Mortality Effects of Cigarette Smoking Among Amosite Asbestos Factory Workers [1, 2]

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ABSTRACT—Nine hundred and thirty-three amosite asbestos factory workers began work from June 1941 through December 1945. At 20 years from onset of employment (1961–65), 582 were known to be alive and to have had asbestos work experience solely at the factory. Smoking habits were ascertained at 20 years from onset of employment, and observation was then maintained prospectively through 1977; 304 deaths occurred, approximately twice the number expected. The excess mortality was largely due to malignant neoplasms: 116 neoplasms as compared to 33 expected. Sixty cancer deaths were from lung cancer and 14 more were due to mesothelioma. Nevertheless, had it not been for cigarette smoking, many of the excess deaths would have been avoided. Asbestos alone would have accounted for much of the increase, including some excess deaths from lung cancer, but not for most of the deaths (60) from this neoplasm. With the use of smoking-specific mortality rates, it was calculated that the combination of cigarette smoking and asbestos exposure increased the risk of lung cancer death about 80 times. Thus the experience of the amosite asbestos factory workers agreed with that of insulation workers and provided evidence that the asbestos-smoking interaction is specific for asbestos rather than other possible influences in industrial environments.—JNCI 65: 507–513, 1980.

It was shown in 1967 that asbestos exposure sharply increased the already high risk of death from lung cancer associated with cigarette smoking among asbestos insulation workers (*1*). The limited data on which these observations were based were later extended and confirmed by much larger studies, which demonstrated that between 1 in 4 and 1 in 5 deaths among these workers was due to lung cancer if they smoked cigarettes. In addition, as compared to other nonsmokers, nonsmoking insulation workers also had an increased risk of lung cancer, but inasmuch as risk among nonsmokers has been relatively low, even multiplying this risk severalfold did not result in many lung cancers (*2, 3*). Cancer of the esophagus was also considerably increased among smoking insulation workers but not among their nonsmoking co-workers. However, pleural mesothelioma, peritoneal mesothelioma, cancer of the stomach, and cancer of the colon and rectum occurred with approximately equal excess among both smokers and nonsmokers (*3*).

These studies were detailed and prolonged because we preferred to utilize only prospective observations made after establishing the smoking history from the individual concerned, with subsequent observation, and to compare observed deaths with those expected among men of the same age, in the same years, with the same distribution of smoking habits, also observed prospectively. This situation led to some delay in accumulating data to compare with our original observations, but we believe that this method has made the comparisons more secure.

We have been conscious of the fact that our findings have been derived from one category of asbestos-exposed individuals—asbestos insulation workers. Whereas it has been clearly established that these individuals have very significantly increased risks of death from asbestos-associated diseases (*4, 5*), there remained the possibility that other factors in their work environment could have interacted with their cigarette smoking exposure and that some or all of their increased lung cancer risk could be so attributed. It has therefore been of interest to us to investigate the mortality experience of another group of asbestos-exposed workers who did no insulation work and who were not employed in the construction industry.

We now report results from this study. We found that factory workers employed in a plant making amosite asbestos products for shipyards and other industrial facilities had lung cancer mortality experience consistent with that of the insulation workers. Asbestos exposure alone increased the risk of lung cancer in the absence of cigarette smoking, but for reasons given, the total number of such cancers was not great. On the other hand, it appears that the remarkable increase in risk among cigarette-smoking asbestos workers is due

ABBREVIATIONS USED: ACS = American Cancer Society; BE = best evidence available; DC = death certificate information only.

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to the exposure to the mineral dust rather than other factors, multiplying the already very high risk of cigarette smoking.

MATERIALS AND METHODS

In 1941, with the encouragement of the U.S. Navy, a factory was established in Paterson, New Jersey, to manufacture asbestos products for the armed forces and for industrial uses. We have ascertained that amosite asbestos was used almost exclusively (with very small amounts of chrysotile asbestos also being used). Our analyses of fiber retrieved from the factory's storerooms, filters from workers' dust masks, samples of the products made, and fibers extracted from workers' lungs at autopsy have confirmed this almost exclusive use of amosite asbestos and are consistent with the fact that the product specification called for such fiber, that the machinery was designed specifically for this purpose, and that the company that supplied the asbestos from its South African mine had provided only this fiber variety (*6*). This specificity is of some interest because it provides information concerning one of the three major fiber types. Insulation workers had been exposed only to chrysotile asbestos at first and later to both chrysotile and amosite (*5*).

