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Gastrointestinal Cancer Mortality of Workers in Occupations With High Asbestos Exposures

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Asbestos, which is a well-known risk factor for lung cancer and malignant mesothelioma, has also been suggested as a gastrointestinal (GI) carcinogen. This study was conducted to assess the relationship between high asbestos exposure occupations and the occurrence of GI cancer. Death certificate data were analyzed from 4,943,566 decedents with information on occupation and industry from 28 states from 1979 through 1990. Elevated proportionate mortality ratios (PMRs) for mesothelioma were used to identify occupations potentially having many workers exposed to asbestos. All PMRs were age-adjusted and sex- and race-specific. The PMRs for GI cancers in white males were then calculated for these occupations after excluding mesothelioma, lung cancer, and non-malignant respiratory disease from all deaths. We identified 15,524 cases of GI cancer in the 12 occupations with elevated PMRs for mesothelioma. When these occupations were combined, the PMRs for esophageal, gastric, and colorectal cancer were significantly elevated at 108 (95% confidence interval = 107–110), 110 (106–113), and 109 (107–110), respectively. Esophageal cancer was elevated in sheet metal workers and mechanical workers. Gastric cancer was elevated in supervisors in production and managers. Colorectal cancer was elevated in mechanical and electrical and electronic engineers. However, high exposure occupations like insulation, construction painter supervisors, plumbers, furnace operators, and construction electricians showed no elevations of GI cancers. In conclusion, this death certificate study supports an association between asbestos exposure and some GI cancer, however the magnitude of this effect is very small. Am. J. Ind. Med. 31:713–718, 1997. *© 1997 Wiley-Liss, Inc.*

KEY WORDS: asbestos exposure; occupation; mortality; mesothelioma; gastrointestinal cancer

INTRODUCTION

Asbestos, which is a well-known risk factor for lung cancer and malignant mesothelioma, was also suggested as a gastrointestinal (GI) carcinogen by Selikoff et al. [1964]. Since then, many studies have explored the relationship between asbestos exposure and GI (esophageal, gastric, intestinal, and colorectal) cancer. In 1985, a report of the presence of asbestos bodies and fibers in the colon and mesentery from a case of asbestosis and adenocarcinoma of the colon increased interest in this topic [Ehrlich et al., 1985]. Translocation of inhaled asbestos fibers from the lung to other organs has been reported, though they have been demonstrated only in the pleura and peritoneum. This means that inhaled asbestos can be translocated into extra pulmonary tissues [Suzuki and Kohyama, 1991].

Some studies have concluded that a positive association may exist between asbestos exposure and GI cancer [Seidman et al., 1986; Hilt et al., 1985; Gerhadsson et al., 1992; Chow et al., 1994]. Others failed to demonstrate an association [Garabrant et al., 1992; Demers et al., 1994]. Further

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reviews and meta-analyses have not clarified the relationship between exposure to asbestos and the risk of GI cancer [Miller, 1978; Morgan et al., 1985; Neugut and Wylie, 1987; Edelman, 1988; Frumkin and Berlin, 1988; Weiss, 1990; Homa et al., 1994; Weiss, 1995].

The uncertainty regarding the risk of GI cancer following exposure to asbestos has raised concerns about a potential risk to human health associated with ingestion of asbestos in drinking water [U.S. DHHS, 1995]. The United States used 28,000 tons of asbestos in 1994 [U.S. Bureau of Census, 1995] and many asbestos-containing materials still remain in the United States environment, although the consumption of asbestos in the United States decreased substantially to 359,000 tons in 1980 from more than 500,000 tons in the 1960s and 1970s. Even though asbestos consumption is decreasing, 3,000,000 tons of asbestos were still produced in the world in 1993 [U.S. Bureau of Census, 1995].

The purpose of this study is to help clarify the relationship between high asbestos exposure occupations and the occurrence of GI cancer.

MATERIALS AND METHODS

We analyzed data from 4,943,566 death certificates collected from 1979 to 1990 through the National Occupational Mortality Surveillance (NOMS)[1] system. The NOMS database includes data from 28 states. The source of information for the NOMS is death certificates, where the cause of death is entered by the attending physician, medical examiner, or coroner, and industry and occupation are entered by the funeral director. The usual occupation and industry of the decedent was coded according to the 1980 Bureau of the Census classification system [U.S. Bureau of Census, 1980]. The underlying cause of death was coded according to the International Classification of Diseases, Ninth Revision (ICD-9) [WHO, 1977]. Further details of the NOMS system have been described elsewhere [Burnett and Dosemeci, 1994].

