MORTALITY PATTERNS AMONG FIBROUS GLASS PRODUCTION WORKERS *

David L. Bayliss, John M. Dement, Joseph K. Wagoner, and Héctor P. Blejer

National Institute for Occupational Safety and Health
Center for Disease Control
Public Health Service
Department of Health, Education, and Welfare
Cincinnati, Ohio 45202

INTRODUCTION

Both fibrous glass and asbestos are fibrous substances. As a result of the carcinogenic and fibrogenic effects of inhaled asbestos in humans, it was only natural that concern should be directed toward the evaluation of the pathogenicity of inhaled fibrous glass.

Until recently, experimental studies using fibrous glass had produced conflicting results.[1-9] This is not unexpected as many of these experimental studies suffered from one or more defects, such as insufficient lapse of time since onset of exposure, questionable mode of exposure (route of entry and size of fiber), inadequate sample size, and lack of controls. Nevertheless, fibrogenicity and carcinogenicity of fibrous glass were demonstrated both by intrapleural and intraperitoneal experiments, and this pathogenicity was related more to the dimensions of the fiber than to its physicochemical properties.[10-14]

Only in recent years has sufficient time elapsed to make feasible studies among humans of the potential latent effects of occupational exposure to fibrous glass, since its industrial production and commercial use began about 1933 in the United States. Studies of employees in the fibrous glass industry by Wright,[15] Gross *et al.*,[16] Utidjian and de Treville,[17] Nasr *et al.*,[18] and Hill *et al.*[19] indicated essentially negative results. All of these studies had a common limitation: they were cross-sectional prevalence studies of current employees only, with no reference to terminated or former employees, groups shown in many other industrial settings to manifest the majority of latent biological effects. In support of this premise are recent observations by Enterline and Henderson [20] of an increased risk of chronic bronchitis as a cause of retirement disability among fibrous glass production workers. With this limitation in mind and the potential carcinogenicity of fibrous glass posed by Stanton and Wrench [12] and Pott and Friedrichs,[10] the National Institute for Occupational Safety and Health (NIOSH) undertook a three-part occupational health study of the fibrous glass manufacturing industry. The three parts were a retrospective cohort analysis of mortality patterns, industrial hygiene surveys, and a case-control study of respiratory disease.

* A synopsis of this paper, along with comments and rebuttal, are available from the National Technical Information Service, Springfield, Va. 22151.

Retrospective Cohort Analysis

Methods

Selected for study were all white males initially employed during the period January 1, 1940 through December 31, 1949 in one major fibrous glass construction products manufacturing facility. Employees potentially eligible for inclusion in the study cohort were ascertained through a complete search of all available company employment files. The study cohort was subsequently limited to individuals who were initially employed, as noted, and who had achieved 5 or more years of employment in fibrous glass production, packing, or maintenance activities by June 1, 1972. This restriction was imposed to eliminate effects of silica exposure present in the predecessor glass bottle plant, which had ceased production at this facility prior to 1940, and to minimize possible bias that might result from the destruction of some company employment files during a flood in or about 1960. The final study cohort consisted of 1,448 white males.

Table 1

Status as of June 1, 1972 among White Males Who Were Initially Employed between January 1, 1940 and December 31, 1949 and Subsequently Achieved 5 or More Years of Employment in Fibrous Glass Production, Packing, or Maintenance Activities

Status as of June 1, 1972	Study Cohort Members No.	%
Known to be alive	1,072	74.0
Known to be deceased	376	26.0
Death certificate obtained	376	26.0
Death certificate outstanding	0	0.0
Not known to be alive or deceased	0	0.0
Total	1,448	

Follow-up of all study cohort members was attempted from the time of termination of employment to June 1, 1972. Vital status ascertainment was made through records maintained by federal, state, and local governmental agencies, including sources such as the Social Security Administration, city and county health departments, and area funeral homes. With this follow-up program, no members of the study cohort were lost to observation (Table 1).

Death certificates were obtained for those known to be dead, and causes of death were interpreted by a qualified nosologist according to the revision of *The Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death* in effect at the time of death.[21] No cohort members remained for whom causes of death were not obtained.

A modified life-table technique was used to obtain person-years at risk of dying by 5-year calendar time periods, 5-year age groups, duration of employment, and number of years since onset of initial employment. Comparison was made between the observed number of deaths in the study cohort and the number expected on the basis of age, calendar time, and cause-specific mortality rates for the general white male population of the United States.

