MORTALITY PATTERNS, 1940-1975 AMONG WORKERS EMPLOYED IN AN ASBESTOS TEXTILE FRICTION AND PACKING PRODUCTS MANUFACTURING FACILITY

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INTRODUCTION

All commercial forms of asbestos tested have been found to be carcinogenic to mice, rats, hamsters and rabbits (WHO, 1977). In humans, occupational exposure to asbestos has been associated with a high incidence of respiratory tract cancer, mesothelioma of the peritoneum and pleura and an increased risk for gastrointestinal cancer (WHO, 1977). It has also been asso-ciated with a high incidence of nonmalignant respiratory disease and cardiopulmonary disease (WHO, 1977).

In particular, worker populations exposed to chrysotile asbestos have shown an increased risk for respiratory tract cancer, gastrointestinal tract cancer, nonmalignant respiratory disease and cardiopulmonary disease (WHO, 1977).

In this regard, McDonald *et al.*, (1973, 1974) reported an increased risk of lung cancer among men employed in Quebec chrysotile mines and mills. Those workers most heavily exposed showed a 5-fold increase in lung cancer deaths as compared to those least exposed. These same chrysotile miners and millers of Quebec as of 1977 had experienced 11 mesotheliomas of which 4 were from Asbestos and 7 from Thetford mines (McDonald, 1977). The diagnosis of mesothelioma in 2 of the 7 cases from Thetford mines was doubtful, but all were pleural and occurred in men employed from 18 to 53 years while the latency periods were not defined. The author concluded for the Thetford mines that "There is therefore no good reason to doubt chrysotile exposure as the cause" (McDonald, 1977). No such association could be made for Asbestos. In Italian chrysotile miners one case of mesothelioma has been reported accounting for 0.30% of all deaths as compared to the Quebec miners' and millers' experience of 0.24% of all deaths (McDonald, 1977).

In another study, Kogan *et al.*, (1972) demonstrated a 2-fold increase (over that found in the general population) in lung cancer risk for males both mining and milling chrysotile in the Urals. For workers over 50 years of age, the risk was much greater: 5-fold for male miners and 6-fold for male millers; 9-fold for female miners and 39-fold for female millers. However, the authors reported no mesotheliomas.

Wagoner *et al.*, (1973) reported in 1973 an increased risk for malignant and nonmalignant respiratory disease among a cohort of workers in a large man-

ufacturing plant utilizing predominantly chrysotile asbestos in textile, friction and packing products. At each duration of employment category down to and including one through nine years, an excess of respiratory cancer was demonstrated. Whereas the authors reported no mesotheliomas among the population, they did note the need for further investigation of the lymphatic and hematopoietic malignancies observed among these workers. As part of this research and subsequent efforts, one of the authors (RAL) of this paper has coordinated NIOSH investigations pertinent to potential health problems associated with occupational exposure to asbestos in this plant. The current study is the first of a series which were initiated on Mr. Lemen's suggestion based on his observations in these earlier research efforts.

In the same year that the paper by Wagoner *et al.* was published, Enterline and Henderson (1973) found a 2.4-fold risk of respiratory cancer for retirees who had been employed in the asbestos industry and exposed only to chrysotile. In 1972, Enterline *et al.* had reported finding only one death due to mesothelioma out of 802 death certificates for retirees age 65 and over who were exposed to amosite or mixed fibers. In contrast, a subsequent investigation by Borow *et al.* (1973) accumulated 72 cases of mesothelioma in one plant in the Enterline study where chrysotile was the main fiber used.

On the basis of these and other data the World Health Organization in 1976 concluded that in humans occupational exposure to chrysotile, amosite, anthophyllite, and mixed fibers containing crocidolite was associated with a high incidence of lung cancer. In addition, many instances of pleural and peritoneal mesothelioma were noted after exposures to crocidolite, amosite and chrysotile. As well, an excess risk of gastrointestinal tract cancers was demonstrated in groups occupationally exposed to amosite, chrysotile or mixed fibers containing crocidolite (WHO, 1977).

