FINAL

Proportion of Cancer Due to Exposure to Asbestos*

by

Philip E. Enterline**

If the current level of cancer mortality continues nearly 50 million persons alive today in the United States will die of cancer. This prediction could be modified by the discovery of better methods of treatment, or by altering known cancer causing exposure patterns. Since resources are finite it is important to allocate these so as to achieve greatest impact on the cancer problem. For this reason it would be useful to know the proportion of cancer due to various environmental agents. This kind of effort has been greatly stimulated by a widely quoted document, sometimes referred to as the "estimates" document, that concludes that 13-18% of all cancer is caused by a single substance--asbestos (Eridbord et al. 1978). There has been disagreement with this estimate (Higginson et al. 1980; Peto 1980; Hogan and Hoel 1981).

Current Approaches

One approach to the question of how much cancer is caused by asbestos is to find a well reported disease clearly caused by asbestos exposure and from epidemiologic investigations calculate the ratio of this disease to all cancer caused by asbestos. Higginson, for example, has noted that there are about 1,000

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**Professor of Biostatistics, Graduate School of Public Health University of Pittsburgh, Pittsburgh, PA 15261.

annual mesotheliomas in the United States, and that in a long term mortality study of heavily exposed asbestos workers for every one mesothelioma there were about 1.5 bronchogenic cancers related to asbestos (Higginson et al. 1980). From this he estimated the total annual incidence of asbestos caused bronchogenic cancer in the United States to be about 1,500. This makes up 2% of the approximately 70,000 such deaths each year. Higginson's report leads to an estimate of 4,000 asbestos caused cancers in the U.S. each year (including mesotheliomas) or 1% of the total 400,000 cancer deaths that occur annually.

Another approach is to estimate the number of asbestos exposed workers and based on epidemiologic studies of subsets of this or other populations, predict the proportion and numbers of these asbestos exposed workers who will develop cancer as the result of asbestos exposure. This is the approach used by the group that produced the "estimates" document. In that study it was estimated that 4 million workers alive today have had a heavy asbestos exposure like asbestos workers, and based on data from a study of asbestos insulators, it was calculated that 1.6 million or 40% of these are expected to die of asbestos related cancers. It was estimated, in addition, that 1.5-3.5 million workers have had lesser exposures and .4 to .7 million of these will die of asbestos related cancers. Thus, over the next 30-35 years the expected number of cancer deaths associated with asbestos is between 58,000 to 75,000 per year or 13-18% of the 400,000 to 450,000 cancer deaths that occur each year.

Hogan and Hoel have used essentially the same approach followed in the "estimates" document but with different results (Hogan and

Hoel, 1981). They estimated that 7-8 million workers were potentially exposed to asbestos during the years 1940-67 of which 50% are alive today. Of these 15% were heavily exposed and will have cancer mortality rates like asbestos insulators, while the remainder will have 1/8 to 1/4 this cancer risk. On this basis they estimate that future excess cancer deaths due to asbestos exposure will contribute 3% of all cancer deaths (range 1.4-4.4%).

Heavily Exposed Workers

In thinking about the problem of asbestos caused cancer it helps to consider the magnitude of another health problem related to asbestos-asbestosis. Asbestosis was seen in asbestos workers long before cancer, and in fact it was the high frequency of coexisting asbestosis and lung cancer that led to the conclusion that asbestos was a cause of lung cancer. In the past many felt that asbestosis was a prerequisite for asbestos lung cancer and that prevention of asbestosis would also prevent lung cancer (Eckardt 1959). It is partly for this reason that the present TLV for asbestos is based on preventing asbestosis rather than lung cancer. Moreover, many also feel that only very heavy asbestos exposure is likely to lead to an asbestosis death and that the presence of asbestosis at death is important since it indicates the heavy exposure needed to produce asbestos lung cancer (Wagner 1965).

Table 1 shows asbestosis deaths observed in the United States during the period 1950-77 by year, sex and race. Clearly the trend is upward until 1972 when numbers of deaths seem to have

leveled off. It is important to note that all asbestosis deaths observed in epidemiologic studies conducted in the United States are included in the numbers shown in Table 1. It is possible for each study, then, to see how its asbestosis deaths related to the U.S. total and thus estimate the size of the population which produces all asbestosis deaths in the entire United States.

