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# Mortality of a Cohort Exposed To Chrysotile Asbestos

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A 30-year historical cohort mortality study was made of 264 men hired during 1935-45 who worked in a chrysotile asbestos products factory for one year or more and were alive January 1, 1945. Follow-up was 94% complete. The overall standardized mortality ratio (SMR) was only 0.61 while the SMR for all cancers was 0.75, for lung cancer 0.93, and for gastrointestinal cancer 1.05. Two men died of asbestosis. The overall SMR was higher for men who worked five years or more than for men who worked one to four years but the 30-year mortality rates were the same after age adjustment. For asbestos-related diseases the differences in work duration had no effect on mortality. The data show a favorable mortality experience for men exposed to chrysotile alone.

That asbestos is a serious health hazard has been well established but the influence of fiber type and kind of exposure has not been adequately delineated.[1] On a human level the study of these factors has been difficult because exposures to a single fiber type are rare and usually limited to mining. Most workers manufacturing asbestos products have been exposed to a mixture of fiber types, however, the information that is available suggests that chrysotile is not as dangerous as crocidolite.[2] While it is difficult to draw conclusions from comparisons between studies of different populations in which exposure conditions are not exactly the same something may be learned by recording observations of occupational cohorts exposed to pure forms of asbestos with the hope that confounding variables may be kept to a minimum.

Selikoff et al[3,4] have reported observations of mortality in a cohort of men employed in an amosite asbestos factory. The opportunity to do a similar study of mortality in a cohort of men employed in a factory using only chrysotile asbestos provided data for this report.

## Method

An asbestos manufacturing plant opened in 1896 has used only chrysotile asbestos throughout its history. The employee records for the period 1935-45 were reviewed. There were 332 male production workers who worked one year or more in this period; 267 were first hired during the period 1935-45 and three of these men died before January 1, 1945. Thus, there were 264 men who were first hired in 1935-45, worked at least one year, and were alive on January 1, 1945; this was the cohort for the study. Its age distribution in 1945 was: under 20, 6%; 20-29, 50%; 30-39, 20%; 40-49, 9%; 50-59, 10%; 60 and over, 6%. The distribution of work durations from hire was: 1-4 years, 56%; 5-9, 16%; 10-19, 11%; 20 or more, 17%.

Deaths were determined through the Social Security Administration as of December 31, 1974. Vital status at that time was known for 94% of the total group. There were 16 men whose social security records revealed no information for variable periods prior to December 31, 1974; these men were assumed to be living throughout the follow-up period. There were 66 deaths and death certificates were obtained for all but four. No smoking information was available. Causes of death were coded by the author.

The number of man-years of observation was computed for each five-year period from 1945 through 1974 for each of two work duration groups: 1-4 years and 5 years or more. The number of observed deaths was compared with the number expected and the standardized mortality ratio (SMR) calculated. The expected number of deaths was obtained by applying the mortality rates of U.S. white males (almost all of the workers were white) for the middle year of each five-year period by five-year age groups to the man-years in each cell and cumulating the results. The statistical significance of differences between observed and expected deaths was estimated from the Poisson distribution.[5]

From 1935 to 1960 the plant produced chrysotile asbestos papers and millboard, bound with starch or occasionally Portland cement. The fiber was received from the mine in loosely packed jute bags. Rail cars were unloaded by hand and bags stored in a warehouse. Stock was prepared in beaters, each bag being emptied into the tub, the bag shaken to recover as much fiber as possible, and stacked for later return to the mine or sale. Stock was formed on multi-cylinder wet machines. Finished paper was converted to rolls, corrugated paper, corrugated sheets or blocks, or aircell pipe insulation. Paper products and millboard were cut by

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saws with limited ventilation. Loose fiber was cleared by airhose. Fiber could be seen in the air throughout the plant except in the finished goods warehouse, boiler house, and maintenance shop. Fiber and dust were visible on the floor, work surfaces, and interior structural surfaces. Cleaning was done by dry sweeping. At the end of each shift employees used an airhose to remove fiber from hair and clothing. Work clothes were usually worn home.

After 1960 the mine began to compress fiber into blocks shipped in paper bags, and dust in fiber handling was much reduced. The principal stock preparation unit was changed from a beater to a pulper. The paper bags were slit at the edge of the tub and the block broken into chunks which were dropped into the pulper. For certain products the bag was pushed unbroken into the pulper. More and more products were bound with synthetic latices and resins, producing less dusty items. Converting operations declined. Production of aircell pipe insulation ceased in 1968 and millboard production was discontinued. All saws were removed. As a result, no dust is visible in the air, on surfaces, or on clothing. Plant floors are cleaned by vacuum or wet sweeping. Most employees change clothing and shower at the end of the work shift.

