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Projections of Cancer Risks Attributable to Future Exposure to Asbestos

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To assess the maximum possible impact of further government regulation of asbestos exposure, projections were made of the use of asbestos in nine product categories for the years 1985–2000. A life table risk assessment model was then developed to estimate the excess cases of cancer and lost person-years of life likely to occur among those occupationally and nonoccupationally exposed to the nine asbestos product categories manufactured in 1985–2000. These estimates were made under the assumption that government regulation remains at its 1985 level. Use of asbestos in the nine product categories was predicted to decline in all cases except for friction products. The risk assessment results show that, although the cancer risks from future exposure to asbestos are significantly less than those from past exposures, in the absence of more stringent regulations, a health risk remains.

KEY WORDS: Asbestos; risk assessment; cancer; regulation.

1. INTRODUCTION

Since the early part of this century, evidence has accumulated indicating that lung cancer, mesothelioma, and asbestosis are causally related to exposure to asbestos fiber.[1] Over the past 12 years, the U.S. Government has been actively involved in regulating the asbestos-using industries to minimize the occurrence of such asbestos-induced disease. Several studies have estimated the number of cases of asbestos-related diseases that we can expect to observe over the next few decades as a result of occupational exposure to asbestos in the past.[2–4] However, to assess the impact of further government regulations, it is necessary to predict what the health risks would be in their absence. This paper describes a health risk assessment model designed to estimate the excess lung cancer and mesothelioma cases that we can expect to observe over the next 100 years as a result of *future* occupational and nonoccupational exposure to asbestos; specifically, exposure to asbestos products manufactured during the years 1985–2000. The estimates are derived assuming that government regulation remains at its 1985 level, and, therefore, represent the maximum benefits achievable through more stringent regulations.

The basic logic employed in the health risk assessment model is illustrated by the flow diagram in Fig. 1. The exposure data for the level of exposure and number of people exposed for the year 1983 were obtained from government and private sources. Projections of the total amounts of asbestos used in each of nine asbestos product categories for the years 1985–2000 relative to the amount used in 1983 were combined with the data on persons exposed in 1983 to estimate the number of people exposed to asbestos attributable to products manufactured during the years 1985–2000. In most cases, current exposure levels were assumed to remain constant until the end of the century. Linear dose–response relationships were used to relate lung cancer and mesothelioma

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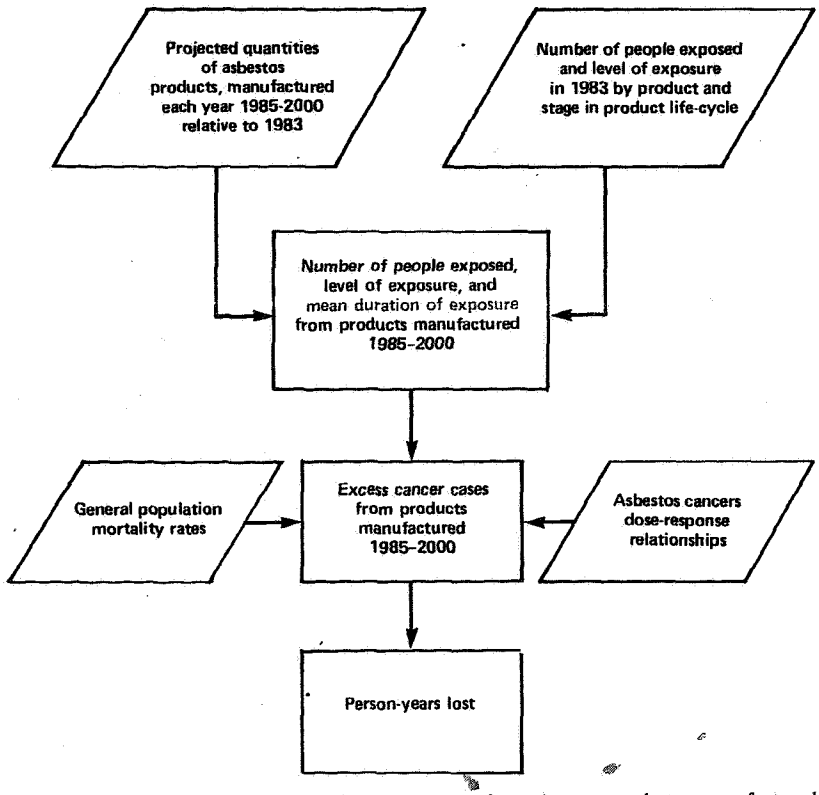