More recently, Weiss *et al.* (1977) suggested a favorable mortality experience over a 30 year period for a cohort of workers employed in a plant reported to be using only chrysotile. Closer examination of this study suggests several possible explanations for the favorable mortality experience of these workers. First, the study population was small (n=264) and only 66 workers had died at the time of analyses. Second, the analyses included observation periods which were less than 25 or 30 years from the onset of initial exposure. Frequently asbestos-associated disease is not manifest until after 25 years from initial exposure. Third, the age distribution of the study population is not listed; this may have had an important role in the mortality patterns of these workers. Fourth, over 50% of the workers studied were employed for less than four years, which diluted the study population at risk. However, the author concluded "while the dearth of pertinent information makes for an unsatisfactory state of affairs, the favorable experience of the cohort reported in the current study suggests that the hazard of chrysotile in asbestos products manufacturing is minimal. The results are consistent with what is known of the relative dangers of chrysotile, amosite, and crocidolite" (Weiss, 1977).

At the same time these findings were reported, NIOSH was updating its study, originally reported by Wagoner *et al.* (1973) of workers exposed to predominantly chrysotile asbestos. The purpose of reinitiating this study in 1976 was two-fold. First, the original report had a lost-to-follow-up rate exceeding 5%. Second, additional follow-up through 1975 permitted an additional eight years of observation for assessing the long latent effects associated with asbestos exposure.

PLANT DESCRIPTION

The plant under study began operations in the early 1900's and historically used three different kinds of asbestos: amosite, chrysotile, and crocidolite. Chrysotile was (and is) used in all departments in the plant involved with asbestos which include the textile, friction, and packing departments. Chrysotile constituted over 99% per year (5000-6000 tons) of the total quantity of asbestos processed, except for 3 years during World War II. During these 3 years (1942 through 1944), amosite asbestos was selectively used to a limited extent because of Naval specifications. During these 3 years, utilization of amosite increased from <1% to approximately 5% (375 tons) of the total quantity of asbestos used per year. After the war, amosite use dropped to <1%. Crocidolite asbestos was processed to a very limited extent in this plant; its usage was always less than 1% (approximately 7,500 lbs./yr.) of the total quantity of asbestos consumed, even during the war years (Weaver, Personal communication).

Of the asbestos processed at this plant 99% was chrysotile, while amosite constituted 1% with the exception of the three year period during World War II as previously mentioned. Use of crocidolite was always less than amosite, and therefore accounted for a fraction of a percent of total production (Marsh and Lewinsohn, Personal communication).

METHODS

To evaluate the mortality experiences of workers occupationally exposed to asbestos at this plant, employment histories were ascertained from company personnel files. The study cohort was restricted to workers who had achieved at least one year of employment in the plant with one day of that employment being between January 1, 1940, and December 31, 1967. No one hired for the first time after January 1, 1963, was included. The study cohort was further restricted to exclude all office workers in non-exposed areas of the plant. 3276 workers met the cohort definition.

Follow-up was made of the vital status of cohort members from the time of termination of employment or 12/31/67 (whichever came earlier) through December 31, 1975. Vital status was determined through records maintained by federal, state and local government agencies, inlcuding such sources as the Social Security Administration, state vital statistics offices, state motor vehicle registration files and state drivers license files. For individuals not located through these conventional sources, other sources such as city directories, postal service mailing correction services, voter records, and various other local records, were used. As a result of the follow-up program, less than 3% of the cohort was lost to observation (Table 1).

Death certificates were obtained for the known dead, and causes of death were interpreted by a qualified nosologist, according to the *Revision of the International Lists of Diseases and Causes of Death* in effect at the time of death, then converted into Seventh Revision codes, for purposes of standardization.