One estimate can be made from a study we conducted in the early 1960's. In that study we identified 21,755 male white workers from 35 of the larger U.S. asbestos products plants (SIC 3292) where we can be fairly certain exposures were high (Enterline and Kendrick, 1967). These workers had all been first employed sometime during the years 1948-51. During the years 1950-63 at ages under 65 there were 44 deaths from asbestosis.* During the same period it can be seen from Table 1 that for the entire United States the number of deaths from asbestosis at all ages among white males was 184. Tabulations of asbestosis deaths by age are available for the years 1962-77. During that period 52% of all asbestos deaths were at ages under 65. From this it can be estimated that there were 96 deaths from asbestosis at ages under 65 during the years 1950-63. Thus, this population of 21,755 workers produced 45.8 percent of the asbestosis deaths that occurred in the U.S. among white males at ages under 65 during the years 1950-63. From this it can easily be calculated that the total number of white males producing asbestosis deaths at ages under 65 (exposed like asbestos products workers) must have been equivalent to 47,465. Since the ratio

*Only deaths at ages under 65 were examined in this study.

of all deaths in Table 1 to white male deaths is 1.23 for the years 1950-63 this suggests a total population producing asbestosis of 58,381. In another study, of 1075 retired male asbestos products workers from a single asbestos company at ages 65 and over, we observed 17 asbestosis deaths during the period 1950-73 (Henderson and Enterline, 1979). During the same period in the entire U.S. it can be calculated from Table 1 that there were 515 male deaths due to asbestosis of which an estimated 48% or 248 were at ages 65 and over. Thus, the total population at ages 65 and over producing asbestosis deaths must have been 15,683. Again adjusting to the total population this becomes 17,054. The total at all ages, therefore, must have been 75,435.

Another estimate can be made from a large study by Selikoff and his co-workers of 17,800 male asbestos insulators from the United States and Canada (Selikoff et al. 1980). These workers were observed for deaths over the years 1967-76 and 78 deaths were coded from death certificates as due to asbestosis. It's not reported how many of these were Canadian workers, but perhaps about 10% so that we can estimate asbestosis deaths among U.S. asbestos insulators at about 70. Table 1 shows that during the same period in the entire U.S. there were 371 male deaths from asbestosis. Thus, from these data we can estimate that during the years 1967-76 these asbestos insulators produced 18.9% of all male asbestos deaths. The total U.S. male population producing asbestosis deaths (exposed like asbestos insulators) was, therefore, equivalent to 84,906. This is somewhat higher than the estimate made on the basis of asbestos products workers, but since it relates to a later

period (1967-76) it may reflect the presence of a larger heavily exposed population. These two study populations produced over half of all the concurrent asbestosis deaths in the United States and point clearly to the importance of asbestos insulators and asbestos products workers in estimating heavily exposed populations. That asbestos insulators and asbestos products workers are the primary source of asbestosis deaths is confirmed by individual case reports which almost entirely represent occupations of asbestos insulator and asbestos products worker (McVittie 1965).

Actual numbers of asbestos insulators and asbestos products workers are published regularly by the U.S. Government. Table 2 shows the number of workers employed by asbestos products companies in the U.S. (SIC 3292) for the years 1947-76, the membership of the International Association of Heat and Frost Insulators and Asbestos Workers, and asbestos and insulation workers reported in each decennial U.S. census. The number of insulators increased steadily during the period 1950-76 from around 15 to 25 thousand. On the other hand the number of workers employed by asbestos products producers stayed at around 20,000 during the 1950's and 1960's but dropped rather sharply in the 1970's.* The period of interest is the period prior to 1970 since after that time increased awareness of the health hazards associated with asbestos probably greatly decreased the cancer risk.

In addition to workers in asbestos products industries (SIC 3292), there are workers making asbestos paper included in a larger category "Building paper and building board mills" (SIC 2611) and asbestos exposed workers in a category "Steam and other

*In our 1967 study we identified 12,707 white males working in the first quarter of 1948, so our cohort apparently contained over half of all workers employed by asbestos products companies in 1948.

packing, and pipe and boiler covering" (SIC 3293). A 1976 study by Weston Environmental Consultants places the total employment in primary asbestos products industries at 37,539 of which an estimated 22,670 are exposed to asbestos.