Fig. 1. Flow diagram for cancer risk assessment for asbestos products manufactured 1985–2000.

incidence rates to cumulative exposure. Using a life table model, these disease incidence rates were combined with the estimated number of people exposed to give estimates of the expected number of cases of lung cancer and mesothelioma attributable to exposure to asbestos products manufactured in 1985–2000 for each year after 1985. Estimates were also obtained of the mean age of the victims and the total person-years of life lost due to this asbestos exposure.

2. ASBESTOS EXPOSURE AND ILL HEALTH

Numerous human and animal studies have documented the correlation of exposure to asbestos fibers with increased incidence of many diseases, including asbestosis, lung cancer, mesothelioma, gastrointestinal cancer, and other cancers. Although much of the research has focused on the effects of exposure to the high levels of asbestos typically associated with occupational exposure before 1972, there is evidence that low exposures may be hazardous. At low concentration levels, lung cancer and mesothelioma present the greatest threat to human health.[5,6] Therefore, these are the only diseases analyzed in this paper. To the extent that asbestosis and other cancers are induced by low exposures, these estimates of the health effects from future exposure represent a lower bound.

2.1. Time between Onset of Exposure and Diagnosis of an Asbestos-Related Disease

This analysis is restricted to the health effects attributable to exposure to asbestos products manufactured between 1985 and 2000. These effects of future exposure would not be apparent until after 1995 because of the long time that usually elapses between onset of exposure and diagnosis of an asbestos-related disease. The time between onset of

exposure and diagnosis of disease for lung cancer usually ranges from 20 to 40 years with a minimum of 10 years. This range can be partially explained by the apparent action of asbestos to increase the general population risk by a factor proportional to cumulative exposure. Seidman *et al.*[7] reported a shorter lag time when initial exposure occurred at the cancer ages (over age 40), which would be consistent with this explanation. Mesothelioma also has a long time to diagnosis (i.e., 20–50 years with a minimum of 10 years) from onset of exposure, but this latency appears to be independent of age at first exposure.[8]

2.2. Level and Duration of Exposure

Epidemiological studies of their dose–response relationships have been performed with heavily exposed industrial cohorts. The data from these studies are consistent with the assumption that excess mortality from lung cancer and mesothelioma is proportional to both the level and duration of exposure to asbestos fibers. The most direct evidence for a linear dose–response relationship for lung cancer comes from two studies.[9,10] However, the implied dose–response constants for these two studies as well as those derived from other studies vary in magnitude, from 6.0×10^{-4} to 6.8×10^{-2}.[11] Data are also available for mesothelioma that are consistent with a linear relationship to cumulative exposure,[7,12,13] with implied dose–response constants varying from 0.7×10^{-9} to 0.2×10^{-7}.[11] None of these studies showed any evidence of a threshold level of exposure below which exposure to asbestos is safe.

2.3. Fiber Type

Chrysotile is the main type of asbestos fiber used in the United States except for A/C pipe, which also contains amosite. Animal studies indicate that the physical condition of the fiber is the most important factor in inducing asbestos-related diseases. Differences in risk of lung cancer or mesothelioma, formerly attributed to fiber type, may reflect differences in distribution of fibers of various sizes in the dust of each fiber type.[14] In this paper, however, it is assumed that the dose–response constants do not vary according to the fiber type or size to which the person was exposed.

