PLAINTIFF'S EXHIBIT

SWB-65

Cancer Produced by Nonoccupational Asbestos Exposure in the United States

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Philip E. Enterline
University of Pittsburgh

There is considerable evidence of asbestos fibers in the general environment, and asbestos fibers can be found in the lungs of most adults in urban areas of the western world. Concentrations in large urban areas appear to average around 3 ng/m³ of air and in rural areas around 0.1 ng/m³ of air. For the entire U.S. population it can be estimated that the average population exposure is 1.5 ng/m³. Based on the results of case control and other studies, it is estimated that about a third of the 1000 or so cases of malignant mesothelioma that occur in the U.S. each year appear to be related to this type of nonoccupational asbestos exposure. This is a lifetime risk of 100 per million population. Lung cancer caused by asbestos in the general environment can be estimated from linear extrapolations of dose-response data arising from occupational studies. Using data from a study of retired asbestos products workers it is estimated that the lifetime risk of lung cancer due to continuous asbestos exposure at 1.5 ng/m³ is 2 per million. Possible causes for a fifty-fold difference between lung cancer and mesotheliomas caused by nonoccupational exposure to asbestos are discussed. These estimates are compared with estimates derived from a recent report commissioned by the West German government.

Coated fibers have been found in the lungs of residents of urban areas of the western world for nearly 20 years now. The meaning of this for human health remains as unclear today as it was when the phenomenon was discovered. At first, it was believed that all of the fibers were asbestos but it was soon realized that many were not. Nevertheless, a sufficiently large proportion have been positively identified as asbestos to warrant the descriptive term "asbestos bodies".

The discovery of asbestos bodies in human lungs has led to a careful examination of the air in urban areas and the conclusion that all urban areas are polluted with asbestos fibers.[1] The source of these fibers appears to be primarily asbestos building products. Table I summarizes levels of asbestos found in U.S. cities based on 24 h samples from each city.[2] On average, the concentration in large urban areas appears to be about 3.6 ng/m³, ranging from 0.09 to 78 ng/m³. In rural and in small urban areas of the U.S., asbestos concentrations are much lower—around 0.1 ng/m³.[3] Weighting these concentrations by number of people in large urban as compared with other areas gives an average outdoor asbestos exposure level of around 1.5 ng/m³.

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This is an attempt to estimate the number of cancers th are likely to be produced by asbestos among the nonoccup tionally exposed populations in the U.S., that is, the popu tions exposed to asbestos at the estimated average outdo background level of 1.5 ng/m³ of air. Not included in this e timate is cancer caused by nonoccupational indoor air poll tion. Only two of the health effects of asbestos will be co sidered: malignant mesotheliomas and lung cancers. Mali nant mesotheliomas produced by background levels of a bestos will be estimated directly from reports on the numbe of these cases and their probable asbestos exposures, whi lung cancers will be estimated by extrapolation from lun cancers produced by asbestos in occupational environment Studies which attempted to estimate the overall impact o occupational asbestos exposures on the cancer problem in th U.S. will first be reviewed, as these demonstrate some of th problems encountered in the estimation of health effects a background levels.

Table I. Concentrations of asbestos in urban areas of the U.S.

Asbestos concentration ng/m³	Mt. Sinai Number of samples	Battelle Institute Number of samples
<1	61	27
1–2	58	33
2–5	45	42
5–10	12	22
10–20	8	1
20–50	1	2
50–100	2	0
Mean	3.58	3.62
Median	1.57	2.29

Table II and Figure 1 summarize six studies that estimate the contribution of occupational asbestos exposures to annual cancer incidence in the U.S.[4–9] Five out of the six reports are in relatively close agreement, whereas the U.S. government's estimate that 13–18% of all cancer is the result of occupational asbestos exposure is quite different. The only other estimate of this magnitude was by Dr. Irving Selikoff, who estimated 40,000 cancer deaths per year caused by occupational exposure to asbestos, or about 10% of all cancer deaths.[10]

The U.S. government report has had a great impact on world opinion about asbestos since it was announced in 197

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Table II. Annual number of cancer deaths in the U.S. due to occupational exposure to asbestos--various estimates.