How many asbestos products workers and asbestos insulators employed during the period 1940-70 are alive now in 1981? From data in Table 2 it appears that on average during the period 1947-70 there might have been about 40,000 such workers in the United States. Our experience in carrying out historical prospective studies of working populations is that over a 30 year period the number of different workers with a year or more work experience is about 3 times the average daily worker census. Thus, I would estimate that an average census of 40,000 asbestos products workers and asbestos insulators over the period 1940-70 represents 120,000 different workers with a year or more work experience. Of course, all these workers would not be alive today. Assuming a death rate of 1% per year, somewhat higher than the U.S. death rate but similar to the annual death rate observed in the study of 17,800 asbestos insulators, and a mid-point of 1955 we would expect about 85% or about 100,000 to be alive in 1970 and, if no new heavily exposed workers have been added since that time, somewhat less in 1981 -- say about 90,000. These estimates are somewhat higher than those made on the basis of asbestosis deaths, but indicate that certainly less than 100,000 workers and former workers are alive today who had exposures to asbestos like asbestos insulators and asbestos products workers.

I would estimate that as of 1981 there are between 70 and 90 thousand persons alive in the U.S. who have had heavy asbestos

exposure--mostly present and former asbestos insulators and asbestos products workers.* This is very low in contrast to estimates made by others, but is in line with an estimate of 20,000 "high risk" workers in the United Kingdom, where the population is about a quarter as large as the U.S. population (Smither 1979).

Assuming a best estimate of 80,000 heavily exposed or high risk workers alive in 1981 how many of these will die of asbestos lung cancer? Since we have actual epidemiologic data on both asbestos products workers and asbestos insulators this is not difficult to estimate. If data from the cohort of 17,500 asbestos insulators is used to predict, it appears that after 20 years from first exposure 22% will die of asbestos lung cancer (Selikoff et al. 1980).** If data from cohorts of asbestos products workers were used this percentage would be somewhat lower. Among retired asbestos products workers from the Johns Manville Corporation about 14% died of asbestos lung cancer, while in a study of workers from a British asbestos textile plant 15% died from asbestos lung cancer (Henderson and Enterline 1979; Peto et al. 1977). If we can assume 20% is a good overall estimate for the entire

*I had earlier estimated somewhere between 43,000 and 1,700,000 heavily exposed workers alive in 1967, with a best guess of 250,000 (Hogan and Hoel 1981). The reason for the best guess of 250,000 was that at that time death certificate data on the number of deaths among the 17,800 asbestos insulators had not been reported and I assumed that death certificates showed far fewer than 78 asbestosis deaths.

**This is calculated as:

$$\text{Percent excess} = \frac{\text{Observed Lung Cancer} - \text{Expected Lung Cancer}}{\text{Expected Deaths from All Causes}}$$

cohort then the number of asbestos lung cancers which will appear in this group of 80,000 is 16,000. If this cohort live just 30 more years asbestos lung cancer deaths will occur at a rate of 530 per year.

Less Than Heavily Exposed Workers

It was Hueper's view that heavy exposure to asbestos dust produced asbestosis, whereas lower levels permitted lung cancer to develop (Heuper 1955). He was undoubtedly right, and the cancer problem is not confined to heavily exposed workers, but rather includes those more lightly exposed - so that, there is a population of exposed workers that does not experience many asbestosis deaths, but which does have an increased cancer risk. Populations most frequently mentioned are shipyard workers, construction workers and automotive brake mechanics.

World War II Shipyard Workers

Hogan and Hoel (1981) have estimated that during World War II there were 4.3-5.4 million shipyard workers exposed to asbestos and that perhaps 50% of these or about 2.5 million are alive today. The only data that appear to apply to the probable lung cancer excess in these shipyard workers are those published by Blot et al. (1978, 1980). In two case-control studies involving interviews of lung cancer cases and a matched control group relative risks for lung cancer among World War II shipyard workers of 1.6 and 1.7 were calculated.