Fiber counts were not made until 1972, so that no exposure information of a quantitative nature is available for the years when dust concentrations were obviously high. Fiber counts in recent years have been generally under 2/cm³. Use of an exposure scale was not feasible from the available records.

## Results

The duration of follow-up from the date of hire was 30-39 years. In the last decade of observation 45% of the men had attained the age of 50 or over. Twenty-five percent of the men died (Table 1). The group that worked 5+ years had a crude rate which was about 50% higher than that of the men who worked less than five years but this was due to a difference in age distribution. Using the total group as the standard population, the age-adjusted rates were the same.

Table 2 gives the number of man-years and Table 3 observed and expected deaths as well as SMRs. The overall SMR was only 0.61. During the first 10-year period it was 0.56 and rose to 0.62 for the remaining two 10-year periods. The SMR for the men who worked five or more years was higher than that for men who worked less than five years but both were statistically significantly lower than 1.00. The difference between the two groups was large during the first 20 years but almost disappeared in the last ten years.

Cause-specific SMRs for diseases of interest are given in Table 4 for the 30-year period. The number of deaths for each cause were too small to provide reliable ratios in each 10-year period. The SMR for all cancers was slightly higher than the all causes SMR but the SMRs for lung cancer and gastrointestinal cancer were close to 1.00, relatively higher than the all causes SMR. There were ten deaths attributed to severe pulmonary fibrosis on the death certificates; for the purpose of this analysis both were categorized as due to asbestosis. Since asbestosis does not occur in the absence of exposure to asbestos, SMRs could not be calcualted.

Table 5 shows asbestos exposure periods, date of death, age at death, and cause of death in ten men who died of diseases which might have been related to the work exposure at the plant. Although the duration of exposure was short (one year) in one case of lung cancer and the elapsed time from onset of exposure to death was short (17 years) in another, asbestos could have been a factor in the causation of all four cases of lung cancer. In one of the two cases of gastric cancer, the elapsed time from onset of exposure to death (four years) makes asbestos unlikely as a cause and the same might be true of one of the two cases of colon cancer in which there was only 12 years between the onset of exposure and death. However, information is lacking on whether there was asbestos exposure elsewhere before or after the time worked at this particular plant, making the total duration of exposure in these cases uncertain.

## Discussion

Methodologically, it should be emphasized that this 1935-45 cohort was established with men who began work in 1935-45 to avoid bias. If men who started work before 1935 and were working in this period had been included, this would not have taken into account those men who might have left the company for reasons of illness prior to the period in which the cohort was defined.

Although Wagner et al[5] are of the opinion that chrysotile is the least dangerous form of asbestos with respect to adverse health effects, the evidence is very scanty and there are a number of methodologic problems in developing a well-founded judgment among the cohort studies in the literature.

A minor problem in almost all investigations of this nature is exemplified by the current study: 6% incomplete follow-up and 6% failure to obtain death certificates to allocate cause of death. However, these deficiencies are so small that even if all the individuals whose vital status was unknown at the end of 1974 had died and all the deaths for which certificates were unobtained among the known deaths were due to asbestos-related disease the conclusions would not change.

More serious problems are inherent in differences between and deficiencies in studies reported in the literature. These make comparisons difficult. Ideally one would like to have data on large groups of workers exposed to different forms of asbestos under conditions in which all other factors related to the host, the environment, and the agent are held constant. Such utopian conditions do not exist, however.

Perhaps the most important problem lies in confounding the factor of asbestos type by the factor of work conditions. It is

**Table 1. — 30-Year Mortality Rates.**
**In a Cohort of 264 Men from 1945 through 1974 by Number of Years Worked at a Chrysotile Asbestos Manufacturing Plant.**

| No. of Years Worked | No. at Risk | Deaths | | |
|---|---|---|---|---|
| | | No. | Crude % | Age-Adjusted % |
| 1-4 | 148 | 31 | 20.9 | 24.2 |
| 5+ | 116 | 35 | 30.2 | 25.4 |
| Total | 264 | 66 | 25.0 | |

**Table 2. — Number of Men and Man-Years of Observation.**
**In a Cohort of 264 Men from 1945 through 1974 by Number of Years Worked at a Chrysotile Asbestos Manufacturing Plant in 10-year Periods.**

| No. of Years Worked | No. of Men Jan. 1, 1945 | No. of Man-Years | | | |
|---|---|---|---|---|---|
| | | 1945-54 | 1955-64 | 1965-74 | Total |
| 1-4 | 148 | 1,456 | 1,399 | 1,276 | 4,131 |
| 5+ | 116 | 1,127 | 1,022 | 871 | 3,020 |
| Total | 264 | 2,583 | 2,421 | 2,147 | 7,151 |