In 1941-45, 933 men began work in this plant. Some worked for a month or less and others until the plant closed in 1954. We have sought to trace the entire cohort of 933 men and to keep them under surveillance. We have already found that this group has, overall, a significantly increased risk of death from asbestos-associated disease (*7*).

Other studies have shown that the risk of death from neoplasms known to be associated with asbestos exposure becomes considerable only after 20 years from onset of exposure to asbestos. We therefore believed it useful to limit our analyses to the experience of the men who worked in this plant, starting with their 20th year from onset of employment and excluding all experience subsequent to asbestos employment elsewhere. For some men (those who began work in 1941) the experience was from 1961 onward. For others, observation for current purposes did not begin until 1965 (i.e., men who began work in 1945). Of the original cohort of 933 men, 351 did not qualify for this analysis. Of these 351 men, 39 had prior asbestos work employment elsewhere, 270 were dead, and 42 were lost to follow-up. Almost all of those lost to follow-up were lost immediately after termination of their factory employment. Actually, this cohort exhibited decidedly unfavorable mortality even before the 20-year point (*8, 9*). Table 1 demonstrates that 582 men came under surveillance at the 20-year point and that the experience for each man from that time to December 31, 1977, included 6,311 man-years of observation. Table 1 also gives the age distribution of these men.

Most of the men in this factory had a history of cigarette smoking. Table 2 outlines the smoking habits of the 582 men we have studied; 430 (73.9%) had a history of regular cigarette smoking (and almost 85% of these were still smoking at onset of observation) whereas 137 men had no such history. Of these 137 men, 59 (10.1%) had smoked pipes and/or cigars but not cigarettes, whereas only 78 (13.4%) had no history of any tobacco smoking. Smoking histories were unobtainable for 15 men (2.6%).

The entire cohort has been under surveillance since 1961, with the majority undergoing periodic clinical examination. For men for whom this periodic examination was not possible, intermittent contact was achieved. Whenever a death occurred, all available clinical and pathologic material was sought and reviewed, including reports of personal medical attendants, hospital records, and histologic material obtained at operation and/or autopsy. For each death a certificate of death was obtained.

Each individual in the cohort remained in the analysis "at risk" from the point at which he reached 20 years from onset of employment during the period 1961-65 until he took up other asbestos employment or otherwise until December 31, 1977, if he was still alive or until date of death if he died before then. The group as a whole then included 6,311 man-years of observation (table 1): 4,811 man-years for individuals with a history of cigarette smoking, 868 for those who never smoked regularly, 528 for those who had smoked pipes and/or cigars, and 104 for those whose smoking history was unknown (table 3).

Using the attained age of each man in each calendar year period after admission to the cohort following the

TABLE 1.—*Number of men at 20-year point and man-years observation, 1961-77, 20 or more years from onset of employment 1941-45, for 582 amosite asbestos factory workers by age*

Age group, yr	No. of men	Man-yr
35-39	84	150
40-44	99	663
45-49	90	1,062
50-54	86	1,192
55-59	65	1,037
60-64	55	787
65-69	40	560
70-74	35	406
75-79	20	266
80-84	6	126
85+	2	62
Total	582	6,311

TABLE 2.—*Number of men at 20-year point and man-years observation, 1961-77, 20 or more years from onset of employment 1941-45, for 582 amosite asbestos factory workers by smoking habits at 20-year point*

Smoking history	No. of men	Man-yr
History of cigarette smoking	430	4,811
Current smokers	359	3,993
Exsmokers	71	818
No history of cigarette smoking	137	1,396
Never smoked regularly	78	868
Pipe and/or cigar only	59	528
Smoking history unknown	15	104

TABLE 3.—*Number of men at 20-year point and man-years of observation, 1961–77, 20 or more years from onset of exposure, 1941–45, for 582 amosite asbestos factory workers by smoking habits at 20-year point and by age*

Age group, yr	History of cigarette smoking		Never smoked regularly		Pipe and/or cigar only		Smoking history unknown	
	No. of men	Man-yr	No. of men	Man-yr	No. of men	Man-yr	No. of men	Man-yr
35–39	67	121	10	19	3	6	4	4
40–44	80	529	10	80	9	39	0	15
45–49	70	837	15	140	3	64	2	21
50–54	70	942	8	166	6	67	2	17
55–59	52	814	6	132	3	69	4	22
60–64	38	630	8	93	8	47	1	[illegible]
65–69	25	433	9	69	6	51	0	7
70–74	19	287	7	67	9	52	0	0
75–79	7	148	4	56	8	62	1	0
80–84	1	49	1	37	3	39	1	1
85+	1	21	0	9	1	32	0	0
Total	430	4,811	78	868	59	528	15	104

20-year-from-onset point, 1961–77, we computed the number of expected deaths for this cohort for these periods of years.