In view of the fact that World War II shipyard and employment must have been fairly brief and, for occupations other than asbestos insulators, exposure levels must have been relatively low these risks are surprisingly high. From labor turnovers rates it appears that the average duration of employment in the shipyard was about a year (Selikoff et al. 1979). Moreover, shipyards only operated at high employment levels for 2 or 3 years. Under these conditions the entire cohort would have had to be exposed to very high levels of asbestos to produce relative risks of 1.6 and 1.7.* As Blot et al. point out, however, the comparison of occupational histories between cases and controls may have been effected by a lower interview response rate among controls and by publicity surrounding the issue of asbestos and cancer. The second point needs emphasis in light of a report by Nigerian and Colton (1979) of a five-fold excess in leukemia among shipyard workers exposed to ionizing radiation based on a similar kind of case-control study. Subsequently, they discovered that reports of occupational histories by next-of-kin for decedents were biased by the knowledge that radiation and leukemia were related (Colton 1979). This was confirmed by a recent historical prospective study which shows no excess in leukemia among radiation exposed workers in the same shipyard (Rinsky et al. 1981). More direct evidence that brief casual exposure in World War II shipyards probably caused little lung cancer excess comes from a recent

*On the basis of a 22% lung cancer excess observed among asbestos insulators where exposure apparently averaged 25 years at 8 f/cc it can be estimated that a 2 1/2% excess (RR = 1.4) after one years exposure would require continuous exposure at around 22 f/cc.

study of shipyard workers in Hawaii which shows essentially no excess in lung cancer followed 15-24 years and exposed to asbestos for less than 10 years (Kolonel et al. 1980). This study also indicates that about 40% of shipyard workers have no asbestos exposure at all. To apply a 1.6-1.7 relative risk to this large cohort of former shipyard workers seems inappropriate.

One way to predict lung cancer deaths among the cohort of 2.5 million former World War II shipyard workers alive today is by extrapolation from the several studies of asbestos insulators and asbestos products workers. To do this some estimate of the actual exposure levels is needed.

The time weighted asbestos exposure for insulation workers has been estimated at 4-12 f/cc (Nicholson 1976). What might the exposure of other exposed shipyard workers have been? There are no published data for shipyard workers to answer this. However, an informed guess is that during World War II for those workers actually exposed to asbestos, fiber counts were probably around 2 f/cc (Thorton 1981).* From the study by Kolonel (1980) we can estimate that about 60% or 1.5 million of the 2.5 million World War II shipyard workers were actually exposed to asbestos. For these 1.5 million workers alive today with a years exposure at 2 f/cc nearly 40 years ago we can estimate future asbestos lung cancer incidence by extrapolation from asbestos insulation workers data.

From data on asbestos products workers and asbestos miners it appears that the dose-response relationship is linear (Enterline and Henderson 1979; McDonald 1974). Moreover a time weighted measure of exposure seems appropriate since intensity and duration

*Current (1979) fiber counts in one shipyard ranges from .08 to .66 (Matanoski, 1981).

appear to make roughly equal contributions to lung cancer incidence (Enterline et al. 1973; McDonald et al. 1974).

If for asbestos insulators the exposure averaged 8 f/cc (4-12 f/cc) on a continuous basis and produced a 22% excess in mortality for lung cancer after about 25 years of exposure, we can easily calculate the excess for a single years exposure at 2 f/cc. The total exposure for asbestos insulators needed to produce a 22% excess in mortality due to asbestos lung cancer can be calculated as (8)(25) = 200 fiber years. For shipyard workers, on the other hand, exposure would have been (2)(1) = 2 fiber years. Therefore, we can calculate that the percent cancer excess in the 1.5 million World War II shipyard workers will be 2/200 or 1/100th of the lung cancer excess in asbestos insulators, or a .22% excess. Applied to the 1.5 million workers alive today this suggests 3,300 will die of asbestos lung cancer. If these workers live just 20 more years this is about 165 excess deaths per year.

Post World War II Shipyard Workers

A much more important population consists of workers who continued employment in shipyards after World War II, or were hired after World War II since their exposure lasted a considerable period of time. Shipyard employment is reported to have been around 200,000 since 1945 (Selikoff et al. 1979). Assuming labor turnover as in the industrial cohorts we have studied this would produce about 600,000 different workers with a year or more

work experience.* With a mortality rate at 1% per year we would expect 528,000 of these to be alive in 1970 and about 475,000 to be alive today. Based on the Kolonel study (1980) we can estimate that 285,000 of these were exposed to asbestos.

There have been several epidemiologic studies dealing with long term shipyard workers. Three of these show a clear lung cancer excess like the Blot et al. study (Puntoni et al. 1979; Kolonel 1980; Gottlieb 1980), one an intermediate excess (Milham 1976) and two essentially no excess (Lumley 1976; Beaumont 1980). These offer little consistent guidance for estimating a lung cancer excess. However, an informed guess as to post World War II continuous fiber levels is around 1 f/cc for those exposed, so we can estimate the lung cancer excess as for World War II shipyard workers (Thorton 1981).