Results

During the calendar period January 1, 1940 to June 1, 1972, a total of 376 deaths were observed among the study cohort, whereas 404.24 deaths would have been expected (TABLE 2). This deficit of overall mortality is not unanticipated. A certain level of good health was required of all individuals initially employed at this fibrous glass production facility through an extensive

TABLE 2

OBSERVED AND EXPECTED DEATHS ACCORDING TO CAUSE AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING, OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972

Causes of Death	List No.*	Observed	Expected
Tuberculosis	001–019	0	4.69 †
Malignant neoplasms	140–199	54	64.09
Digestive system	150–159	25	22.93
Respiratory system	160–164	16	20.23
Other & unspecified	140–149	13	20.93
Vascular lesions affecting central nervous system	330–334	30	32.84
Diseases of heart	400–443	163	179.86
Nonmalignant respiratory disease	470–527	25	19.96
Influenza & pneumonia	480–493	6	9.92
Other respiratory-disease	470–475, 500–527	19	10.04 †
Cirrhosis of liver	581	2	8.93 †
Violent deaths	800–958	39	34.37
All other known causes		63	59.50
Unknown causes		0	—
Total		376	404.24

* Seventh revision of *International Lists of Diseases and Causes of Death.*
† Significant at $p < 0.05$.

preemployment medical screening program fully implemented prior to 1940. In support of this interpretation is the observation that the deficit of overall mortality among fibrous glass workers disappeared within 15 years since onset of employment. Further support for this interpretation is the significant deficit of deaths due to tuberculosis (0 observed vs. 4.69 expected; $p < 0.05$).[22]

An excessive risk was demonstrated for only one cause of death category, nonmalignant respiratory diseases excluding influenza and pneumonia, where 19 were observed, whereas only 10.04 were expected, an excess significant at $p < 0.05$.[22]

Among the total study cohort, no excessive mortality risk was demonstrated for all malignant neoplasms combined. In the category of respiratory malig-

TABLE 3

OBSERVED AND EXPECTED MALIGNANT RESPIRATORY DISEASE DEATHS ACCORDING TO TIME SINCE ONSET OF EMPLOYMENT AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING, OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972

Interval Since Onset of Employment (years)	Observed	Expected
5–9	2	1.44
10–14	2	2.63
15–19	4	4.09
20–24	1	5.87
25–29	7	5.30
≥ 30	0	.90
Total ≥ 5 years	16	20.23

nancy, 16 deaths were observed among these workers, as contrasted with 20.23 expected. Moreover, when consideration was given to the risk of respiratory malignancy according to interval since onset of employment, no excessive risk was demonstrable, even after 20 years of observation (TABLE 3).

The finding of an excessive risk of nonmalignant respiratory disease among these fibrous glass production workers raised the possibility that individuals who choose employment in the fibrous glass industry may have a predisposition to respiratory disease. Consequently, to evaluate the possible role of selective factors, analyses were made of the nonmalignant respiratory disease risk according to time-interval since onset of initial employment in the fibrous glass industry. Examination of the mortality pattern in TABLE 4 shows that such an

TABLE 4

OBSERVED AND EXPECTED NONMALIGNANT RESPIRATORY DISEASE DEATHS (LESS INFLUENZA AND PNEUMONIA) ACCORDING TO TIME SINCE ONSET OF EMPLOYMENT AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING, OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972

Interval Since Onset of Employment (years)	Observed	Expected
5–9	0	0.53
10–19	9	2.90
20–29	10	6.11
≥ 30	0	0.50
Total ≥ 5 years	19	10.04 *
Total ≥ 10 years	19	9.51 †

* Significant at $p < 0.05$.
† Significant at $p < 0.01$.

TABLE 5

SUMMARY OF FIBER CONCENTRATIONS (FIBERS/ML) IN LARGE FIBER INSULATION PRODUCTION FACILITIES

Summary for All Operations	Insulation Plant			
	A	B	C	D *
Mean conc. (fibers/ml)	0.06	0.11	0.13	0.08
Range	0.01–0.13	0.00–0.47	0.04–0.26	0.01–0.83
No. of samples	47	48	21	49

* Study plant.

excessive risk occurred only after 10 years since onset of employment. This lack of an excessive nonmalignant respiratory disease risk during the first 9 years is not consistent with a self-selection of individuals who have overt disease. Although the role of prior employment in industries other than fibrous glass production cannot be totally evaluated in the etiology of this excessive nonmalignant respiratory disease risk, note should be made that several individuals had incidental periods of employment in dusty trades.