We computed malignant mesothelioma proportionate mortality ratios (PMRs) for 473 individual and grouped occupational categories by comparing the proportion of deaths due to malignant mesothelioma (ICD codes: 158.8, 158.9, 163) for each occupation with a parallel estimate for decedents in all occupations. All PMRs were age-adjusted and stratified by sex and race. The 95% confidence intervals (95% CI) were calculated based on the Poisson distribution [Bailar and Ederer, 1964] if the observed number of deaths was 1000 or less; otherwise, the Mantel-Haenszel χ^2 analysis was used [Mantel and Haenszel, 1959]. We analyzed the data for only white males, because females and black males had few cases of mesothelioma or no significant elevated PMRs for malignant mesothelioma in the occupational groups. Occupational groups with significantly elevated PMRs for mesothelioma were combined for analysis of GI cancer; a subset of occupations which had PMRs greater than 200 were also combined for analysis of GI cancer.

Because of the lack of a code for mesothelioma in ICD-9, it could have been coded as a malignancy of the pleura or lung, an unidentified cancer or a chronic respiratory disease. The cause of death by asbestosis, which indicates that the decedent was highly exposed to asbestos, is also coded as a non-malignant respiratory disease. After selecting twelve occupations, we removed the records with diagnoses of mesothelioma (158, 163), lung cancer (162), unidentified cancer (199), and non-malignant respiratory diseases (490–519) for the analysis of GI cancer. The PMRs for the GI cancer (ICD codes; 150–154), esophageal (150), gastric (151), and colorectal cancer (153–154) were calculated by comparing the proportion of deaths due to these cancers in the selected occupation with a parallel estimate for all decedents except those with the excluded causes of death. The 95% confidence intervals (95% CI) were calculated based on the Poisson distribution [Bailar and Ederer, 1964] if the observed number of deaths was 1,000 or less; otherwise, the Mantel-Haenszel χ^2 analysis was used [Mantel and Haenszel, 1959].

RESULTS

We identified 1,033 cases of malignant mesothelioma in white males. Twelve occupations had statistically significant elevated PMRs for malignant mesothelioma: insulation workers, construction painter supervisors, plumbers, sheet metal workers, furnace, kiln and oven operators, mechanical engineers, plant stationary engineers, electrical and electronic engineers, construction electricians, supervisors in production occupations, construction carpenters, and managers. These 12 occupations were combined into a group identified as occupations potentially having many workers exposed to asbestos. These occupations experienced a total of 321 cases of malignant mesothelioma among 346,548 deaths. These occupations also tended to have an excess of asbestosis deaths, with 170 cases of asbestosis (Table I).

The PMRs for mesothelioma ranged from 3,667 (95% CI = 2,174–5,796) for insulation workers to 140 (95% CI = 112–172) for managers (Table II). These 12 occupations were combined as the Group and those which showed PMRs greater than 200 for mesothelioma (all except managers, construction carpenters, and supervisors in production occupations) as the Subgroup. We identified 15,524 cases of GI cancer among decedents in the Group and 4,274 cases in the Subgroup. After removing decendents who died of mesothelioma, lung cancer, unidentified cancer, and non-malignant respiratory disease from total death certificates, the overall

1. The NOMS database is supported by individual state vital statistics departments, the National Center for Health Statistics, the National Cancer Institute, and the National Institute for Occupational Safety and Health.

TABLE I. Distribution of Possible Asbestos Related Death-Containing Codes for White Male Decedents in 28 States, 1979–1990

Occupation	Total number of deaths	ICD 9 code A + B	A (163)	B (158.8,9)	C (501)
Insulation workers (593)*	1,079	18	7	11	38
Construction painter supervisors (556)	2,282	6	6	0	1
Plumbers (557, 585, 587)	22,349	49	46	3	40
Sheet metal workers (653, 654)	7,433	14	14	0	9
Furnace, kiln and oven operators (766)	6,257	8	8	0	2
Mechanical engineers (57)	6,956	10	9	1	4
Plant stationary engineers (696)	12,084	15	12	3	4
Electrical and electronic engineers (55)	10,106	11	8	3	1
Construction electricians (555, 575, 576)	27,285	29	28	1	22
Supervisors in production occupations (633)	49,728	37	30	7	18
Construction carpenters (554, 567, 569)	60,990	37	32	5	9
Managers (19)	139,999	87	82	5	22
		321	282	39	170

A (163): Pleural mesothelioma, B (158.8,9): Peritoneal mesothelioma, C (501): Asbestosis.
*The number in parenthesis refers to the Census 3 digit occupation code.