3. NUMBER OF PEOPLE EXPOSED TO ASBESTOS AND LEVEL OF EXPOSURE IN THE BASE YEAR 1983

Individuals may be exposed to asbestos both in occupational and nonoccupational settings. Occupational exposure may occur among individuals employed in the manufacture, installation, repair, and disposal of the asbestos product or among those using the product at work. Nonoccupational exposure can be subdivided into ambient exposure and consumption exposure. Ambient exposure may occur among persons living or working close to the asbestos product manufacturing site or close to a site where the asbestos product is used or discarded. Consumption exposure may occur among the consumers of asbestos products.

For each stage in the product life-cycle for each product, data on the mean level of exposure and the number of people exposed were developed, to the extent possible, from data collected by the Occupational Safety and Health Administration, the Research Triangle Institute, Versar Incorporated, Consad, and the Bureau of Labor Statistics. All data were assumed to correspond to the 1983 production level of asbestos products. Table I presents the 1983 exposure estimates for nine product types used in the analysis.

4. PROJECTED NUMBER OF PEOPLE EXPOSED TO ASBESTOS, LEVEL OF EXPOSURE, AND MEAN DURATION OF EXPOSURE DURING THE YEARS 1985–2000

To estimate the number of people exposed to asbestos during the years 1985–2000, projections of the total amounts of asbestos used in each of the nine asbestos product categories relative to the amount used in 1983 were made for these years. These projections of growth rates of asbestos use were made using the model described by Research Triangle Institute[15] and are shown in Table II. From these growth rates, indexes were constructed, using 1983 as the base year, for the relative levels of asbestos use for the nine product categories for each year during 1985–2000. With the exception of friction materials, the growth rates for asbestos use were predicted to be negative.

In order to use the linear no-threshold dose–response relationships described below to estimate

Table I. Level of Exposure to Asbestos and Number of People Exposed in 1983

	Friction products	A/C pipe	Coatings and sealants	Paper products	V/A floor tile	Gaskets and packing	Textiles	A/C sheet	Plastics
1. Occupational									
Primary manufacturing[a]									
10^6 fibers per year	1,360	240	620	260	120	740	740	1,380	560
number of people	5,104	512	1,327	387	276	315	413	203	324
Secondary manufacturing[a]									
10^6 fibers per year	540	—	—	—	—	160	1,180	900	200
number of people	1,504	—	—	—	—	9,972	164	345	2,450
Installation[b]									
10^6 fibers per year	—	200	330	100	49	652	520	260	—
number of people	—	1,675	100,000	10,168	6,800	7,500	1,500	1,265	—
Use[c]									
10^6 fibers per year	—	—	—	—	—	—	1,560	—	—
number of people	—	—	—	—	—	—	2,507	—	—
Repair/disposal[b]									
10^6 fibers per year	30	—	—	—	—	—	520	—	—
number of people	551,207	—	—	—	—	—	1,500	—	—
2. Nonoccupational									
Primary manufacturing[d]									
10^6 fibers per year	0.0063	2.79	0.00003	0.00153	0.045	0.666	0.0504	2.79	0.00003
number of persons	1.65×10^6	1.7×10^6	6.35×10^6	2.25×10^6	1.66×10^6	0.78×10^6	0.27×10^6	1.17×10^6	1.32×10^6
Secondary manufacturing									
10^6 fibers per year	—	—	—	—	—	—	—	—	—
number of persons	—	—	—	—	—	—	—	—	—
Installation[e]									
10^6 fibers per year	—	—	—	—	0.13	—	—	—	—
number of persons	—	—	—	—	300,000	—	—	—	—
Use[f]									
10^6 fibers per year	0.008	—	—	—	0.797	—	—	—	—
number of persons	139.2×10^6	—	—	—	75×10^6	—	—	—	—
Repair/disposal[f]									
10^6 fibers per year	0.16	—	—	—	0.6	—	—	—	—
number of persons	1.5×10^6	—	—	—	64×10^6	—	—	—	—

[a]Derived from OSHA data (1979–1985) and RTI survey data (1984) presented in Source 1.
[b]Derived from Consad data, Versar data, and BLS data presented in Sources 2, 3, 4, and 5.
[c]Derived from EPA data and Lumley (1971) presented in Sources 6 and 7.
[d]Derived from Versar data presented in Sources 3 and 5.
[e]Derived from Versar data and BLS data presented in Sources 3, 4, and 5.
[f]Derived from Versar data and RTI data presented in Sources 3 and 5. The persons exposed to V/A tile use and repair/disposal are included in the 139.2×10^6 persons living in urban regions who are exposed to use of friction products.