A modified life table based on Cutler-Ederer (1958) technique was used to obtain person-years at risk of dying by race and sex in 5-year calendar time periods, 5-year age groups, 5-year exposure groups, and 5-year latency group.[1] Comparison was made between the observed number of deaths in the

[1] Latency refers to the number of years since onset of initial asbestos exposure.

TABLE 1. Vital Status as of December 31, 1975 Among White Males and Females Employed Between 1/1/40 and 12/31/67 in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Vital Status	Males	Females	Total
Known to be alive	1754	409	2163
Known to be deceased	912	128	1040
Death certificates obtained	897	122	1019
Death certificates outstanding	15	6	21
Not known to be alive or deceased	56	17	73
Total	2722	554	3276

study cohort and the number expected on the basis of sex, age, race, calendar-time, cause-specific mortality rates for the total United States. Cause-specific expected deaths for the study population were obtained by applying death rates, calculated from yearly tallies of deaths and census data, to the person years of observation of the cohort members. The yearly deaths for the U.S. from 1940 to 1975 were obtained from the yearly *Vital Statistics of the United States*, National Center for Health Statistics. U.S. population estimates were obtained from the decennial data of the Bureau of Census, U.S. Department of Commerce. The mid-points of the five-year calendar periods were estimated using linear interpolation. Statistical significance (2-tailed) was determined using "Confidence Limits for the Expectation of a Poisson Variable" (Pearson and Hartley, 1958). The 73 individuals with unknown vital status were assumed to be alive as of December 31, 1975, so as not to overestimate the true risk of mortality associated with exposure to asbestos. In addition, the 21 individuals known to be deceased, but for whom no death certificates were available, were assumed to be deceased, cause unknown.

RESULTS AND DISCUSSION

Table 2 shows that a total of 912 deaths occurred among white male asbestos workers as contrasted with 741.3 deaths expected, an excess significant at $p<0.01$. For males several major cause of death categories contributed to the total excess: 1) All malignant neoplasms (168 observed, 128.7 expected $P<0.01$); 2) Diseases of the heart (375 observed, 315.4 expected, $p<0.05$); 3) Nonmalignant respiratory disease (92 observed, 38.0 expected, $p<0.01$); and 4) Suicides (30 observed, 17.1 expected, $p<0.01$). The excess of malignant neoplasms was largely contributed by respiratory tract cancer, (49 observed, 36.1 expected, $p<0.05$). The excess in heart disease may be partially explained by a review of the death certificates which showed occupationally induced respiratory disease as a direct or contributing cause of death for 29 white male asbestos workers. The excess in nonmalignant respiratory disease was entirely a function of nonmalignant respiratory disease after excluding bronchitis, acute upper respiratory infection, influenza and pneumonia, (76 observed, 16.4 expected, $p<0.01$).

Table 3 shows a total of 128 observed deaths among female asbestos workers as contrasted with 88.3 expected, an excess significant at $p<0.01$. As was previously seen for male asbestos workers, this excess was a function of several major cause of death categories: 1) All malignant neoplasms (37 observed, 22.4

TABLE 2. The Observed and Expected Deaths According to Cause Among White Males Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Cause of Death	List No.[1]	Observed	Expected	SMR
Malignant neoplasms	140-205	168	128.7	131[3]
Digestive system	150-159	50	41.4	121
Bronchogenic Cancer	162-163	49	36.1	136[2]
Other and unspecified	140-149, 156B, 161, 164, 165, 170-199, 200-205	69	51.2	135[2]
Vascular lesions affecting the central nervous system	330-334, 345	49	57.0	86
Diseases of the heart	400-443	375	315.4	119[2]
Nonmalignant respiratory disease	470-527	92	38.0	242[3]
Influenza, pneumonia, bronchitis, and acute upper respiratory infection	470-475, 480-483 490-493, 500-502	16	21.4	74
Other respiratory disease	510-527	76	16.4	463[3]
Suicides	963, 970-979	30	17.1	175[3]
Other violent deaths	800-962, 964, 980-985	45	59.3	76
All other known causes		138	125.8	110
Unknown causes		15	0	-
Total		912	741.3	123[2]