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Table 3. — Standardized Mortality Ratios (All Causes).
In a Cohort of 264 Men from 1945 through 1974 by Number of Years Worked at a Chrysotile Asbestos Manufacturing Plant in 10-Year Periods.

| No. of Years Worked | Period 1945-54 Obs. | 1945-54 Exp.* | 1945-54 SMR | 1955-64 Obs. | 1955-64 Exp.* | 1955-64 SMR | 1965-74 Obs. | 1965-74 Exp.* | 1965-74 SMR | Total Obs. | Total Exp.* | Total SMR |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1-4 | 5 | 12.20 | 0.41 | 9 | 19.72 | 0.46 | 17 | 28.76 | 0.59 | 31 | 60.68 | 0.51† |
| 5+ | 8 | 11.19 | 0.71 | 13 | 15.71 | 0.83 | 14 | 21.14 | 0.66 | 35 | 48.04 | 0.73‡ |
| Total | 13 | 23.39 | 0.56 | 22 | 35.46 | 0.62 | 31 | 49.91 | 0.62 | 66 | 108.76 | 0.61 |

*Expected number of deaths derived from U.S. white male mortality statistics by age and period
†$P < 0.001$
‡$P < 0.05$

Table 4. — Standardized Mortality Ratios for Asbestos-Related Diseases.
In a Cohort of 264 Men from 1945 through 1974 by Number of Years Worked at a Chrysotile Asbestos Manufacturing Plant.

| Cause of Death and No. of Years Worked | Period 1945-54 Obs. | 1945-54 Exp. | 1955-64 Obs. | 1955-64 Exp. | 1965-74 Obs. | 1965-74 Exp. | Total Obs. | Total Exp. | Total SMR |
|---|---|---|---|---|---|---|---|---|---|
| All cancer | 3 | 3.23 | 5 | 5.53 | 5 | 8.66 | 13 | 17.41 | 0.75 |
| 1-4 | 2 | 1.56 | 2 | 2.87 | 3 | 4.69 | 7 | 9.11 | 0.77 |
| 5+ | 1 | 1.67 | 3 | 2.66 | 2 | 3.97 | 6 | 8.30 | 0.72 |
| Lung cancer | 0 | 0.52 | 3 | 1.23 | 1 | 2.57 | 4 | 4.32 | 0.93 |
| 1-4 | 0 | 0.22 | 2 | 0.58 | 0 | 1.36 | 2 | 2.16 | 0.93 |
| 5+ | 0 | 0.30 | 1 | 0.65 | 1 | 1.21 | 2 | 2.16 | 0.93 |
| GI cancer | 2 | 0.95 | 2 | 1.27 | 0 | 1.61 | 4 | 3.82 | 1.05 |
| 1-4 | 2 | 0.46 | 0 | 0.67 | 0 | 0.87 | 2 | 2.00 | 1.00 |
| 5+ | 0 | 0.49 | 2 | 0.60 | 0 | 0.74 | 2 | 1.82 | 1.10 |
| Asbestosis | 0 | — | 1 | — | 1 | — | 2 | — | — |
| 1-4 | 0 | — | 1 | — | 1 | — | 2 | — | — |
| 5+ | 0 | — | 0 | — | 0 | — | 0 | — | — |

Note: Expected number of deaths derived from U.S. white male mortality statistics by age and period

probably not justifiable to equate exposure conditions of mining with those in manufacturing or those in use of asbestos products. Even in manufacturing, conditions vary. Enterline and Kendrick[7] studied three cohorts in asbestos manufacturing operations of different types: building products, friction materials, and textiles. The risks of death from asbestosis increased in the order given. In comparison with U.S. white male mortality rates in the same period, the SMR for all causes was elevated only in textile workers and this cohort experienced the highest SMRs for lung cancer, digestive tract cancer and chronic respiratory disease. Unfortunately the cohorts were composed of people with varying durations of exposure prior to registration and follow-up was limited to 16.5 years after registration. The duration of exposure and of follow-up are both important risk factors.[4,8,9]

Additional methodologic discrepancies between studies account for some of the wide variation in mortality risks among asbestos workers. These include differences in the populations used to calculate the expected number of deaths, and limiting correction of causes of death on death certificates by means of additional medical information to the study population without similar correction of the certificates of the reference population. Recently Enterline[10] adjusted the results of six studies for differences in length of follow-up, the reference populations, and correction of death certificates, and found that much of the variation disappeared in the SMRs.