Sources: (1) Research Triangle Institute, *Regulatory Impact Analysis of The Proposed OSHA Asbestos Standard*. Prepared for U.S.D.O.L., Contract No. J-9-F-4-0027, November 1985. (2) Consad Research Corporation, *Asbestos Task Order for Construction Alternatives*. Prepared for U.S.D.O.L., Contract No. J-9-F-4-0024, May 1984. (3) Versar Incorporated, *Exposure Assessment for Asbestos*. Prepared for U.S. E.P.A., Contract No. 69-01-6271, 1983. (4) U.S. Department of Labor, Employment and Earnings 30, #6, BLS, June 1983. (5) Research Triangle Institute, *Regulatory Impact Analysis of Controls on Asbestos and Asbestos Products*. Prepared for U.S. E.P.A., August 1985, Volume III, Appendix N. (6) K. P. S. Lumley, "Asbestos Dust Levels Inside Firefighting Helmets with Chrysotile Asbestos Covers." *Annals of Occupational Hygiene* **14**, 285–286. (7) Memo from Amy Moll, U.S. Environmental Protection Agency, October 1, 1985.

Table II. Projected Annual Growth Rates for Nine Asbestos Product Categories[a]

Asbestos products	Net growth rate (%)
Friction products	3.78
A/C pipe	−6.11
Coatings and sealants	−10.42
Paper products	−6.90
V/A floor tile	−6.41
Gaskets and packing	−2.22
Textiles	−15.60
A/C sheet	−3.18
Plastics	−53.6

[a]Source: Research Triangle Institute, "Asbestos Products Baseline Projections," *Regulatory Impact Analysis of Controls on Asbestos and Asbestos Products: Appendix C.* Prepared for U.S. Environmental Protection Agency under Contract No. 68-02-4055 (1985).

the health effects of asbestos exposure, it was necessary to have estimates of the number of persons exposed, the mean duration of exposure to asbestos for those exposed, and the level of that exposure. The total number of person-years of exposure for each exposure category was estimated using the population exposure data for 1983, the production indexes described above (except for the consumer use category), and the mean duration of exposure for each year of production. The person-years of exposure were then divided into estimates of number of persons exposed and their mean duration of exposure.[15] The total number of people estimated to be occupationally exposed to asbestos products manufactured from 1985 to 2000 was approximately 0.9 million, and nonoccupationally exposed, 150 million. Those nonoccupationally exposed included many persons who might be exposed to more than one source of asbestos. Mean levels of exposure were assumed to remain constant at the 1983 level during the years 1985 to 2000 except for the consumer use category where they were assumed to vary with projected production levels.

5. PROJECTION OF HEALTH EFFECTS

5.1. Life Table Model Overview

The health effects of exposure to asbestos products manufactured between 1985 and 2000 were estimated on a product-by-product basis. For each product, the population at risk was subdivided into the exposure categories according to when in the product life-cycle exposure takes place and whether they were exposed occupationally, in the ambient air, or in use of the product. Each exposure category was further subdivided into 10-year age groups. All members of each group were assumed to have the age in 1985 equal to the midpoint of the group range. For each member of an exposure category, *future exposure* was defined as the duration and intensity of exposure to asbestos that results from products manufactured during the years between 1985 and 2000. For one member of each age subgroup, the health effects of this future exposure were then estimated using an adaptation of the life table model described in Eddy.[16]

To make this estimate, a nonstationary Markov process was constructed containing 11 states that an individual might be in during a specific 1-year time period:

1. Alive, with no known lung cancer or mesothelioma.
2. Alive, but cured of lung cancer or mesothelioma.
3. Dying of lung cancer due to base rate or exposure to asbestos products manufactured prior to 1985.
4. Dying of lung cancer due to exposure to asbestos products manufactured between 1985 and 2000.
5. Dying of mesothelioma due to exposure to asbestos products manufactured prior to 1985.
6. Dying of mesothelioma due to exposure to asbestos products manufactured between 1985 and 2000.
7. Dead of lung cancer—base rate or previous exposure.
8. Dead of lung cancer—future exposure.
9. Dead of mesothelioma—previous exposure.
10. Dead of mesothelioma—future exposure.
11. Dead of other causes.