INDUSTRIAL HYGIENE SURVEYS

The apparent lack of an unusual malignant respiratory disease risk among individuals in the study cohort must be evaluated in terms of the characteristics of the work air environment to which the workers were exposed. To characterize such environmental work exposures, industrial hygiene surveys were conducted in the study plant and in three other insulation plants that produce similar products. These surveys determined fiber and total dust air concentrations, in addition to airborne fiber diameter and length distributions.

TABLE 5 provides a summary of airborne fiber concentrations for the four insulation plants surveyed. An average fiber concentration of 0.08 fibers/ml of air was observed for plant D, the study plant. In addition, very low fiber concentrations were noted for plants A, B, and C. TABLE 6 presents a summary of total airborne dust concentrations for these four plants. The study had an

TABLE 6

SUMMARY OF TOTAL DUST CONCENTRATIONS (MG/M^3) IN FIBER INSULATION PRODUCTION FACILITIES

Summary for All Operations	Insulation Plant			
	A	B	C	D *
Mean conc. (mg/m^3)	0.8	1.3	2.7	0.3
Range	0.1–3.8	0.3–4.8	0.2–14.5	0.1–1.0
No. of samples	44	48	21	33

* Study plant.

TABLE 7

SUMMARY OF AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES (LARGE-DIAMETER GLASS FIBERS)

Summary for All Operations Combined	Insulation Plant			
	A	B	C	D *
Count median diam. (μm)	2.9	1.2	1.5	1.8
% $\leq$ 1.0 μm	5	42	23	20
% $\leq$ 3.5 μm	61	90	85	85

* Study plant.

average total dust concentration of 0.3 mg/m^3 of air, with the other plants being very similar.

To evaluate further these industrial environments in terms of respirable fiber concentrations, airborne fiber length and diameter distributions were determined. A summary of the resulting diameter and length distributions is given in TABLES 7 and 8, respectively. The study plant had a median fiber diameter of 1.8 μm and a median fiber length of 28 μm. Again, the other three plants that produce insulation products are seen to have very similar diameter and length distributions.

These data show that respirable fiber concentrations are extremely low in the study plant. It was difficult to compute a lifetime fiber-exposure index for persons in the present study cohort, because only limited historical dust measurements were available. In an earlier report of this plant, Wright [15] included results of an unpublished environmental study done in 1963 by Kehoe, who measured average dust concentrations of 2.24 mg/m^3, with a median fiber diameter of 6 μm. Sixteen percent of the airborne fibers in the survey were less than 40 μm in length. Impinger counts averaged 0.22×10^6 particles/ft^3 of air, and less than 1% of the particles were fibrous. It thus appears that respirable fiber concentrations in the study plant were also extremely low about 10 years prior to the present industrial hygiene survey.

TABLE 8

SUMMARY OF AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES (LARGE-DIAMETER GLASS FIBERS)

Summary for All Operations Combined	Insulation Plant			
	A	B	C	D *
Count median length (μm)	52	23	39	28
% $\leq$ 5.0 μm	<2	6	3	4
% $\leq$ 50 μm	51	73	58	69

* Study plant.

Small-Diameter Fibers

Industrial hygiene surveys were also conducted in six facilities that produce or make use of small-diameter (most < 3 μm and many < 1 μm) fibers. Table 9 shows the resulting airborne fiber concentrations found in these six facilities. The concentrations in these six plants are seen to be many orders of magnitude higher than those in the plant under study, with the highest single concentration being 44 fibers/ml. Tables 10 and 11 show fiber diameter and length distributions for these six plants. A large majority of the airborne fibers are seen to be less than 0.5 μm in diameter and greater than 5 μm in length and, therefore, are much more capable of deep lung penetration than are airborne fibers in the study plant. However, because commercial production of small-diameter (< 1 μm) fibers only began in the 1960s, no facility has been in operation sufficiently long to permit evaluation of the manifestation of any potential pathogenic lung effects, including carcinogenicity, that result from inhalation and exposure to small-diameter glass fibers.