Assuming continuous exposure was at 1 f/cc for 25 years and projecting from the experience of asbestos insulators we would expect the excess in lung cancer to be $\frac{25}{200}$ or 1/8 the excess in asbestos insulators. Thus, of the 285,000 workers alive today 2.7% or 7,695 would be expected to die of asbestos lung cancer. Over the next 30 years this would average 256 deaths per year. This estimate of excess lung cancer yields a relative risk of 1.4, which is a rough average of relative risks actually reported for long term shipyard workers.

*This is consistent with a count made by Matanoski (1981). As part of a study on the health effects of low level radiation she has estimated that there have been nearly 500,000 workers employed at shipyards now employing about 120,000 workers.

Auto Mechanics and Garage Workers

Frequently mentioned as an asbestos exposed population are auto mechanics and garage workers. This population has been estimated at about 900,000 continuously exposed to asbestos (Weston et al. 1976). Since World War II this number has probably grown considerably and might have averaged around 750,000 over the period 1940-70. Again, using experience from industrial epidemiologic studies, we might estimate that there were 2.2 million workers exposed a year or more. At a death rate of 1% per year 1.9 million would have been alive in 1970 and about 1.7 million alive today.

Studies indicate that the asbestos fiber concentrations to which auto mechanics and garage workers are continuously exposed are around .3 f/cc, well below that for shipyard workers (Hickish and Knight 1970). Probably the most applicable epidemiologic studies for estimating the effects of this kind of exposure comes from asbestos products workers. One study provides estimates of actual fiber exposure (Peto 1977). In that study the lung cancer excess was 15% and historic fiber concentrations were around 12 f/cc.* Exposure was about 25 years duration. Extrapolating from that experience permits an estimate of a $\frac{(.3)(25)}{(25)(12)} = \frac{7.5}{300}$ or (.025) (15) = .375% lung cancer excess. Applied to a population of 1.7 million this suggests 6,375 asbestos lung cancer deaths, over the next 30 years or 212 deaths per year.

*Substantially higher exposure estimates appear in a paper just made available but which the author feels must be regarded as provisional (Peto, L. Lung Cancer and Measure Dust Levels, Biological Effects of Mineral Fibers edited by J.C. Wagner, IARC, Lyon, 1980).

Secondary Asbestos Products

Weston Environmental Consultants estimate that there are 240,000 employees in manufacturing operations using asbestos products and whose exposure is fairly high. From their data it appears that exposure of these workers is around 2 f/cc. Considering this group of industries during the period 1940-70 and a mortality rate of 1% per year there are probably about 540,000 workers and former workers alive in 1981. If historic fiber counts were double the counts available when Weston made its survey (4 f/cc), workers had an average of 25 years employment and if the experience in the primary asbestos industries applies, then we can estimate that 5% of this population or 27,000 will die of asbestos lung cancer. This is 900 deaths per year over the next 30 years.

All Other Workers

Finally, what about all other potentially exposed workers in the United States? This must include construction workers, other workers using asbestos at their jobs, plus workers employed very briefly (< 1 year). A few of these might be in jobs with heavy but unrecognized exposure. The 1973 NIOSH Occupational Hazards Survey identified 1.6 million workers in the U.S. potentially exposed to asbestos of which 430,000 were in construction and 648,000 were in manufacturing. We have thus far dealt only with 40,000 heavily exposed, 240,000 secondary asbestos products workers, 200,000 shipyard workers, and 900,000 auto mechanics and garage workers. It's not entirely clear how these groups relate to the 1.6 million workers identified by the Occupational

Hazards Survey. Many garage workers and auto mechanics appear to have been omitted. If so there must be over a million workers with potential exposures, many from the construction industry, that still need to be accounted for. I would estimate this represents 3.5 million persons alive today. Considering occasional exposures and brief exposures as well as some longer or heavy exposures, I would estimate an exposure equivalent to .3 f/cc for 25 years--like garage workers and auto mechanics. Projecting as before from the experience of asbestos products workers yields an estimated lung cancer excess of $\frac{7.5}{300}$ or (.025)(.15) = .375%. Applied to a population of 3.5 million this yields an estimate of 13,140 asbestos lung cancer deaths or 438 deaths per year over the next 30 years.