It was assumed that in 1985, an individual has a probability 1 of being in state 1 and at age 90 a probability 1 of being dead; i.e., in states 7–11. It was also assumed that an individual can only contract either lung cancer or mesothelioma once and that he or she cannot contract both diseases. The

appropriate transition matrix for each year between 1985 and age 90 was estimated and used to calculate the change in probability of an individual being in states 8 and 10 each year. The probability of contracting lung cancer or mesothelioma each year was also calculated using the transition probabilities. Estimates of excess cancers from previous exposures are not presented in this paper. These probabilities, generated for each remaining year of the person's life, were multiplied by the number of people in the subgroup to obtain estimates of the expected cancer deaths/cases for the whole population subgroup.

The outputs just described were for a single age subgroup of an exposure category for a single product category. To compute the total health effects of future exposure to asbestos, these outputs were obtained for all the age subgroups of all exposure categories for all the product categories and combined. Timing of exposure was important here. For the occupational exposure categories, it was assumed that manufacture and installation take place in the same year. Disposal or repair of products manufactured between 1985 and 2000 takes place after lifetime use is completed. Thus, the deaths/cases avoided for this occupational exposure category were displaced by a number of years equal to average lifetime use before being added to those related to manufacture and installation. Those exposed nonoccupationally to the manufacture, installation, and disposal of products were treated the same way as the corresponding occupationally exposed groups. Those exposed during the use of the asbestos product were assumed to start exposure at the time of manufacture and continue to be exposed for the average life of the product.

Results of such calculations for the nine asbestos product categories were summed to determine expected total cancer deaths/cases. The lifetime probability of contracting lung cancer or mesothelioma due to exposure to asbestos products manufactured between 1985 and 2000 was also estimated using the life table model, as were the person-years of life lost due to the asbestos exposure.

5.2. Life Table Model Inputs

A different transition matrix for each year starting from 1985 for each population subgroup for each product was calculated to determine health effects using the data described in this section.

Table III. Sex, Race, and Age Distribution of Exposed Populations[a]

Characteristic	Proportion of population (decimal share)	
	Occupational	Nonoccupational
Sex		
Male	0.79	0.49
Female	0.21	0.51
Race		
White	0.91	0.88
Nonwhite	0.09	0.12
Age		
0–9	0	0.146
10–19	0.1	0.174
20–29	0.205	0.176
30–39	0.210	0.139
40–49	0.193	0.108
50–59	0.175	0.099
60–69	0.117	0.083
70–79	0	0.055
80–89	0	0.020

[a]Sources: U.S. Department of the Census, *Statistical Abstract of the United States* (Bureau of the Census, Washington, D.C., 1980). USDOL, *Employment and Earnings 30*, No. 6 (Bureau of Labor Statistics, Washington, D.C., June 1983).

It was assumed that the nonoccupationally exposed population was identical to the U.S. population in terms of sex, race, smoking habits, and age distribution (see Table III). All occupational categories were assumed to have the same demographic characteristics, and these were estimated from industry data (see Table III). Smoking habits were assumed to be the same as those in the general population. Duration of previous exposure by age for the occupationally exposed was assumed to vary between 0 years for 10–19 year olds and 11 years for those over 50 years.[17] For nonoccupationally exposed categories, previous exposure dating back to birth was assumed.

Excess lung cancer incidence rates were assumed to be age dependent and also to vary according to smoking habits, sex, race, and exposure duration and intensity, while excess mesothelioma incidence rates were assumed to be independent of age, sex, race, and smoking habits, to depend on exposure duration and intensity, and to depend on age only through age at onset of exposure. Age-specific mortality rates from other causes were assumed to be constant over time, and the age distribution of the population was assumed to be stable.