[1] 7th Revision of International Lists of Diseases and Causes of Death
[2] Significant at $p<0.05$
[3] Significant at $p<0.01$

expected, $p<0.01$); 2) Heart disease (38 observed, 30.2 expected); and 3) Nonmalignant respiratory disease (16 observed, 3.3 expected, $p<0.01$). Again, the excess in all malignant neoplasms was largely contributed by respiratory tract cancer (14 observed, 1.7 expected, $p<0.01$). The excess in nonmalignant respiratory disease was solely a function of nonmalignant respiratory disease after excluding bronchitis, acute upper respiratory infection, influenza and pneumonia (14 observed, 0.9 expected, $p<0.01$).

Nonmalignant Respiratory Disease

Table 4 shows elevated risk for nonmalignant respiratory disease throughout all four decades of latency for both males and females. This observation is consistent with other previous epidemiologic studies of asbestos workers (U.S. HEW, 1976) and with disease of occupational etiology. Upon review of death certificates,

TABLE 3. The Observed and Expected Deaths According to Cause Among White Females Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Cause of Death	List No.[1]	Observed	Expected	SMR
Malignant neoplasms	140-205	37	22.4	165[2]
Digestive system	150-159	8	6.0	133
Bronchogenic Cancer	162-163	14	1.7	824[2]
Other and unspecified	140-149, 156B, 161 164, 165, 170-199, 200-205	15	14.7	114
Vascular lesions affecting central nervous system	330-334, 345	10	10.0	100
Diseases of the heart	400-443	38	30.2	126
Nonmalignant respiratory disease	470-527	16	3.3	390[2]
Influenza, pneumonia, bronchitis, and acute upper respiratory infection	470-475, 480-483, 490-493, 500-502	2	2.4	83
Other respiratory disease	510-527	14	0.9	1555[2]
Suicide	963, 970-979	0	1.3	–
Other violent deaths	800-962, 964, 980-985	3	3.8	79
All other known causes		18	17.3	109
Unknown causes		6	0	–
Total		128	88.3	145[2]

[1] 7th Revision of International Lists of Diseases and Causes of Death
[2] Significant at $p<0.01$

TABLE 4. Observed and Expected Deaths Due to Nonmalignant Respiratory Disease (Less Bronchitis, Acute Upper Respiratory Infection, Influenza and Pneumonia) According to Time Interval Since Onset of Employment Among Whtie Males and Females Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Years Since	Males			Females		
Onset of Employment	Observed	Expected	SMR	Observed	Expected	SMR
< 10	2	1.1	181	–	.06	–
10-19	12	3.3	364	6	.14	4286
20-29	22	5.6	393	1	.32	313
≥ 30	40	6.5	615	7	.40	1750
Total	76	16.5[1]	461[2]	14	.92[1]	1522[2]

[1] Differs from Tables 2 and 3 due to rounding error.
[2] Significant at $p>0.01$

asbestosis was observed to be by far the most consistently reported cause of death. The excess risk observed for females is much greater than the excess observed for males in this study.

Suicide

Table 5 shows a significant excess in the number of deaths due to suicides by years from onset of first employment for male workers. No suicides were observed in females. An overall 1.7 fold risk of observed to expected deaths from suicide occurred in male asbestos workers. This excess followed an increasing trend through the second latency interval since onset of employment, similar to that seen for nonmalignant respiratory disease. At present an association between suicide and pre-existing asbestos-related disease cannot be ruled out; however, continuing investigations are examining medical records and coroners' reports for further information.