PMRs for GI cancer among occupations potentially having many workers exposed to asbestos was 109 (95% CI = 107–110). The finding was similar when the nine occupations with PMRs greater than 200 for mesothelioma were combined. The PMRs for GI cancer were statistically significantly elevated in the individual occupations of mechanical engineer (PMR = 128), electrical and electronic engineer (PMR = 124), supervisor in production occupations (PMR = 116), manager (PMR = 112), and plant stationary engineer (PMR = 112), which had relatively low PMRs for mesothelioma. The PMRs for GI cancer were not elevated in the occupations of insulation workers, construction painter supervisors, plumbers, sheet metal workers, furnace, kiln and oven operators, construction electricians, and construction carpenters, which had high PMRs for mesothelioma (Table II).

PMRs for the Group were slightly elevated for esophageal (PMR = 108, 95% CI = 104–112), gastric (PMR = 110, 95% CI = 106–113), and colorectal cancer (PMR = 109, 95% CI = 107–111) (Table III). In the Subgroup, the PMR remained elevated for esophageal cancer (PMR = 116, 95% CI = 107–125) and colorectal cancer (PMR = 107, 95% CI = 104–111), but PMR was not significantly elevated for gastric cancer (106, 95% CI = 99–114). For the individual occupations, the esophageal cancer PMR was significantly elevated in three occupations: sheet metal workers, mechanical engineers, and construction carpenters. The gastric cancer PMR was significantly elevated in two occupations: supervisors in production occupations and managers. Four occupations showed significant elevated PMRs for colorectal cancer: mechanical engineers, electrical and electronic engineers, supervisors in production occupations and managers; the PMR for colorectal cancer was decreased for construction carpenters (Table III).

DISCUSSION

Since Selikoff et al. [1964] reported that cancer of the stomach, colon, and rectum was three times as frequent as expected in insulation workers exposed to asbestos, there have been many conflicting reports about the relationship between asbestos exposure and GI cancer [Hilt et al., 1985; Morgan et al., 1985; Zoloth and Michaels, 1985; Seidman et al., 1987].

Garabrant et al. [1992], who did a case-control study of 746 colon cancer cases in Los Angeles county, found a weak association (odds ratio (OR) = 1.16) between exposure to asbestos and colon cancer, but found no association (OR = 0.99) after controlling for the confounders of family history, diet, body weight, and physical activity. They concluded that occupational exposure to asbestos is not a risk factor for colon cancer in the general population of Los Angeles and that observed associations between asbestos and colon cancer should not be interpreted as causal unless confounding by non-occupational factors has been evaluated and controlled.

Gerhardsson et al. [1992], however, reported a different result after controlling for similar confounding factors within the Swedish general population. They conducted a case-reference study of 569 cases of colorectal cancer and found relative risks from 1.8 to 2.2 for males exposed to asbestos. The relative risks did not change after adjustment for diet, body mass, and physical activity. Demers et al. [1994] conducted a case-control study of 261 cases of colorectal cancer and 183 controls in construction workers. This study failed to find that employment in an asbestos-exposed occupation was a risk factor for colorectal cancer. Chow et al. [1994] studied a cancer registry in Sweden, using 19 years of data. They reported increased gastric cancer incidence in some occupations and industries with potential asbestos exposure. The standardized incidence ratio of gastric cancer in masonry and concrete workers, who may have high exposure to asbestos, was 1.2.