Calculations were made in several ways. First, expected deaths were based on age-specific data of the U.S. National Center for Health Statistics for New Jersey white males (1963–66, 1967–71, and 1972–77) without regard to smoking habits. Expected deaths were also computed in relation to smoking habits on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study (*10*); this analysis was limited to the experience of men other than farmers with at most a high school education and a history of occupational exposure to dust, fumes, chemicals, gases, or radiation. The 1967–71 ACS study rates were extrapolated to 1972–77 according to changes in age-specific death rates among total U.S. white males between 1967–71 and 1972–77. Expected deaths for the few men with unknown smoking habits were computed on the basis of the age-specific and calendar-year-specific rates of all the men (regardless of smoking history) in the ACS study. A third set of expected deaths was computed on the basis of the death rates of the amosite asbestos factory workers who never smoked regularly. To ensure more stable figures, we initially used age-specific death rates for all of the men with experience past 20 years from onset of employment (regardless of smoking habits), 1961–66, 1967–71, and 1972–77. The resulting expected deaths were then proportionately adjusted to levels such that totals of expected and observed deaths were equal for the workers who never smoked regularly.

Observed deaths were then compared with deaths expected. We classified the causes of death in the observed deaths in two ways, according to DC and according to BE. Some investigators may prefer to use the DC figures and argue that the deaths in the populations used as controls have not been subject to a review similar to that of the BE deaths. Their preference assumes that whatever errors exist in the DC figures are much the same in the population being investigated (factory workers) as in the control populations (ACS or U.S. National Center for Health Statistics data). This procedure is certainly far less laborious.

However, some serious difficulties arise. When the distributions of causes of death in the population being investigated and control groups are markedly different, one might anticipate the distributions of error to be different. Mesothelioma and asbestosis have been decidedly underreported on the death certificates of asbestos workers. However, because of the infrequency of these conditions, they have not posed corresponding difficulties in the general population.

Whatever one does, one cannot ensure strict comparability between the cause-of-death classifications for the population investigated and the control populations. In the interest of showing as accurate results as we can for the population being studied, we have oriented the exposition in this paper to the BE findings. However, the DC results have also been shown in the tables for those who prefer such data.

Paralleling the observations for insulation workers (*3, 11*), some differences were observed among the various categories between cause of death as recorded on the death certificate and cause of death established after review (table 4). However, on the whole, this set of findings speaks well for the diagnostic acumen of the medical attendants of these men.

RESULTS

Three hundred and four deaths occurred among the 582 men observed sometime between 1961 and 1965 (the point at which each man reached 20 yr from onset of employment) to December 31, 1977. Cancer of one or another site accounted for 116 of these deaths (38.2%), and 18 deaths were due to asbestosis (5.9%). Table 5 shows that the following cancers in five areas caused most of the deaths: lung cancer (60 deaths); pleural mesothelioma (7 deaths); peritoneal mesothelioma (7 deaths); gastrointestinal cancer including esophagus (16 deaths); and cancers of the oral cavity, pharyngeal, and laryngeal tissues (5 deaths). Altogether, there were approximately three and one-half times as many cancer deaths as expected. The increase in cancer deaths,

TABLE 4.—*Categorization of 304 deaths, 1961–77, 20 or more years from onset of employment, 1941–45, for 582 amosite asbestos factory workers*

Cause of death	Based on DC only	Based on BE	Difference between BE and DC
All causes	304	304	0
Cancer, all sites	103	116	+13
Lung	52	60	+8
Pleural mesothelioma	1	7	+6
Peritoneal mesothelioma	0	7	+7
Mesothelioma not specified above	3	0	−3
Larynx, oral cavity, and pharynx	3	5	+2
Esophagus	1	1	0
Kidney	2	2	0
Colon–rectum	11	11	0
Stomach	4	4	0
Prostate gland	4	4	0
Bladder	2	2	0
Pancreas	5	4	−1
Other and unspecified	15	9	−6
Noninfectious pulmonary diseases, total	29	24	−5
Asbestosis	8	18	+10
Cardiovascular diseases	130	122	−8
Other and unspecified causes	42	42	0

coupled with deaths due to asbestosis, serves to explain most of the virtual doubling of the expected deaths for these men over the period under study. In contrast to the insulation workers, the factory workers had an excess, though a limited one, in mortality from cardiovascular diseases (*3*, *11*).