TABLE 5. Observed and Expected Deaths Due to Suicide According to Time Interval Since Onset of Employment Among White Males Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Years Since Onset of Employment	Observed	Expected	SMR
< 10	6	3.6	167
10-19	12	5.6	214
20-29	9	5.1	176
≥ 30	3	2.9	103
Total	30	17.2[1]	174[2]

[1] Differs from Table 2 due to rounding error.
[2] Significant at $p<0.01$

Bronchogenic Cancer

Table 6 shows a significant increase in bronchogenic cancer for both males and females. The relative risk for males is elevated throughout all 4 decades. This pattern is consistent with previous studies of workers exposed to asbestos (U.S. HEW, 1976). As noted in the 1973 report of Wagoner *et al.* the risk for females is much greater than the risk for males. This earlier study by Wagoner *et al.* showed a 2.4 fold combined male/female risk of respiratory cancer as compared to the present combined 1.6 fold risk. This difference in risk is explained in part by the significant increase in risk for mesothelioma observed in the present study (17 observed) as compared to the 1973 report (0 observed).

The true risk for bronchogenic cancer in males is probably even greater than observed, since the rate for bronchogenic cancer among U.S. males is 38.0/100,000 compared to 30.1/100,000 for Lancaster County, Pa. (Mason and McKay, 1974) the location of the asbestos plant. In addition two cross-sectional medical studies (1964 and 1971) of the active worker population (Lemen, 1979) have shown that the smoking patterns among the male asbestos workers were not significantly

TABLE 6. Observed and Expected Deaths Due to Bronchogenic Cancer (UCOD 162,163) According to Time Interval Since Onset of Employment Among White Males and Females Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Years Since	Male			Female		
Onset of Employment	Observed	Expected	SMR	Observed	Expected	SMR
< 10	3	2.5	120	1	.1	–
10-19	11	7.6	145	1	.3	–
20-29	17	12.5	136	5	.6	833
≥ 30	18	13.5	133	7	.7	1000
Total	49	36.1	136[1]	14	1.7	824[2]

[1] Significant at $p<0.05$
[2] Significant at $p<0.01$

different than the patterns observed for males in the general U.S. population during similar time periods (U.S. HEW, 1971). U.S. population patterns were used because data for states and counties do not exist (Horn, 1977). The risk for females for bronchogenic cancer in Lancaster County and the U.S. do not differ significantly nor do their smoking patterns as determined from the same sets of data as used for males.

These data are therefore inconsistant with the hypotheses that these asbestos workers smoked more than the general population and that smoking is the sole factor in the etiology of the excess bronchogenic cancer risk observed among male female workers exposed to asbestos.

Mesothelioma

In general, previous epidemiologic studies have shown a much stronger association of mesothelioma with occupational exposure to amphibole fibers, as contrasted to occupational exposure to predominantly chrysotile. Those reported mesotheliomas associated with predominantly chrysotile were mostly pleural in origin rather than peritoneal (McDonald, 1977; Borow *et al.*, 1973; McDonald *et al.*, 1970). Table 7 lists 17 cases of mesothelioma which occurred among this asbestos workers study cohort. As may be seen, their distribution by site was 5 pleural, 6 peritoneal, and 6 with site unstated. A review of death certificates and cinical records indicated the presence of pre-existing asbestosis in 3 of 5 pleural mesothelioma cases and in 5 of 6 peritoneal mesothelioma cases among members of the study cohort.

Whereas case-series of mesotheliomas in the general population revealed a sex ratio of about 3:1 in favor of males; in fact, when analyses are made on a population base of workers exposed to asbestos, no differential risk of mesothelioma is seen. That this is so is evidenced by a rate of 2.7/10,000 for female asbestos workers and 1.9/10,000 for male asbestos workers in the present study.

Among a number of occupationally exposed groups previously studied, approximately 5-10% of deaths were due to mesothelioma (Gilson, 1973; Hammond and Selikoff, 1973; Selikoff, 1976). More recently, however, an estimate projected that the deaths of between 8-11% of former asbestos workers from a particular factory in the U.K. will be due to mesothelioma (Newhouse and Berry, 1976). Among asbestos workers in the present study, 4.3% of deaths were due to mesothelioma.