Source	Exposed population alive 1980	Annual number of cancer deaths	Percent of all U.S. cancer deaths
McDonald[a] (1981)[4]		3,330	<1%
Higginson (1979)[5]		4,000	1%
Enterline (1981)[6]	7,600,000	4,084	1%
Nicholson *et al.*[7] (1981)	9,200,000	8,500	2%
Hogan and Hoel (1981)[8]	7–8,000,000	12,000	3%
U.S. Govt. (1978)[9]	7–10,000,000	58–75,000	13–18%

[a] U.S. and Canada.

and received extensive press coverage throughout the world. In its 1979 report to the Congress, HEW listed this report as a significant accomplishment during the year 1978.[11] If the finding that 13–18% of all cancer is due to occupational exposures to asbestos were credible, clearly the banning of all asbestos should be considered. There is little support for the government document, however, as Table II suggests. In fact, partly as the result of this estimate, Congress commissioned a study that included an evaluation of the importance of occupational exposure to asbestos. The study concluded that asbestos may cause about 5% of all present day lung cancer.[12] Inasmuch as lung cancer constitutes about 20% of all cancer, this suggests that asbestos-caused lung cancer accounts for about 1% of all cancers—a finding which tends to support most of the estimates shown in Table II.

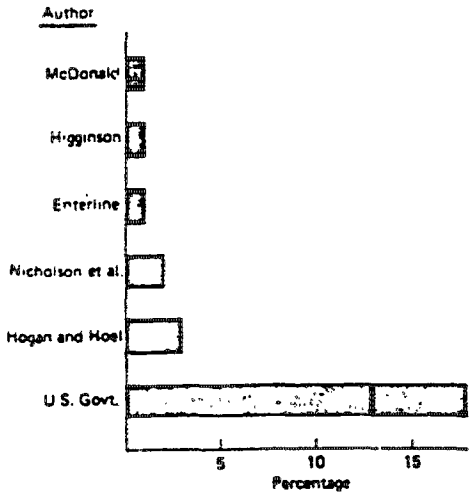

Figure 1. Estimates of the percentage of all cancer due to occupational exposure to asbestos in the U.S. in 1980 (McDonald estimate reflects U.S. and Canada).

While there are very large differences in the cancer deaths shown in Table II, there is not much disagreement on the size of the occupationally exposed population. The reasons for the differences in the estimates of health effects are twofold: (1) a difference of opinion as to the intensity of exposures received by various groups of workers, and (2) a difference of opinion as to the appropriate dose-response relationship for asbestos and cancer.

When dealing with the health effects of outdoor background asbestos concentrations, there are too few data on the intensity of exposure to cause much disagreement. The main problem is to calculate the response likely at very low exposure levels.

Attempts to directly measure the amount of lung cancer produced by low doses of asbestos have been unsuccessful. For example, in 1976 a group of government investigators reported on the effects of very low human exposures to asbestiform fibers among workers at a gold mine at Lead, SD.[13] They reported a threefold excess in respiratory cancer and suggested that this was due to very low fiber exposures. A second study of workers from the same mine by researchers from McGill University showed essentially no excess in respiratory cancer.[14] The U.S. government commissioned a third study by a private research group. This study found no excess in respiratory cancer among these same workers.[15]

One method for estimating lung cancer caused by low dose environmental exposure is by extrapolation from effects of exposure at higher levels.[16] An estimate of lung cancer caused by nonoccupational exposure was prepared (using the 1981 Enterline estimate from Table II), assuming a general population exposure of 1.5 ng/m^3 and that 1 ng contains 40 fibers of asbestos greater than 5μm in length, seen by the optical microscope.[6] The number of lung cancers at 1.5 ng/m^3 was estimated by a simple linear extrapolation from the experience in a single British study by Peto, *et al.* of the dose-response relationship between asbestos and lung cancer in a population of asbestos textile workers.[17] These estimates of asbestos-caused lung cancer are shown in Table III.

Table III. Annual number of cancer deaths in the U.S. caused by exposure to asbestos

Type of exposure and cancer	Annual number of cancer deaths	Percent of all U.S. cancer deaths
Occupational exposure	4034	1
Lung cancer	2501	
Mesothelioma	333	
Other cancer	2150	
Nonoccupational exposure	361	<0.1
Lung cancer	28	
Mesothelioma	333	

Also shown is malignant mesothelioma caused by asbestos exposure. This was calculated as follows: There are about 1000 new malignant mesotheliomas in the U.S. each year with a male-female ratio of 3:1, or about 750 males and 250 females.[18] McDonald and McDonald estimate that for males with mesothelioma, 53% are the result of an occupational asbestos exposure.[4] From their study it also appears that no more than 5% of females with a malignant mesothelioma have this as the result of an occupational asbestos exposure. Thus, there are 397 males and 13 females out of the 1000 malignant mesotheliomas reported each year whose mesotheliomas were caused by occupational asbestos exposure. This is a slight upward revision of the 333 reported in 1981 (Table III), since it is based on data not available in 1981. By subtraction, there are 353 males and 237 females whose mesotheliomas were caused by something other than occupational exposure to asbestos.