Data in tables 6 and 7 are organized to shed some light on the excess deaths associated with amosite asbestos exposure, tobacco habits, and amosite asbestos plus tobacco exposures.

(table 6). This observation shows the strong influence of amosite asbestos exposure in itself.

For the total cohort of asbestos factory workers, if expected deaths had been calculated on the basis of the experience of asbestos factory workers who never smoked regularly, it could be estimated that 244 deaths would have occurred, whereas 91 deaths would have been anticipated for men who had neither a history of tobacco smoking nor asbestos employment (table 6). The influence of asbestos alone can be seen as the difference, or 153 deaths. Similarly, if expected deaths were based on men with the same smoking habits, but not with a history of asbestos employment, 137 deaths would have occurred instead of the 91 (table 6). The influence of smoking alone would thus account for 46 deaths. The remainder of 14 when 91+153+46 is subtracted from the observed total of 304 deaths can be regarded either as the joint effect of asbestos and smoking or as discrepancies in data that do not purport to be absolutely consistent. The 304 deaths then are distributed as 30% neither asbestos related smoking related, 50% related to asbestos alone, related to smoking alone, and 5% remaining.

Performing the same computations for the asbe factory workers who were cigarette smokers, we mated that of the 214 deaths (table 6), 57 (27%) neither asbestos nor smoking related, 115 (54%) related to asbestos alone, 46 (21%) were related smoking alone, and −4 (−2%) were the remaind

Although these data can hardly be regarded definitive, they do illustrate the general nature of composite effects evident for all causes of death. composite effects include, among others, specific sm ing effects, asbestos effects—pleural mesothelioma, itoneal mesothelioma, asbestosis, and the excess dea from cancers of the stomach, colon-rectum, oral cav pharynx, and larynx as well as the limited numbe excess deaths from lung cancer among the nonsmok

TABLE 5.—*Observed and expected deaths, 1961–77, 20 or years from onset of employment, 1941–45, among 582 amo asbestos factory workers by cause of death*

Cause of death	Observed deaths		Expected deaths	
	DC	BE	New Jersey[a]	Smokin adjustec ACS stuc
All causes	304	304	158.6	137.1
Cancer, all sites	103	116	33.4	29.8
Lung	52	60	10.1	10.1
Pleural mesothelioma	1	7	Fn e	Fn e
Peritoneal mesothelioma	0	7	Fn e	Fn e
Mesothelioma not specified above	3	0	Fn e	Fn e
Larynx, oral cavity, and pharynx	3	5	1.6	1.0
Esophagus	1	1	0.8	0.6
Kidney	2	2	0.8	0.9
Colon–rectum	11	11	6.2	3.7
Stomach	4	4	2.0	1.4
Prostate gland	4	4	2.7	2.5
Bladder	2	2	1.4	0.9
Pancreas	5	4	1.8	1.8
Other and unspecified	15	9	7.0	6.9
Noninfectious pulmonary diseases, total	29	24	4.7	7.4
Asbestosis	8	18	Fn e	Fn e
Cardiovascular diseases	130	122	95.3	81.2
Other and unspecified causes	42	42	25.2	18.7

[a] Expected deaths were computed on the basis of age-speci data of the U.S. National Center for Health Statistics for N Jersey white males (1963–66, 1967–71, and 1972–77) without gard for smoking habits.

[b] Expected deaths were computed on the basis of age- a smoking-specific experience (1963–66 and 1967–71) of white ma in the ACS prospective study. This analysis involved men with most a high school education and with a history of occupatio exposure to dust, fumes, chemicals, gases, or radiation; farme were excluded from this group. The 1967–71 ACS study ra were extrapolated to 1972–77 according to changes in age-s cific death rates in total U.S. white males between 1967–71 a 1972–77.

[c] New Jersey and ACS study death rates are not available, these diseases have been rare causes of death in the general pop lation.