Table 3 summarizes the results of these calculations with regard to asbestos lung cancer. To this I have added other cancers resulting from asbestos exposure under the assumption that for every 2 asbestos lung cancers there is one other cancer. I have also added a third of the approximately 1000 mesotheliomas that occur in the U.S. each year on the grounds that at least two thirds are due to exposures other than occupational exposures to asbestos (McDonald 1980). The total 4084 extra annual cancer occupational asbestos deaths is about 1% of all cancer deaths in the United States.

Finally deaths due to nonoccupational exposures to asbestos have been added. Asbestos lung cancer is estimated under the assumption that background is 1.5 ng/m^3 or .00006 f/cc (Nicholson et al. 1978). Predicting from asbestos products workers, but assuming continuous exposure rather than 8 hours per day, 5 days per week,

and assuming 70 years exposure suggests 28 asbestos lung cancer deaths per year for the entire United States: $\frac{(.00006)(70)}{(300)(.24)} \times .15 \times \frac{225,000,000}{70} = 28$. I have added an estimate of mesotheliomas produced by nonoccupational exposure under the assumption that two thirds of all mesotheliomas are due to asbestos and the rest due to something else (McDonald 1980).

Discussion

From available information it appears that about 1% of all cancer is due to asbestos exposure. This means that elimination of all asbestos from our environment would have an imperceptible effect on the cancer problem. It would ultimately have a great effect on asbetosis and mesothelioma deaths, but these are relatively unimportant as causes of death.

We can pinpoint some populations at high risk of cancer as the result of asbestos exposure and some special effort might be made here. Asbestos products workers and asbestos insulators exposed mostly before adequate controls were put in place for their protection are clearly high risk populations. The identities of most of the workers are known either to the asbestos workers union or to the government. Efforts to prevent disease in this population would be an important public service to them, although almost meaningless effort in terms of the overall cancer problem confronting us. Perhaps there are other such populations. I would be concerned about workers using asbestos products in the secondary asbestos products industry. For example workers making asbestos gloves from asbestos textile. These are usually small

highly competitive operations and dust control efforts may sometimes be inadequate.

It would be satisfying to believe that a single substance within our ability to control is responsible for a large part of an otherwise mysterious problem. Unfortunately this is not often the case. The role of asbestos in our overall cancer problem has been greatly exaggerated. One reason may be that we are becoming accustomed to exaggeration in dealing with environmental hazards in the belief that this somehow protects people, and is in the public interest. This may be true in initially drawing attention to a situation, but in the long run exaggeration is foolish since it leads to a wasteful effort and diverts resources from more fruitful areas. Moreover, falsely designating populations at high risk involves other human costs and is clearly not in the public interest.

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Table 1

Deaths from Asbestosis
United States 1950-77
by Sex and Race

Year	Total	White		Other	
		Male	Female	Male	Female
1950	8	6	1	1	-
1951	9	9	-	-	-
1952	14	12	2	-	-
1953	12	9	-	1	2
1954	8	7	1	-	-
1955	11	10	1	-	-
1956	20	16	3	1	-
1957	21	17	1	3	-
1958	14	12	1	-	1
1959	18	17	1	-	-
1960	21	19	-	2	-
1961	22	17	4	1	-
1962	18	10	-	7	1
1963	31	23	3	4	1
1964	24	21	2	1	-
1965	22	18	2	2	-
1966	29	26	3	-	-
1967	36	32	3	1	-
1968	29	25	3	1	-
1969	34	29	3	2	-
1970	26	21	1	4	-
1971	33	26	3	4	-
1972	58	54	2	2	-
1973	42	40	-	2	-
1974	35	30	3	2	-
1975	45	42	2	1	-
1976	54	46	1	7	-
1977	55	49	1	5	-

Table 2

Numbers of Asbestos Products Workers and Asbestos Insulators, 1947-76

Year	Workers Employed by Asbestos Products Companies[1] (SIC 3292)	Annual Average Membership in Asbestos Workers Union[2]	Asbestos and Insulation Workers in US Census[3]
1947	21,600		
1948			
1949			
1950			15,500
1951		6,000	
1952			
1953	22,200		
1954	22,000	9,000	
1955	23,700	10,000	
1956	22,800	10,000	
1957	22,900	13,700	
1958	21,300	13,700	
1959	22,800	10,000	
1960	22,400	10,000	20,000
1961	2[illegible],700	12,000	
1962	21,800	12,000	
1963	19,500	14,000	
1964	20,400	14,000	
1965	21,000	12,500	
1966	22,700	12,500	
1967	21,300		
1968	21,500		
1969	22,200	17,000	
1970	18,900	17,000	25,700
1971	18,900	18,800	
1972	21,000	18,800	
1973	21,300	18,300	
1974	21,000	18,300	
1975	17,300		
1976	16,600		