Case-Control Study

Methods

At a symposium on occupational exposure to fibrous glass sponsored by NIOSH in June 1974, the possibility was raised by officials of the plant under study that many workers at this facility may have been exposed to smaller-diameter glass fibers than are presently produced at the same facility. Further communication with the same officials [23] established that during the period 1941–1949, this facility had a flame attenuation process that produced bulk fiber, which ranged from 1 to 3 μm in diameter. This process was located in one or more of several discrete "pilot plants" that existed at the facility during that period. It appears that in 1950, the flame attenuation process was modified to produce fibers of larger diameter than had been made previously during 1941–1949.

To evaluate the potential pathogenicity of these smaller glass fibers among exposed workers without having to wait for an additional 10–20 years of prospective observation, a case-control study was deemed necessary. Consequently, for each malignant and nonmalignant respiratory disease death, or death where respiratory disease was mentioned as a contributory cause, a control was selected sequentially from an alphabetized list of the remaining study group members, matched on date of birth, plus or minus 6 months, and on race and sex. By definition, both cases and controls had been initially employed in fibrous glass production, packing, or maintenance activities during the period January 1, 1940 through December 31, 1949. Each of the 49 matched pairs were distributed according to whether they had or had not worked in at least one of the several "pilot plants" in existence at this facility. Due to the increase in fiber size after 1950, the study was limited to those who had worked in pilot plant operations other than textiles prior to January 1, 1950, when small-diameter (1–3 μm fibers were being produced. The McNemar chi-square method [24] for matched pairs was used to evaluate the significance of association between employment in pilot plant operations and respiratory disease.

TABLE 9

AIRBORNE FIBER CONCENTRATIONS (FIBERS/ML) IN OPERATIONS THAT PRODUCE OR USE SMALL-DIAMETER GLASS FIBERS

	Plant					
	Bulk Fiber Production		Paper Manufacturing		Aircraft Insulation Fabrication	
Operations	C *	E	F †	G †	H	I †
Bulk fiber handling						
Mean	1.0	9.7	5.8	21.9	1.2	14.1
Range	(0.1–1.7)	(0.9–33.6)	(4.7–6.9)	(8.9–44.1)	(0.4–3.1)	(3.2–24.4)
Number of samples	5	54	2	3	13	3
Fabrication and finishing						
Mean	—	5.3	1.9	10.6	0.8	2.1
Range	—	(0.3–14.3)	(1.6–2.1)	—	(0.2–4.4)	—
Number of samples	—	24	2	1	15	1

* In addition to the large-diameter wool insulation operations, plant C had several lines that produced small-diameter fibers.
† Only limited surveys were conducted in these facilities.

TABLE 10

AVERAGE AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS PRODUCING OR USING SMALL-DIAMETER GLASS FIBERS

Facilities	Percentage of Fibers ≤ Upper Class Interval (μm)							
	Bulk Fiber Handling				Fabrication and Finishing			
	0.5	1.0	1.9	3.8	0.5	1.0	1.9	3.8
Bulk fiber production								
Plant C	85	96	100	100	—	—	—	—
Plant E	72	88	95	98	76	93	98	100
Paper manufacturing								
Plant F	40	62	75	89	55	83	94	100
Plant G	84	96	100	100	53	86	97	100
Aircraft insulation fabrication								
Plant H	48	72	92	97	46	73	89	97
Plant I	75	91	98	99	69	92	98	100

Results

As seen in TABLE 12, the 49 respiratory disease deaths exhibited a distribution of employment different from that of the matched controls. All nine of the respiratory disease cases that were potentially exposed did not have matched controls with such exposure. In contrast, of the 40 respiratory disease cases without potential exposure, only two matched controls had potential exposure in pilot plant operations.

TABLE 11

AVERAGE AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS THAT PRODUCE OR USE SMALL-DIAMETER GLASS FIBERS

Facilities	Percentage of Fibers ≤ Upper Class Interval (μm)							
	Bulk Fiber Handling				Fabrication and Finishing			
	5	11	22	48	5	11	22	48
Bulk fiber production								
Plant C	10	20	40	80	—	—	—	—
Plant E	23	39	54	87	23	38	32	70
Paper manufacturing								
Plant F	8	27	52	73	5	22	54	86
Plant G	33	52	77	86	30	34	60	86
Aircraft insulation fabrication								
Plant H	14	31	50	94	18	37	66	91
Plant I	45	64	73	87	38	61	84	97