TABLE 6.—*Observed and expected total deaths, 1961–77, 20 or more years from onset of employment, 1941–45, among 582 amosite asbestos factory workers by smoking habits at 20-year point*

Smoking history	Observed deaths	Expected deaths based on		
		Amosite asbestos factory workers who never smoked regularly[a]	ACS study	
			Men with matching history of smoking[b]	Men who never smoked regularly[c]
History of cigarette smoking	214	171.9	102.3	56.7
Never smoked regularly	38	38.0	15.5	15.5
Pipe and/or cigar only	41	30.7	18.1	17.7
Smoking history unknown	11	3.0	1.2	0.8
Total	304	243.6	137.1	90.7

[a] Expected deaths were initially computed on the basis of age-specific experience (1961–66, 1967–71, and 1972–77) of all men in the cohort 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death total for the men who never smoked regularly equaled their observed death total.
[b] Expected deaths were computed on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study. This analysis was limited to men with at most a high school education and with a history of occupational exposure to dust, fumes, chemicals, gases, or radiation; farmers were excluded from this group. The rates in the ACS study (1967–71) were extrapolated to 1972–77 according to changes in age-specific death rates in total U.S. white males between 1967–71 and 1972–77.
[c] As in footnote *b*, except that expected deaths were computed on the basis of the experience of the men who had never smoked regularly.

asbestos workers—and the very much larger increase in the lung cancer category as a result of the interaction between asbestos exposure and cigarette smoking.

The last phenomenon, the remarkable multiplication of the already high lung cancer risk of cigarette smoking that occurs in asbestos workers, is clearly seen in table 7. The 0.2 deaths were expected to occur among the nonsmoking asbestos workers if their experience had been the same as comparable nonsmokers who had no history of asbestos work. However, 3 such deaths (BE classification) occurred among the 78 men in the group who had no history of regular cigarette smoking. This observation suggests that asbestos exposure alone produces an increased risk of lung cancer and agrees with observations in our cohorts of asbestos insulation workers (*3*). In terms of total number of lung cancer deaths, the increase is limited although undesirable. However, the increase in number of lung cancer deaths among cigarette smokers is devastating. Among 430 men with a history of cigarette smoking were 55 men who died from lung cancer (BE), approximately six times the number of other cigarette smokers without a history of asbestos employment and approximately 80 times the number expected among men of

TABLE 7.—*Observed and expected lung cancer deaths, 1961–77, 20 or more years from onset of employment, 1941–45, among 582 amosite asbestos factory workers by smoking habits at 20-year point*

Smoking history	Categorization	Observed deaths	Expected deaths based on		
			Amosite asbestos factory workers who never smoked regularly[a]	ACS study	
				Men with matching history of smoking[b]	Men who never smoked regularly[c]
History of cigarette smoking	DC	45	28.8	9.6	0.7
	BE	55	16.5		
Never smoked regularly	DC	5	5.0	0.2	0.2
	BE	3	3.0		
Pipe and/or cigar only	DC	2	3.0	0.2	0.2
	BE	2	1.8		
Smoking history unknown	DC	0	0.5	0.1	0.0
	BE	0	0.3		
Total	DC	52	37.3	10.1	1.1
	BE	60	21.6		

[a] Expected deaths were initially computed on the basis of the age-specific DC and BE experience (1961–66, 1967–71, and 1972–77) of all men in the cohort 20 or more yr from onset of exposure. The expected death totals (DC and BE) were then adjusted proportionately so that the expected death totals for the men who never smoked regularly equaled their observed death totals (DC and BE).
[b] Expected deaths were computed on the basis of age- and smoking-specific experience (1963–66 and 1967–71) of white males in the ACS prospective study. This analysis was limited to men with at most a high school education and with a history of occupational exposure to dust, fumes, chemicals, gases, or radiation; farmers were excluded from this group. The rates in the ACS study (1967–71) were extrapolated to 1972–77 according to changes in age-specific death rates in total U.S. white males between 1967–71 and 1972–77.
[c] As in footnote *b*, except that expected deaths were computed on the basis of the experience of the men who had never smoked regularly.

the same age and same personal background but without a history of either tobacco smoking or asbestos employment (0.7 expected and 55 observed).

For other asbestos-related diseases, considerable interest exists in whether the risks of death associated with asbestos exposure are also influenced by cigarette smoking. However, there are only a small number of deaths at hand for use in investigation of these diseases among the amosite asbestos factory workers, especially among the nonsmoking workers.