What percent of this total 590 are caused by the average outdoor asbestos level of 1.5 ng/m^3 of air? There is evidence that fibers other than asbestos cause malignant mesotheliomas.[19] There is also evidence that nonoccupational exposures to asbestos cause malignant mesotheliomas.[20] The previously estimated 333 malignant mesotheliomas caused by nonoccupational asbestos exposure—a little over half the 590 nonoccupationally caused malignant mesotheliomas[6]—is consistent with an estimate made by McDonald and McDonald in 1981.[4] This will continue to be used as a best estimate.

Since the estimate of nonoccupational lung cancer shown in Table III was made, some assumptions in the study upon which it was based have been revised. Apparently, exposure

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chosen because it was the only study for which historic exposure data were available expressed in fibers, the currently acceptable method of reporting asbestos concentrations. Moreover, it appeared to be well known and well accepted, and was one of three selected for inclusion in the dose-response section of the report by the Advisory Committee on Asbestos.[22] It has now been reported, however, that the fiber exposures in this study were probably about 3 times higher than originally thought. If so, this would make the estimate of lung cancer deaths in Table III around 9 or 10 rather than 28. This has little effect on the overall estimate of 361.

In 1981, the effects of asbestos fibers emitted by hair dryers with asbestos-containing heat shields on lung cancer mortality were reported.[16] This estimate was based on a linear extrapolation from a study of lung cancer among retired U.S. asbestos products workers.[23] Figure 2 shows the dose-response relationship between asbestos concentration expressed as million particles per cubic foot (mppcf) and excess lung cancer expressed as Standardized Mortality Ratios (SMR). SMR are the ratio of observed to expected deaths times 100 and are a commonly used measure of excess deaths in epidemiologic studies. From these data, lung cancer deaths that would result from continuous exposure to very low levels of airborne asbestos can be estimated.

Some results from the 1981 report are shown in Table IV, expressed as lifetime risk of lung cancer resulting from continuous asbestos exposure per million people exposed. In preparing this table, asbestos concentrations expressed as mppcf were converted to fibers per cubic centimeter (f/cc) of air by assuming 1 mppcf = 3 f/cc. Also, as in Table III, 1 ng of asbestos was assumed to contain 40 fibers of asbestos greater than 5μm in length as counted by the optical method. The rationale for these conversions appears in the 1981 report. Lifetime lung cancers were calculated assuming that lung cancer starts 20 yr after the beginning of exposure and that an average lifetime is 70 yr, so that the effective exposure period is 50 yr. The effect of hair dryers appears on the first line followed by the effect of all nonoccupational exposures.

Table IV. Lifetime lung cancer deaths resulting from various levels of asbestos exposure per million exposed.

Level of exposure (ng/m^3)	Equivalent fibers/cc (>5 μm long)	Lung cancer deaths
1	0.000037	0.85
1.5	0.00006	2.08
5	0.0002	4.6
50	0.002	46.06
500	0.02	460 6
5,000	0.2	4,606
50,000	2	46,060

For a U.S. population of 225 million, Table IV suggests 225 × 2.08, or 468 lifetime deaths from lung cancer due to nonoccupational asbestos exposure at 1.5 ng/m^3, whereas Table III suggests 28 × 70, or 1960 lifetime asbestos lung cancers related to nonoccupational asbestos exposure at 1.5 ng/m^3. If the exposure estimate upon which Table III was based are off by a factor of 3, however, these estimates are much closer (653 vs. 468). For malignant mesothelioma, Table III suggests a lifetime risk of 333 × 70 or 23,310 cases for a U.S. population of 225 million.

It is plausible that the original estimates of fiber exposure in the British study of asbestos textile workers were indeed too low. The British study estimated an average fiber count for asbestos textile operations of around 12 f/cc prior to 1950.[17]