Base-line incidence and death rates for lung cancer by sex, race, and age used in the computation were taken from the *Surveillance Epidemiology and End Results* (SEER) study for 1973 through 1978.[18] However, these values were adjusted upward for the older age cohorts because of past smoking rate increases as suggested in Doll and Peto.[19] Initial annual increase rates of 2% for men over 50 and 4% for women over 40 were assumed; these rates of increase were allowed to decline over time.

Death rates for mesothelioma were taken from the SEER study and were assumed to remain constant. Death rates from all other causes were estimated as the difference between death rates from all causes and death rates from lung cancer and mesothelioma. Death rates from all causes by sex, race, and age were estimated based on the 1978 life tables and were assumed to remain constant in the future.[20] All persons still alive at age 89 were assumed to die during their 90th year.

In this paper, the linear, no-threshold dose–response relationships proposed by Nicholson[21] were used to convert information on asbestos exposure into excess lung cancer and mesothelioma incidence rates for each time period.

For lung cancer, Nicholson postulated a relative risk model that includes a minimum 10-year latency period between onset of exposure and increased risk of cancer to give the annual excess risk of lung cancer:

$$I_E = I_L \cdot K_L \cdot f \cdot d_{(t-10)}, \qquad t > 10$$
$$= 0, \qquad t \leqslant 10$$

where I_L is the age-specific lung cancer incidence rate without exposure to asbestos, t is the time from onset of exposure until current age (years), $d_{(t-10)}$ is the duration of exposure from onset until 10 years (latency period) before current age (years), f is the level of exposure (fibers per milliliter, f/ml), and K_L is the dose–response constant.

For mesothelioma, Nicholson postulated an absolute risk model to give annual excess risk of mesothelioma:

$$I_M = K_M \cdot f \cdot \left[(t-10)^3 - (t-10-d)^3\right], \qquad t > 10+d$$
$$= K_M \cdot f \cdot (t-10)^3, \qquad 10+d > t > 10$$
$$= 0, \qquad 10 > t$$

where t is the time since first exposure (years), d is the total duration of exposure (years), f is the level of exposure (f/ml), and K_M is the dose–response constant.

Values used for K_L and K_M were those estimated in the dose–response study of asbestos workers by Selikoff *et al.*,[22] $K_L = 1.0 \times 10^{-2}$, $K_M = 1.5 \times 10^{-8}$. A sensitivity analysis was then performed based on the alternative estimates of these dose–response constants by Seidman *et al.*,[7] where $K_L = 6.8 \times 10^{-2}$, $K_M = 5.7 \times 10^{-8}$. Lower bound values of $K_L = 0.31 \times 10^{-2}$[23] and $K_M = 0.07 \times 10^{-8}$[24] were also used for a sensitivity estimate. K_L and K_M were assumed to be independent of fiber size or type and, thus, are the same for all exposure categories of all products.

The unit measure for exposure level (f) in these equations is fibers per milliliter. These equations were developed from studies that used occupationally exposed workers with a typical exposure to 40 h/week and 50 weeks/yr and a breathing rate of 1 m^3/h. Thus, 1 f/ml is equivalent to $2{,}000 \times 10^6$ f/yr breathed. A normalizing factor of $2{,}000 \times 10^6$ was therefore used to convert exposure data given in fibers breathed per year for both occupationally and nonoccupationally exposed populations into Nicholson's measure of exposure level, f, to use the Nicholson dose–response relationships for all exposed population categories.

Cancer cure rates and annual death rates for the terminal patients were assumed to be constant across demographic groups, exposure categories, and products. These constants were estimated by fitting the equation

$$(\text{relative survival rate})_t = c + (1-c)(1-b)^t$$

for both lung cancer and mesothelioma, where c is the cancer cure rate, b is the annual mortality rate for dying patients, and t is the time since diagnosis (years). Data for these estimations were obtained from Axtell *et al.*[25] and Chahinian.[26] Values estimated and used in the analysis were cure rates of 8% and 2%, and annual death rates of 81% and 71% for lung cancer and mesothelioma, respectively.