Although the results of this study are only of borderline significance ($0.05 < p < 0.10$), their general direction is, nevertheless, consistent with earlier conducted animal bioassays that have demonstrated pathogenicity to the lung after intrapleural and intraperitoneal studies with small-diameter glass fibers. Subsequent communication with the company under study resulted in a revised criterion for employment in flame attenuation operations based on record assessment and personal recall by company officials of the cases and controls. With this revised criterion, five of the nine respiratory disease deaths would be considered to have had employment in the flame attenuation operation, whereas the four appeared not to have had such employment. The two respiratory disease deaths without potential exposure which matched with controls so exposed satisfied the revised criterion for inclusion. Consequently, these findings have initiated a full review by NIOSH of the company records to identify, through a double-blind study, all such exposed workers prior to conducting a new retrospective analysis of a more specific population of workers, that is, those exposed to small-diameter (1–3 μm) glass fibers.

TABLE 12

RESPIRATORY DISEASE AND RELATED DEATHS FOR CASES AND MATCHED CONTROLS BY EXPOSURE ASSESSMENTS

Controls	Cases: Potentially Exposed	Cases: Not Potentially Exposed	
Not potentially exposed	9	38	47
Potentially exposed	0	2	2
Totals	9	40	49

$$\chi^2 = \frac{(|9-2|-1)}{9+2} \simeq 3.27 \quad (0.05 < p < 0.10)$$

SUMMARY AND CONCLUSIONS

Study of mortality patterns among 1,448 workers occupationally exposed to low concentrations of airborne fibers with a median diameter of 1.8 μm demonstrated a significant excess of nonmalignant respiratory disease. This excess of nonmalignant respiratory disease is consistent with an earlier report of an increased risk of chronic bronchitis as a cause of retirement disability among fibrous glass production workers. Although the exact contribution of prior employment in dusty trades other than fibrous glass production cannot be completely discounted, several cases of nonmalignant respiratory disease were determined to have had incidental employment in such trades.

No excess risk of malignant respiratory disease was demonstrated, even after 20 years since onset of exposure. However, a case-control study among the same population did demonstrate an association of borderline significance between cases of respiratory tract disease (malignant and nonmalignant) and work in pilot plant operations. Some of these operations involved a special process that emitted and created exposures to small-diameter glass fibers.

In view of the work of other investigators that demonstrated fibrogenicity and carcinogenicity of small-diameter glass fibers in laboratory animals and of the results of the present case-control study, further in-depth investigations of the potential pathogenicity (fibrogenicity and carcinogenicity) of small-diameter glass fibers is strongly indicated. In the meantime, exposures to respirable glass fibers should be assessed and should be kept at a minimum by the use of good work practices and engineering controls.

Acknowledgments

The study reported here represents the combined efforts of many individuals. The authors express their thanks to each person who contributed to the success of this undertaking. Attention should be called to the following individuals of NIOSH without whose help this study could not have been accomplished: Laura Andersen, Cheryl Blechinger, Mary Gabriel, Marian Heid, Margaret Ivory, Shirley Kinney, Dr. Joseph Mallov, Linda Sexton, Frank Stern, Gerald Tremmel, Kenneth Wallingford, Claire White, and Ralph Zumwalde.

Special mention should be made of the contribution by Mr. Bruce Gehring of the Southwest Ohio Regional Computer Center for his dedicated and sustained effort in the preparation of the computer programs under demanding time constraints.

The authors also acknowledge their indebtedness to Mrs. Clorinda Battaglia for her perseverance in tracing the whereabouts of many employees of the company.

Gratitude is extended to the staff of Owens-Corning Fiberglas Corporation for its exemplary cooperation in this study, both by joining in the collection of the data and in the tracing of former employees.

