		Obs	Exp	Obs	Exp	Obs	Exp
0–9.9	White	0	0.4	0	0.1	0	0.5
	Nonwhite	0	0.6	0	0.1	0	0.7
10–19.9	White	1	0.8	0	0.3	1	1.1
	Nonwhite	0	1.5	1	0.4	1	1.9
⩾20	White	3	1.2	3	0.7	6	2.0
	Nonwhite	2	3.7	2	2.0	4	5.7
Total	White	4	2.5	3	1.1	7	3.5
	Nonwhite	2	5.8	3	2.6	5	8.3
	All	6	8.2	6	3.6	12	11.9

*Obs, observed deaths; Exp, expected deaths.

TABLE VII. Observed and Expected Deaths From Stomach Cancer by Induction-Latency Period and Duration Worked in High-and Intermittent-Exposure Jobs*

Induction Latency (years)	Duration					
	1 Month-4.9 Years		⩾ 5 Years		Total	
	Obs	Exp	Obs	Exp	Obs	Exp
0–9.9	0	0.52	0	0.06	0	0.58
10–19.9	2	0.76	1	0.23	3	0.99
⩾20	1	1.10	2	1.00	3	2.10
Total	3	2.37	3	1.30	6	3.67

*Obs, observed deaths; Exp, expected deaths.

DISCUSSION

A substantial body of literature supports the concepts that fiber dimensions influence their respirability, deposition, carcinogenicity, and perhaps fibrogenicity. Respiratory tract and gastrointestinal tract deposition of fibrous aerosols in humans is dependent on a number of fiber characteristics, the most important being aerodynamic diameter [Timbrell, 1973; Dement and Harris, 1979]. Most airborne attapulgite clay fibers are found in jagged agglomerated particles; thus only a minor portion would be expected to be respirable. However, essentially all of the less common individual airborne fibers (0.1 μm in diameter) would be respirable, probably penetrating beyond the upper airways and into the smaller airways and alveoli [Bignon et al, 1978; Timbrell, 1973]. One individual exposed to airborne clay fibers and who had lung fibrosis was found to have clay fibers in his alveolar spaces [Bignon et al, 1978].

Fibrogenicity of fibers is thought to be influenced by fiber length. Both inhalation and pleural injection studies indicate that long fibers are more fibrogenic than shorter ones or particles [Davis et al, 1978; National Research Council, 1984]. Wagner [1980] has postulated that pulmonary fibrosis is a possible hazard for humans when exposures occur to fibers <2 μm in diameter and 10–50 μm in length. Our results appear to be consistent with this hypothesis. Airborne attapulgite fibers measured were short, 0.1–2.5 μm in length, and there was no excess of NMRD mortality. In fact, even after a 10-year induction-latency period, a striking deficit of

NMRD was seen (SMR = 41, 90% CI 19–77), which did not vary by duration of employment.

Carcinogenicity of fibers also has been shown experimentally to reflect the importance of fiber dimensions [Harington, 1981; Pott et al, 1976; Stanton et al, 1981; Wagner, 1982]. Until now, the direct evaluation of the carcinogenicity of attapulgite has depended on experimental data alone. In making such an evaluation, it is important to be aware of the strikingly different fiber sizes between U.S. attapulgite (short fibers) and attapulgite from Spain or palygorskite from the U.S.S.R. (long fibers). Pott et al [1974] demonstrated the tumorigenicity of palygorskite from the U.S.S.R. Stanton et al [1981] found minimal sarcoma induction after pleural implantation of two different samples of U.S. attapulgite, both of which consisted mainly of short, thin fibers. These results were consistent with their overall findings that the tumorigenicity of various types of fibers implanted pleurally in rats appeared to be correlated with the number of fibers less than 1.5 μm in diameter and greater than 4.0 μm in length. Furthermore, the tumorigenicity appeared to be most highly correlated to the number of fibers <0.25 μm in diameter and >8.0 μm in length [Stanton et al, 1981]. The diameters of airborne attapulgite fibers measured at our study plant fell into this range, but the lengths were much shorter (<2.5 μm). Nevertheless, it is still open to question whether the experimental results of Stanton et al can be extrapolated to the inhalation of fibers (and the cocarcinogenic effect of cigarette smoke) and subsequent development of epithelial tumors in humans.

We found a slight excess of stomach cancer after a 10-year induction-latency period but no statistically significant associations by exposure level or duration. This slight excess could not be explained by local rates for stomach cancer mortality [Riggan et al, 1983].

Our epidemiologic results for lung cancer were mixed. White employees experienced approximately a twofold excess of lung cancer that increased slightly but not substantially with induction-latency period, duration of employment, or exposure level. Nonwhite employees, on the other hand, experienced an overall lung cancer risk approximately half that expected based on U.S. rates. The pattern of local rates for lung cancer for white and nonwhite males did not account for this distinct difference in lung cancer risk. Deficits of lung cancer mortality among nonwhite employees were seen for all subcategories of duration of employment, induction-latency period, or exposure level except among those who worked at least 5 years in intermittent- and high-exposure jobs or at least 5 years in high-exposure jobs alone. Neither of these latter two excesses was statistically significant. However, the excess lung cancer mortality among all employees, regardless of race, who worked at least 5 years in high-exposure jobs was significant (5 observed vs 1.62 expected; SMR = 309, 90% CI 122–649).

Cigarette smoking must be considered as a potential explanation of the lung cancer pattern that we observed. No data on cigarette smoking were available for the cohort, but for whites and nonwhites the deficits of mortality from NMRD and circulatory system disease certainly do not appear to be consistent with an increased prevalence of cigarette smoking. Furthermore, among the current work force of white males and nonwhite males, there are, respectively, 44% and 46% current cigarette smokers, 21% and 11% exsmokers, and 35% and 44% nonsmokers (J. Gamble, personal communication). Despite the fact that smoking patterns may change over time in a dynamic cohort, and that cigarette smoking is a strong risk factor for lung cancer, it is doubtful that smoking explains any SMRs >200 [Steenland et al, 1984].

In addition to the lack of cigarette smoking data for the cohort, other limitations of this study must be noted, including 1) an inability to confirm the complete enumeration of the cohort with an independent source of data, 2) persons lost to follow-up, 3) missing death certificates, 4) missing race on 50 persons, 5) lack of longitudinal person-specific dosimetry, 6) few expected deaths beyond 20 years induction-latency, 7) few persons who worked for long durations at the facility, and 8) use of the general U.S. population for comparison. Because of these limitations, our findings should be interpreted as neither strongly supportive of nor contradictive of an association between the lung carcinogenicity of U.S. attapulgite, or short thin fibers in general, and lung cancer risk in humans. Given the importance of this cohort in its potential to further the understanding of fiber carcinogenicity and fibrogenicity, it is important that smoking data be gathered on this cohort and that mortality follow-up be extended prospectively in the future to increase our statistical power and observation of risk at longer induction-latency periods.

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