Table 8 shows the data available for mesothelioma, gastrointestinal cancer, and asbestosis deaths among the factory workers (cigarette smokers and nonsmokers). To supplement these insubstantial findings, table 8 also gives corresponding data for the asbestos insulation workers (Hammond EC, Selikoff IJ, Seidman H: Unpublished data.) The BE classification of causes of death is used because all comparisons in table 8 are within the asbestos worker groups. Moreover, the DC classification does not provide results of any value with respect to the incidence of deaths from mesothelioma and asbestosis.

We found no increased risk of death from mesothelioma in amosite asbestos factory workers with a history of cigarette smoking compared with nonsmokers. The same was true for cancer of the stomach, colon, and rectum. These observations agree with those for the asbestos insulation workers. However, the factory workers do not show the strong influence of cigarette smoking on risk of death from asbestosis that was so striking in insulation workers (table 8).

CONCLUSIONS

The mortality experiences of amosite asbestos factory workers are much the same as those of asbestos-exposed insulation workers, i.e., remarkably increased risk of deaths from asbestosis and cancers of several sites (lung cancer; pleural mesothelioma; peritoneal mesothelioma; gastrointestinal cancer; and oral cavity, pharyng and laryngeal cancers) (*11, 12*). Among men 20 or m years from onset of asbestos exposure, observed p spectively from a 20-year point reached 1961–65 December 31, 1977, 304 deaths were seen, approximat twice the number expected. Eighteen deaths fr asbestosis occurred. Excess deaths, however, were larg due to neoplasms. Neoplasms caused 116 deaths, th and one-half times the number expected. Sixty can deaths were from bronchogenic carcinoma and 14 m were due to either pleural or peritoneal mesothelion

Nevertheless, had it not been for cigarette smokir many of the excess deaths would have been avoid Asbestos alone would have accounted for an importa increase, including some excess deaths from lu cancer, but not for most of the lung cancer deat Here asbestos exposure greatly multiplied the alrea high risk that would have been present with cigare smoking alone. The combination of cigarette smokii and asbestos exposure among these amosite asbest factory workers was calculated to have increased the risk of lung cancer death about 80 times as cor pared with like men of the same age who neith smoked cigarettes nor worked with asbestos. Th increased risk is very much the same as that see among asbestos insulation workers. This observatic indicates that the increased risk of death from lur cancer among cigarette-smoking asbestos workers is specific interaction rather than coincidental, and no for example, the result of other agents in the enviror ment of the construction trades.

The factory in which these men worked closed i 1954, and no further occupational exposure to asbesto occurred for most of the men. For most worker asbestos employment ceased in 1945 at or before th end of World War II. In other studies (*8, 9*) we foun that cessation of occupational exposure to asbestos i this factory was not accompanied by cessation of risl of asbestos-associated disease, including mesothelioma

TABLE 8.—*Observed and expected deaths from selected causes (BE) 20 or more years from onset of employment for 582 amosite asbesto factory workers, 1961–77, and 12,051 asbestos insulation workers, 1967–76, for men with a history of smoking cigarettes compared wit those who never smoked regularly*

Causes of death	Cigarettes or NSR[a]	Amosite asbestos factory workers		Asbestos insulation workers	
		Observed deaths, BE	Expected deaths, NSR[b]	Observed deaths, BE	Expected deaths, NSR[c]
Mesothelioma	Cigarettes	9	14.8	105	108.8
	NSR	3	3.0	15	15.0
Cancer of stomach, colon–rectum	Cigarettes	9	10.1	43	41.5
	NSR	3	3.0	6	6.0
Asbestosis	Cigarettes	11	10.0	97	32.8
	NSR	2	2.0	5	5.0

[a] NSR = never smoked regularly.

[b] Expected deaths were initially computed on the basis of the age-specific BE experience (1961–66, 1967–71, and 1972–77) of all amosite asbestos factory workers 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death totals for the men who never smoked regularly equaled their observed death totals.

[c] Expected deaths were initially computed on the basis of the age-specific BE experience (1967–71 and 1972–76) of all asbestos insulation workers 20 or more yr from onset of exposure. The expected death totals were then adjusted proportionately so that the expected death totals for the men who never smoked regularly equaled their observed death totals.

gastrointestinal cancer, and asbestosis. This finding makes it all the more necessary to repeat an admonition previously made (12): Asbestos workers who smoke cigarettes should stop smoking immediately and those who do not smoke should never begin. Furthermore, all avoidable exposure to asbestos must be prevented.

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