A very serious problem is the lack of dose information in most studies. There are few investigators (for example, Newhouse,[6] Enterline et al.[9] and McDonald et al[11]) who have attempted to develop data for degree of exposure and some of the effort is based on semiquantitative estimates rather than quantitative measurements. Changing processes over time may have a profound effect on dose, making for additional uncertainty in the absence of systematic determination of dust or fiber concentrations.

Finally, seldom are the results of investigations corrected for variations in smoking habits, although smoking is now known to be an important risk factor. Selikoff et al[12] and Berry et al[13] have shown the importance of cigarette smoking in the risk of lung cancer among asbestos workers and Weiss[14] has reported its influence in the prevalence of asbestosis among textile workers.

Despite all these difficulties, there is a little information suggesting that chrysotile asbestos may not be as hazardous as other forms of asbestos or mixtures of chrysotile with amosite or

Table 5. — Exposure of Men whose Deaths might have been Due to Asbestos.

| Case No. | Asbestos Exposure Began | Asbestos Exposure Years | Death Cause | Death Age | Years from First Exposure |
|---|---|---|---|---|---|
| 1 | Oct. 1, 1936 | 1 | lung cancer | 62 | 24 |
| 2 | Sept. 14, 1936 | 4 | lung cancer | 48 | 22 |
| 3 | Oct. 17, 1939 | 10 | lung cancer | 51 | 31 |
| 4 | Oct. 2, 1939 | 17 | lung cancer | 69 | 17 |
| 5 | Nov. 24, 1942 | 4 | gastric cancer | 62 | 4 |
| 6 | Oct. 26, 1942 | 5 | gastric cancer | 85 | 20 |
| 7 | Jan. 15, 1940 | 1 | colon cancer | 56 | 12 |
| 8 | Nov. 18, 1943 | 8 | colon cancer | 68 | 20 |
| 9 | Oct. 1, 1936 | 1 | pulmonary fibrosis | 53 | 28 |
| 10 | Sept. 7, 1937 | 4 | pulmonary fibrosis | 62 | 32 |



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crocidolite, or both. Data from the Quebec chrysotile mines and mills have shown minimal influence on mortality and the effect is limited to heavily exposed people.

A better comparison with the present study is an investigation by Elwood and Cochrane[15] who studied 1,165 men from 1936 to 1962. These workers were exposed only to chrysotile, making asbestos sheeting for six months or more. In the analysis of men who were followed 15 or more years after first exposure, the overall SMR was 0.96, that for all neoplasms was 1.10, and that for pleural and pulmonary cancer was 2.32. Unfortunately the majority of workers were followed for little more than 15 years. Nevertheless, the risk of respiratory cancer was much less than the SMR of 15.38 recorded 10-14 years after first exposure by Selikoff, Hammond, and Seidman among 326 insulation product workers who were exposed one year or more to amosite.[4]

Enterline and Henderson[2] provided valuable comparisons with regard to type of asbestos and mortality in 1,348 retirees. The lung cancer SMR for workers exposed to chrysotile alone was 2.4, to amosite alone 3.9, and to chrysotile plus crocidolite 5.6. In the cement industry they were able to separate those who were exposed to chrysotile from those exposed to chrysotile plus crocidolite; the SMR was only 1.4 in the former and 6.1 in the latter.

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.

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## Unemployment and Health

. . . A study analyzing the effects of the economy on social problems was recently completed. It is entitled "Estimating the Social Costs of National Economic Policy: Implications for Mental and Physical Health, and Criminal Aggression" by Dr. Harvey Brenner of Johns Hopkins University . . . .

According to Dr. Brenner's study, a 1.4% increase (about 1-1/2 million people) in the number of unemployed, sustained over the six-year period between 1970 and 1975, resulted in 20,240 heart and kidney disease deaths, 495 liver cirrhosis deaths, 920 suicides, and 648 homicides. In all, the study cited that there were 36,887 deaths (27% were of blacks) from various causes. In addition, there were 4,227 state mental hospital first admissions and 3,340 state prison admissions . . . .

We must make it clear that unemployment rates . . . are not just isolated, meaningless, theoretical abstracts, but that they indicate a reality of human stress, illness, and death. Full employment, therefore, is a healthy concept for our society. More people would live healthier and longer lives, during which they could do more work longer, that in turn would beef up the economy so that more people could work.

— From Editorial by Theman E. Evans, M.D., in *Journal of the American Medical Association* May 2, 1977



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