References

1. Schepers, G. W. H. 1958. The influence of fiberglas-plastic dust on tuberculosis—an experimental inhalation study of two varieties of dust: histopathologic observations. Amer. Rev. Tuberc. Pulmon. Diseases **78:** 512–523.
2. Schepers, G. W. H. 1959. The pulmonary reaction to sheet fiberglas-plastic dust. Amer. Ind. Hyg. Ass. J. **20:** 73–81.
3. Schepers, G. W. H. 1961. The pathogenicity of glass-reinforced plastics. Experimental inquiries by injection or external application techniques. Arch. Environ. Health **2:** 620–634.
4. Schepers, G. W. H. 1955. The biological action of glass wool. Studies on experimental pulmonary histopathology. A.M.A. Arch. Ind. Heatlh **12:** 280–287.
5. Schepers, G. W. H. & A. B. Delahant. 1955. An experimental study of the effects of glass wool on animal lungs. A.M.A. Arch. Ind. Health **12:** 276–279.
6. Schepers, G. W. H., T. M. Durkan, A. B. Delahant, A. J. Redlin, J. G. Schmidt, F. T. Creedon, J. W. Jacobson & D. A. Bailey. 1958. The biological action of fiberglas-plastic dust. An experimental inhalation study of the dust generated in the manufacture of automobile body parts from a commercial product with a calcium carbonate filler. A.M.A. Arch. Ind. Health **18:** 34–57.
7. Gross, P., M. Kaschak, E. B. Tolker, M. A. Fabyak & R. T. P. deTreville. 1970. The pulmonary reaction to high concentrations of fibrous glass dust. A preliminary report. Arch. Environ. Health **20:** 696–704.

8. WENZEL, M., J. WENZEL & G. IRMSCHER. 1969. The biological effects of glass fiber in animal experiment. Int. Arch. Gewerbepathol. **25:** 140–164 (in German).
9. BOTHAM, S. K. & P. F. HOLT. 1971. The development of glass-fibre bodies in the lungs of guinea pigs. J. Pathol. **103:** 149–156,
10. POTT, F. & K. H. FRIEDRICHS. 1972. Tumors in rats after intraperitoneal injection of fibrous dust. Naturwissenschaften **59:** 318 (in German).
11. DAVIS, J. M. G. 1972. The fibrogenic effects of mineral dusts injected into the pleural cavity of mice. J. Exp. Pathol. **53:** 190–201.
12. STANTON, M. F. & C. WRENCH. 1972. Mechanisms of mesothelioma induction with asbestos and fibrous glass. J. Nat. Cancer Inst. **48:** 797–821.
13. POTT, F., F. HUTH & K. H. FRIEDRICHS. 1974. Tumorigenic effect of fibrous dusts in experimental animals. Environ. Health Perspect. **9:** 313–315.
14. WAGNER, J. C., G. BERRY & J. W. SKIDMORE. 1974. Studies of the carcinogenic effect of fibre glass of different diameters following intrapleural inoculation in experimental animals. Presented at the NIOSH Symposium on Occupational Exposure to Fibrous Glass, June 26th and 27th.
15. WRIGHT, G. W. 1968. Airborne fibrous glass particles. Chest roentgenograms of persons with prolonged exposure. Arch. Environ. Health **16:** 175–181.
16. GROSS, P., J. TUMA & R. T. P. DETREVILLE. 1971. Lungs of workers exposed to fiber glass. A study of their pathologic changes and their dust content. Arch. Environ. Health **23:** 67–76.
17. UTIDJIAN, H. M. D. & R. T. P. DETREVILLE. 1970. Fibrous glass manufacturing and health: report of an epidemiological study: part I. Presented at the 35th Annual Meeting of the Industrial Health Foundation.
18. NASR, A. N. M., T. DITCHEK & P. A. SCHOLTENS. 1971. The prevalence of radiographics abnormalities in the chests of fiber glass workers. J. Occupat. Med. **13:** 371–376.
19. HILL, V. W., W. S. WHITEHEAD, U. D. CAMERON & G. A. HEDGECOCK. 1973. Glass fibers: absence of pulmonary hazard in production workers. Brit. J. Ind. Med. **30:** 174–179.
20. ENTERLINE, P. E. & V. HENDERSON. 1975. The health of retired fibrous glass workers. Arch. Environ. Health **30:** 113–116.
21. WORLD HEALTH ORGANIZATION. 1957. Manual of the Statistical Classification of Diseases, Injuries, and Causes of Death. 7th Revision. Geneva, Switzerland.
22. PEARSON, E. S. & H. O. HARTLEY. (Eds.) 1958. Biometrika Tables for Statisticians. 2nd edit. Vol. **1:** 130, 131; 203. Cambridge University Press. Cambridge, England.
23. KONZEN, J. 1974. Personal communication.
24. MAXWELL, A. E. 1969. Analysing Qualitative Data : 26–28. John Wiley & Sons, Inc. New York, N.Y.