Source (1) Annual Census of Manufacturers US Department of Commerce
(2) Directory of Labor Unions in the US, US Dept. of Labor
(3) US Census of Population, US Bureau of the Census

Table 3

Estimated Cancer Deaths Which Will be Caused
by Asbestos Exposure Over the Next 20-30 Years

Exposure Group and Cause of Death	Number With Exposures Alive Today	Equivalent Exposure Level (f/cc)	Number of Deaths Per Year
Occupational Exposure:			
Asbestos Lung Cancer			
Insulation and Primary Asbestos Products Workers	80,000	8-12	530
Secondary Asbestos Products Workers	540,000	4	900
World War II Shipyard Workers	1,500,000	2	165
Post WW II Shipyard Workers	285,000	1	256
Auto Mechanics and Garage Workers	1,700,000	.3	212
All other exposed workers	3,500,000	.3	438
Total	7,605,000		2,501
Other Asbestos Cancers	--		1,250
Mesotheliomas caused by occupational exposure to asbestos	--		333
Total extra cancer deaths per year			4,084
Non-Occupational Exposure:			
Asbestos Lung Cancer	225,000,000		28
Mesothelioma	225,000,000		333

FROM JAMES E. HINES
INGOT CASTING DIVISION
ALCOA TECHNICAL CENTER - B

MARIO J. CAPRIO
PURCHASING DEPARTMENT
PITTSBURGH OFFICE - 19

TO DISTRIBUTION

1979 OCTOBER 31

RE: MOLTEN METAL MARINITE USAGE

Tabulated in Table I is the amount of 1219 x 2438-mm (4 x 8-ft) asbestos-containing Molten Metal Marinite sheets reportedly being purchased at each Alcoa ingot plant per year. Although most plants have attempted to reduce their usage of asbestos, there is still a substantial quantity used. The asbestos problem from a health hazard and from the additional costs to Alcoa to comply with OSHA Standards has not diminished. This fact together with Johns-Manville's objective to stop production of Marinite products containing asbestos by the end of 1980 should motivate all plants to make a concentrated effort to evaluate the more promising asbestos-free candidates mentioned below.

To date two products, Marinite I (Johns-Manville) and Pyrotherm B-2 (Pyrotek, Inc.), have been identified by both Alcoa Laboratories and limited plant trials as promising substitutes for regular grade, asbestos Marinite. Marinite I in the as-received form can be used only in non-molten metal contact areas (i.e., trough covers). However, with a simple preheat to approximately 149°C (300°F) to remove atmospheric moisture, it can be used as distributors or floats. An experimental heat treatment to 290°C (550°F) has been scheduled with Johns-Manville. At this exposure temperature, the board has provided good results as basins, troughing, feedboards, and dams. Large plant trials are scheduled initially at Massena, Tennessee and Warrick. Pyrotherm B-2, on the other hand, is supplied in the heat treated condition and can be used as a direct MM Marinite substitute. Pyrotek locations stocking this board are listed in Table III.

In Table II are the trial amounts of 25.4-mm (1-inch) and 50.8-mm (2-inch) sheets requested by the plants, along with the product suggested for evaluation. Some plants have already made these substitutes. They are indicated by asterisks.

ALCOA

TABLE I

MOLTEN METAL MARINITE USAGE BY PLANT*

	Board Grade and Size					
	Regular Grade				Header Grade	
	12.7 mm (0.5 in)	25.4 mm (1 inch)	BF	50.8 mm (2 inch)	BF	25.4 mm (1 inch)
Badin		50	1600	100	6400	50
Davenport		60	1920	-		-
Lafayette		30 ~100 headers	960	5	320	-
Massena		78	2496	-		300
Rockdale		178	5696	225	14400	-
Tennessee		358	11456	150	9600	100
Vancouver		-		-		-
Vernon	12	12 ~50 headers	384	-		-
Warrick		120	3840	240	15360	1600
Total	12	936	(30,000 BF)	720	(46500	2050

51,200 BF

66,000 BF

*Wenatchee and Point Comfort were not included in this survey.