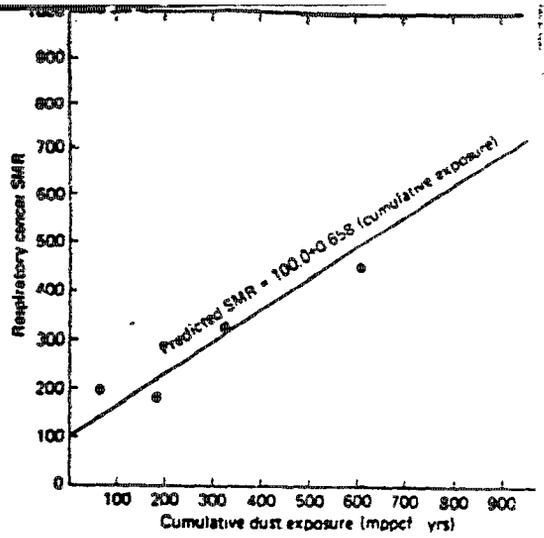

Figure 2. Asbestos dust exposure and respiratory cancer mortality

For retired workers from a Johns Manville asbestos prod plant in the U.S., average worker exposure was 10 mppc If this is converted to f/cc by assuming 1 mppcf = 3 f/cc t this gives an average exposure of 30 f/cc. In a textile operat reported on by Nicholson, historic fiber counts were estim at 80 f/cc.[24] It seems likely, therefore, that exposures in British study could have averaged 36 f/cc rather than 12 and that the estimates of U.S. lung cancer deaths show Table II should be lowered by a factor of 3.

One important assumption in Tables III and IV conce the conversion of ng to fibers seen by the optical met Many fibers cannot be seen by the optical method. More fiber size determines t conversion factor. The conver factor of 40, used in estimating nonoccupationally caused l cancer in Tables III and IV, is perhaps high. For examp conversion factor of 30 was used in EPA's water quality d ument.[25] Bruckman recommends 20.[26] To the extent that t conversion factor is high, it overstates lung cancer cases cau by nonoccupational asbestos exposure.

It is difficult to judge the validity of the estimates sho in Tables III and IV. There have been only a few attempts estimate the health effects of asbestos exposure on the gene population. In 1977, Bruckman reported on the developme of criteria for setting an asbestos air standard for the state Connecticut and concluded that 30 ng/m^3 would be a sa level.[26] It was estimated that at this level, a minimum of 1 mesotheliomas would be produced on a nationwide basis. Th level was justified as one that results in about the sam number of deaths as train mishaps and about 1/10 the year fatalities from airplane accidents. Only a minimum numb of mesothelioma deaths was calculated on the grounds th this would be difficult to criticize as being too strict. An upp limit can be calculated from the data and appears to be abou 10,000. Extensive details on the manner in which these est mates were made are not given.

In 1981, Schneiderman, *et al.* were commissioned by th West German government to prepare a report on the risks b exposure to low levels of asbestos in the general environ ment.[27] Their report provides a basis for calculating the life time risk of asbestos exposures at low levels, provided som conversion of ng to fibers can be made. The concluding tabl in their report, labeled "Risk assessments for the genera population . . .", apparently is related to the risk for a workin population exposed 8 h/day, five days/week. These risks ca be converted to continuous exposure, however, by dividing b 0.24, since working time is 24% of total time. It can then b

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calculated that the lower limit of lifetime risk of lung cancer for a population exposed to 1 f/cc for 1 yr is 0.0015. The lower limit of risk for exposure to 1.5 ng/m^3 with 36 yr effective exposure (assumed by Schneiderman, *et al.*[27]) can be calculated if ng are converted to fibers. Using the conversion factor of 40 gives 0.00008 f/cc. The risk is therefore (0.00006)(36)(0.0015), or 0.00000324. Since the U.S. population is about 225 million this means a lifetime excess of 729 lung cancer deaths due to nonoccupational asbestos exposure (0.00000324)(225 million). Tables in the Schneiderman, *et al.* report also show upper limits, with the upper limit of their lung cancer estimates simply 10 times the lower. Therefore, the upper limit of lung cancer deaths due to nonoccupational asbestos exposure is 7290. These estimates of 729 and 7290 contrast with the above estimates of 428 and 653. The Schneiderman, *et al.* report also permits an estimate of lifetime risk for malignant mesothelioma. This is 0.0033 (lower limit) for 1 yr exposure at 1 f/cc. For exposure at 1.5 ng/m^3, assuming 20 yr effective exposure (assumed by Schneiderman, *et al.*), this is a lifetime lower limit of 891 cases. The upper limit is 5,400 cases. This contrasts with a lifetime excess of 70 × 333, or 23,310, calculated from Table III.