6. RESULTS

Estimates of asbestos-related cancer cases attributable to future exposure to all asbestos products manufactured during the 16 years 1985–2000 are

Table IV. Estimates of Asbestos-Related Cancers by Type of Exposure: Occupational or Nonoccupational

Decade	Lung cancer		Mesothelioma	
	Occupational	Nonoccupational	Occupational	Nonoccupational
1985–1994	0	0	0	0
1995–2004	27	2	1	0
2005–2014	75	17	12	3
2015–2024	90	37	35	17
2025–2034	76	61	55	48
2035–2044	49	77	54	94
2045–2054	19	80	32	131
2055–2064	3	69	8	138
2065–2074	0	42	0	101
2075–2084	0	17	0	51
2085–2094	0	4	0	14
2095–2104	0	0	0	2
Total:	339	406	197	599

presented in Table IV separately by type of exposure, occupational and nonoccupational. The projected total number of cancer cases is 1,539 occurring over the next 100 years. These 1,539 cases of cancer occur among the 0.9 million people projected to be occupationally exposed and the approximately 150 million nonoccupationally exposed to the asbestos products manufactured in 1985–2000. Of the people getting lung cancer, approximately 91% die from the disease. For mesothelioma, the death rate is 95%. Of the total number of projected cancer cases, 536 (35%) are predicted to occur in those occupationally exposed and 1,005 (65%) in those nonoccupationally exposed. These results demonstrate that, as occupational exposure levels fall, an increasing proportion of asbestos-related cancers will occur in those exposed to asbestos in the ambient air.

The results presented in Table IV were obtained using the dose–response constants for lung cancer and mesothelioma estimated by Selikoff[22] from his study of insulation workers. In Table V, the implications of using different dose–response constants are seen. $K_L = 6.8 \times 10^{-2}$ and $K_M = 5.7 \times 10^{-8}$ are the dose–response constants estimated by Seidman *et al.*[7] from a study of workers exposed to asbestos for only short time periods (most less than 1 year). Projected cases of cancer were 8,090 as opposed to 1,539 with the Selikoff constants. $K_L = 3.1 \times 10^{-3}$[23] and $K_M = 0.7 \times 10^{-9}$[24] were used to give lower bound estimates, 268 cases projected.

An important feature of the risk assessment method described in this paper is that projections of the calendar time when the excess cancer cases are expected to occur are also obtained as well as the age of the person getting the cancer. Table IV illustrates the timing of the projected excess cancer cases. For the exposed population as a whole, 50% of the excess cancer cases are projected to occur during the years 2025 and 2054. However, when comparing the timeing of onset of disease between those occupationally exposed and those nonoccupationally exposed, it can be seen that the bulk of the health effects are projected to occur 10–20 years later among those nonoccupationally exposed. This is due both to delays in onset of exposure and to the younger ages of some of the nonoccupational group. Age difference at onset of exposure affects the time lag from onset of exposure until onset of lung cancer or mesothelioma. For the occupationally exposed population, the mean time lag from onset of exposure to lung cancer is 34 years, and to mesothelioma, 46 years. For the nonoccupationally exposed population, the mean time lag from onset of exposure to lung cancer is 44 years,

Table V. Sensitivity of the Estimates of Asbestos-induced Cancers to Choice of K_L and K_M, the Dose–Response Constants

Dose–response constants	Total lung cancer cases	Total mesothelioma cases	Total cancer cases
$K_L = 1.0 \times 10^{-2}$ $K_M = 1.5 \times 10^{-8}$	743	796	1,539
$K_L = 6.8 \times 10^{-2}$ $K_M = 5.7 \times 10^{-8}$	5,092	2,998	8,090
$K_L = 3.1 \times 10^{-3}$ $K_L = 0.7 \times 10^{-9}$	231	37	268

Table VI. The Effects on Person-Years and Life Expectancy of Exposure to Asbestos Products Manufactured 1985–2000