TABLE II

TRIAL QUANTITIES OF ASBESTOS-FREE BOARD REQUESTED

	Boards			
	25.4 mm (1 inch)		50.8 mm (2 inch)	
Badin	50	Marinite I or Pyrotherm B-2	100	Pyrotherm B-2
Davenport	10	Marinite I		
Massena	15	Marinite I or Pyrotherm B-2	15	Marinite I
Rockdale	178	Pyrotherm B-2	225	Pyrotherm B-2
Tennessee	5	Pyrotherm B-2	5	Marinite I
Vancouver	3	Marinite I		
Vernon	12	Marinite I*		
Warrick	120	Pyrotherm B-2	240	Marinite I or Pyrotherm B-2*

*Substitutions already in progress

TABLE III

PYROTEK, INC. LOCATIONS

Plant	Address
Spokane, WA	E. 9601 Montgomery Avenue Spokane, WA 99206 Phone: (501) 926-6211
Carlisle, PA	Box 77, RD 1, Claremont Road Carlisle, PA 17013 Phone: (717) 249-2075
Trenton, IN	Kellwood Drive Trenton, IN 38382 Phone: (901) 855-0104

It is hoped that each plant makes known their testing so that information can be disseminated to the other locations and the level of activity can be monitored. A frequent updating of the Molten Metal Marinite situation and each plant's progress toward eliminating asbestos will be made. If a plant does not have the recommended asbestos-free candidates on hand or on order, Mr. M. J. Caprio in Pittsburgh Purchasing should be contacted immediately.

Any questions, comments, or additions to the attached would be appreciated. Only through a cooperative effort can asbestos be replaced.

JAMES E. HINES

MARIO J. CAPRIO

JEH/MJC:gih

Attachments - 3

(Distribution - Page 3)

PLANT METALLURGICAL ENGINEERS

C. T. Barger, Jr. - Badin Wks.
T. F. Arnold - Cleveland Wks.
A. F. Maloit - Corona Wks.
R. J. Stokwisz - Davenport Wks.
D. O. Collins - Lafayette Wks.
D. E. Scott - Lafayette Wks.
M. Scherbak - Massena Oprs.
G. G. Owens - Rockdale Wks.
G. K. Reichelt - Tennessee Oprs.
J. M. Ekenes - Vancouver Oprs.
W. C. Harvey - Vernon Wks.
D. Fleener - Warrick Oprs.
J. B. Gorss - Warrick Oprs.
P. C. Scheble - Warrick Oprs.
J. E. Gunn - Wenatchee Wks.

PLANT PURCHASING

H. A. Barton - Badin Wks.
T. G. Pollak - Cleveland Wks.
Shirley Price - Corona Wks.
R. W. Murphy - Davenport Wks.
R. C. Miessler - Lafayette Wks.
E. R. Werner - Massena Oprs.
J. R. Fry - Rockdale Wks.
J. Taylor - Rockdale Wks.
W. S. Zuber - Tennessee Oprs.
M. D. Wooden - Vancouver Oprs.
J. Heron - Vernon Wks.
T. A. Innes - Warrick Oprs.
D. Lutz - Warrick Oprs.

INFORMATION COPIES TO:

PLANT SUPERINTENDENTS

J. N. Rumble - Badin Wks.
M. A. Gambill - Davenport Wks.
W. K. Dalton - Lafayette Wks.
G. L. McCullough - Massena Oprs.
G. L. Williams - Pt. Comfort Wks.
T. J. Randall - Rockdale Wks.
D. W. Freeman - Tennessee Oprs.-S
D. F. Simonic - Tennessee Oprs.-N
D. E. Luckett - Vancouver Oprs.
M. L. Redhair - Vernon Wks.
A. Pearson - Warrick Oprs.
R. S. Grundhoefer - Warrick Oprs. (Can Reclamation)
E. T. Vanderheyden - Wentachee Wks.

C. J. Cox - 3 WPH
J. W. McIntee - 3 WPH
H. G. Reavis - AB 23
E. L. Rooy - AB 23
H. G. Sakoian - AB 7
A. J. Sartschev - 3 WPH
D. L. Schaeffer - AB 23
J. Damiano - ATC, C
T. W. Lee - ATC, D
R. E. Spear - ATC, C
J. R. Stemler - ATC, C
R. E. Miller/R. G. LaBar - ATC, B