One of the reasons for the higher lung cancer estimates in the Schneiderman, *et al.* report is that they found reasons to reject the results of four epidemiologic studies which gave risks of less than 1% for 1 yr exposure at 1 f/cc. This included all three studies used in the dose-response section of the report issued by the Advisory Committee on Asbestos.[22] Rather, they relied on dose-response data compiled in 1981 by Nicholson, some of which were previously unpublished.[24] Of the five studies they found acceptable, three were from Selikoff's laboratory in New York City, one of which yielded a dose-response relationship based on only 2 lung cancer deaths.[24] Neither of the others was designed to study dose-response. For one, only duration of exposure was measured for each worker, while for the other, workers could only be subdivided by time since first exposure, since duration of exposure was unknown. The fourth study[28] provides very few data on exposure and was described by Nicholson as containing "only sketchy information".[24] The authors of that study only discuss "possible exposure levels" with no details on how these were determined. The fifth study, a study of asbestos textile workers by Dement, *et al.*, appears to be a good one with well documented exposure levels.[29] Numbers of lung cancer cases were relatively small, however, and the results of this study are quite unlike anything observed elsewhere. Lung cancer response per unit dose was very high. It was 88 times greater than a similarly documented but much larger study of asbestos miners.[30]

Discussion

The above calculations lead to estimates of lifetime risks due to nonoccupational asbestos exposure of 100 per million for malignant mesotheliomas and 2 per million for lung cancers. This is a fifty-fold difference in favor of mesothelioma and is quite unlike contrasts observed in studies of occupationally exposed populations. In occupationally exposed populations, the excess favors lung cancer, with the ratio of excess lung cancer to mesotheliomas around 2 or 3.[4] Using a different set of assumptions, the Schneiderman, *et al.* study gives estimates of a lifetime excess due to nonoccupational exposure of 4–24 per million for mesothelioma and 3–30 per million for excess lung cancers.[27] This also differs from the 2 or 3 ratio of lung cancers to mesotheliomas in occupationally exposed populations.

Is it reasonable to suppose that nonoccupational asbestos exposures produce more mesotheliomas than lung cancers while occupational exposures produce more lung cancer than mesotheliomas? One reason for an excess of mesotheliomas in nonoccupationally exposed populations may be the much earlier age at first exposure and an unusual mesothelioma risk associated with exposure at an early age. This is because the process by which asbestos produces mesotheliomas seems to be different from the process by which asbestos causes lung cancers. Peto, *et al.* feel that in the production of mesotheliomas, asbestos acts at an early stage in a multistage process believed to produce cancer.[31] As a result, they feel that mesotheliomas are manifest only many years after first exposure, and that where exposure to asbestos starts at an early age, mesotheliomas will constitute a high proportion of asbestos related cancer deaths. On the other hand, in the production of asbestos lung cancers there is considerable evidence to suggest that asbestos acts at a late stage in a multistage process. As a result, lung cancers occur more quickly than mesotheliomas, so that even where exposure starts at age 30 or 40 there is time for lung cancers to be manifest before deaths due to other causes intervene. Mesotheliomas may, therefore, be the predominant asbestos cancer related to nonoccupational exposures simply because of the early age at which these exposures start, whereas lung cancer is the predominant cancer in occupationally exposed populations due to the older age at which exposure starts and a lack of time for mesotheliomas to be fully expressed.

Whether this is a sufficient explanation for a fifty-fold excess in mesotheliomas relative to lung cancers may be questioned. Given the well established role of asbestos in the etiology of mesotheliomas, and the exposure of nearly the entire U.S. population to asbestos, it seems likely that nonoccupational exposure causes at least a third of the mesotheliomas that occur in the U.S. each year and perhaps more. Therefore, the estimate of a lifetime risk of 100 per million due to nonoccupational asbestos exposure seems conservative and the true risk may, in fact, be higher than this. On the other hand, the estimate of a lifetime risk of 2 per million for lung cancer could be low. The dose-response relationship may not be linear at low doses, as supposed, or perhaps the response per unit dose is greater than was assumed. No one has seriously proposed a dose-response relationship that is curvilinear downward, as would be needed to increase response at low dose.

There are, however, studies that give a larger response for lung cancer per unit dose than the one used. The highest response yet reported was in the study by Dement, *et al.*[24] If this had been used, instead of the study of retired asbestos workers,[18] then the lifetime risk of asbestos lung cancers resulting from nonoccupational asbestos exposure would have been 40 per million instead of 2. This reduces the apparent mesothelioma-lung cancer discrepancy but does not eliminate it. There does not appear to be any reasonable set of assumptions that would yield, for the nonoccupationally exposed population, the 2–3 excess lung cancers per mesothelioma observed in occupationally exposed populations. The issue, therefore, does not appear to be whether the ratio of mesotheliomas to lung cancers caused by background levels of asbestos exceeds 1, but rather, by how much it exceeds 1.

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Dr. Enterline is a Professor and Chairman, Department of Biostatics, and Associate Director, Center for Environmental Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA 15261. This paper was submitted for editorial review on June 2, 1982; the revised manuscript was received on February 14, 1983.

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