	Occupational exposure	Nonoccupational exposure
Person-years of life lost due to asbestos exposure	5,407	12,038
Changes in individual life expectancy due to asbestos exposure		
1. All persons	2.12 days	0.029 days[a]
2. Those contracting an asbestos-related disease	10.09 years	11.98 years
Probability of getting asbestos-induced cancer		
1. Lung cancer	3.1×10^{-4}	2.7×10^{-6}[a]
2. Mesothelioma	2.1×10^{-4}	4.0×10^{-6}[a]

[a]Calculated assuming 150 million people nonoccupationally exposed to asbestos.

and to mesothelioma, 55 years. The mean age of the victims is approximately 71 years for those occupationally exposed and 69 years for those nonoccupationally exposed.

Table VI presents estimates of the person-years of life that would be gained if no asbestos products were manufactured during the period 1985–2000, subdivided according to occupational or nonoccupational exposure. This table also includes estimates of the increase in life expectancy per person exposed if no asbestos exposure occurred, and of the lifetime probability of getting lung cancer or mesothelioma attributable to products manufactured in 1985–2000. These estimates indicate that those relatively few who were occupationally exposed are much more likely to suffer adverse effects at the individual level than those who were nonoccupationally exposed. However, in the aggregate, more person life-years lost attributable to asbestos-induced cancers were estimated for those who were nonoccupationally exposed.

7. DISCUSSION

Projections of asbestos-related cancers over the next few decades attributable to occupational exposure during the time period 1940–1979 have been made by Nicholson *et al.*,[3] Walker *et al.*,[4] and Hogan and Hoel.[2] Their projections, adjusted to refer to a 16-year exposure period, range from approximately 145,000 to 233,000. The large difference between their estimates for the effects of past exposures and the projections presented here for future occupational exposures, 536 cancer cases, can be explained both by the lower levels of asbestos exposure now experienced and the smaller number of people occupationally exposed because of the sharp decline in the use of asbestos products.

Estimates of cancer cases attributable to exposure to asbestos use dose–response constants that have been estimated from epidemiological studies. However, the range of estimated dose–response constants between the studies is large. Table V illustrates that projections of future cancer cases are sensitive to the dose–response constants assumed. Thus, a single numerical estimate of future cancer cases should be interpreted with caution. Alternatively, a range of dose–response constants can be used to derive a range of excess cancers likely to be observed.

The estimates of excess cancers presented are also sensitive to other assumptions made during the estimation procedure. For example, at these low exposure levels, excess cancer estimates are directly proportional to the level of exposures assumed, as well as to the number of people exposed. The excess lung cancer estimates are also directly proportional to the base-line incidence rates assumed for the period 1995–2095. In this paper, 1977 incidence rates were adjusted upwards for the older age groups to reflect past increases in smoking rates in these cohorts. Since smoking rates now appear to be declining in the younger age cohorts, a decline in lung cancer incidence could be projected for the twenty-first century. Such a decline would result in a proportionate decline in predicted excess lung cancer cases for the same time period.

An important property of the risk assessment model described in this paper is that as well as giving estimates of cancer cases attributable to future exposure to asbestos, information is obtained as to when in the future those cases would occur and what the mean ages are of those persons contracting these diseases. This enables us to discount excess cancer cases back to the present at a social rate of discount, if desired, as well as estimate the person-years of life lost as a result of exposure to asbestos.

In comparing the effects of different government policies or regulations, it is apparent that "lives" saved are not a uniform commodity. The following

three questions illustrate important questions that should be asked when using the results of a quantitative risk assessment as an input into regulatory decision making:

1. Should saving the life of a 40 year old be valued the same as saving the life of a 50 year old or a 20 year old?
2. Should saving the life of a 40 year old today be valued the same as saving the life of a 40 year old in 20 years time?
3. Is it of greater value to society to increase the life expectancy of a few people by many days each or to increase the life expectancy of many people by only a few days each?

Only when such questions can be answered, can the estimated risks for different hazards truly be compared. Risk estimates derived using the methodology proposed in this paper allow for a complete specification of the lives saved in terms of age, timing, and size of population at risk.

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