TABLE 7. Deaths Due to Mesothelioma Among White Males and Females Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Case Number	Asbestosis	Type of Mesothelioma	Sex	No. of Years of Employment	No. of Years Since Initial Employment	Age at Death
1	Yes	Pleural	M	40	43	68
2	Not Stated		F	9	39	70
3	Yes		M	42	47	67
4	No Records		M	47	48	65
5	Yes		M	28	31	53
6	Yes	Peritoneal	M	16	39	54
7	Yes		M	41	50	65
8	Yes		F	14	47	74
9	Yes		M	35	35	51
10	Yes		M	18	24	63
11	No Records		M	23	43	63
12	Not Stated	Unstated Site	F	7	41	59
13	Not Stated		M	33	43	67
14	Yes		M	5	38	68
15	No records		F	6	38	65
16	Not Stated		M	47	53	70
17	Yes		M	21	32	50

Lymphoma and Lymphosarcoma

Several reports have suggested a possible association of asbestos exposure and/or asbestosis with various immunoproliferative and lymphoproliferative diseases including lymphoid tumors (Kagan *et al.*, 1977; Gerber, 1970; Lieben, 1966). Therefore, analyses were undertaken to evaluate the risk of lymphoid cancer among male asbestos workers during the interval 1950-1975. Because deaths due to malignancies of the lymphatic and hematopoietic systems were not listed in U.S. Vital Statistics prior to 1950, these analyses were restricted to risk since that date. As seen in Table 8, seven deaths due to lymphosarcoma or malignant lymphomas were observed among male asbestos workers, whereas only 3.28 were expected on the basis of age-specific rates for all causes of death in the category of lymphosarcoma and reticulosarcoma (ICD 200). No cancers of this category occurred among female workers.

TABLE 8. Deaths Due to Lymphosarcoma Among White Males Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Case	Age	Underlying Cause of Death	No. of Years Since Initial Employment	No. of Years of Employment
1	25	Lymphosarcoma	4	1
2	56	Lymphosarcoma	24	4
3	29	Lymphosarcoma	13	2
4	55	Lymphosarcoma	39	35
5	58	Malignant Lymphoma	17	17
6	34	Malignant Lymphoma	7	5
7	54	Malignant Lymphoma	38	34

7 Observed vs. 3.28 expected, SMR 213

CONCLUSION

A study among a group of textile, friction and packing products asbestos production workers exposed to predominantly chrysotile asbestos demonstrated an excess risk for nonmalignant respiratory disease, suicide, heart disease, bronchogenic cancer and mesothelioma of the pleura and peritoneum among males; and an excess risk for nonmalignant respiratory disease, bronchogenic cancer and mesothelioma of the pleura and peritoneum among females. An increased risk due to lymphoma and lymphosarcoma among male asbestos workers was also suggested. These findings once again lend support to the conclusion that occupational exposure to predominantly chrysotile asbestos poses a carcinogenic risk for malignancies of multiple sites.

TABLE 9. Age Distribution by Person Years for White Males Employed in an Asbestos Textile, Friction, and Packing Products Manufacturing Facility

Ages	Person-Years
15-19	299.04
20-24	3,063.78
25-29	6,195.25
30-34	8,192.92
35-39	9,078.32
40-44	9,169.24
45-49	8,628.53
50-54	7,617.50
55-59	5,848.93
60-64	2,822.63
65-69	2,381.26
70-74	1,343.34
75-79	709.46
80-84	301.93
85+	91.91
Total	66,755.05

TABLE 10. Age Distribution by Person Years for White Females Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Ages	Person-Years
15-19	88.70
20-24	745.00
25-59	1,297.74
30-34	1,738.24
35-39	1,982.35
40-44	2,069.23
45-49	1,961.73
50-54	1.678.37
55×59	1,307.84
60-64	940.42
65-69	539.54
70-74	280.00
75-79	120.18
80-84	50.60
85+	43.37
Total	14,843.31

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