Edelman [1988] reviewed 32 independent cohort studies and concluded that there was no dose-response relationship between exposure to asbestos and risk of GI cancer, and concluded that GI cancer was not increased in workers in asbestos cement, insulation, manufacturing, mining and

TABLE II. The PMRs for GI Cancer in Asbestos High Exposure Occupations Based on Mesothelioma PMR for White Males in 28 States, 1979–1990

	Occupation	Total number of deaths	Mesothelioma			GI cancer		
			No.	PMR	95% CI	No.	PMR	95% CI
A	Insulation workers (593)[c]	1,079	18	3,667[b]	2,174–5,796	32	99	68–140
B	Construction painter supervisors (556)	2,282	6	503[b]	163–1,173	89	101	81–124
C	Plumbers (557, 585, 587)	22,349	49	483[b]	358–639	923	102	96–109
D	Sheet metal workers (653, 654)	7,433	14	404[b]	221–678	336	110	99–123
E	Furnace, kiln and oven operators (766)	6,257	8	287[b]	124–566	237	92	81–105
F	Mechanical engineers (57)	6,956	10	280[b]	128–532	389	128[b]	115–141
G	Plant stationary engineers (696)	12,084	15	279[b]	156–460	572	112[b]	103–121
H	Electrical and electronic engineers (55)	10,106	11	241[a]	121–432	549	124[b]	114–135
I	Construction electricians (555, 575, 576)	27,285	29	236[b]	160–343	1,147	104	98–109
J	Supervisors in production occupations (633)	49,728	37	161[b]	114–222	2,465	116[b]	112–120
K	Construction carpenters (554, 567, 569)	60,990	37	146[a]	103–201	2,163	94[b]	90–98
L	Managers (19)	139,999	87	140[b]	112–172	6,631	112[b]	110–115
	Subgroup (A–I)	95,831	160	366[b]	311–428	4,274	108[b]	105–111
	Group (A–L)	346,548	321	206[b]	184–230	15,524	109[b]	107–110

[a]$P < 0.05$.
[b]$P < 0.01$.
[c]The number in parenthesis refers to the Census 3 digit occupation code.

TABLE III. PMRs for Specific Cancer Sites in Asbestos High Exposure Occupations for White Males in 28 States, 1979–1990

	Occupation	Esophageal		Gastric		Colorectal	
		No.	PMR (95% CI)	No.	PMR (95% CI)	No.	PMR (95% CI)
A	Insulation workers (593)	5	103 (33–241)	5	83 (27–195)	22	104 (65–158)
B	Construction painter supervisors (556)	16	132 (75–214)	18	113 (67–178)	54	91 (68–118)
C	Plumbers (557, 585, 587)	149	118 (100–138)	170	105 (90–122)	595	99 (91–107)
D	Sheet metal workers (653, 654)	62	142 (109–182)	66	120 (93–153)	207	102 (89–117)
E	Furnace, kiln and oven operators (766)	36	104 (73–143)	37	81 (57–112)	163	94 (80–109)
F	Mechanical engineers (57)	57	132 (100–171)	57	104 (79–135)	271	133 (118–150)
G	Plant stationary engineers (696)	79	112 (89–140)	109	119 (98–144)	380	110 (100–122)
H	Electrical and electronic engineers (55)	66	103 (80–131)	97	120 (98–147)	384	130 (118–144)
I	Construction electricians (555, 575, 576)	173	111 (95–129)	198	99 (86–114)	761	103 (96–111)
J	Supervisors in production occupations (633)	313	105 (94–118)	471	124 (113–136)	1,651	116 (111–121)
K	Construction carpenters (554, 567, 569)	357	116 (104–128)	416	101 (91–111)	1,371	89 (84–93)
L	Managers (19)	828	100 (93–107)	1,183	111 (105–117)	4,530	115 (112–118)
	Subgroup (A–I)	643	116 (107–125)	757	106 (99–114)	2,837	107 (104–111)
	Group (A–L)	2,141	108 (104–112)	2,827	110 (106–113)	10,389	109 (107–110)

milling, nitric acid production, railroad, and shipyard industries. Frumkin and Berlin [1988] performed a meta-analysis of 31 cohort studies of asbestos workers and concluded that significant asbestos exposure was associated with an elevated GI cancer standardized mortality ratio (SMR) in the group that had a lung cancer SMR of at least 200. However, Weiss [1990] did not find a causal relationship between asbestos and colorectal cancer after reanalyzing 21 cohorts published before 1988. Recently, Homa et al. [1994] conducted a meta-analysis which showed that amphibole asbestos may be associated with colorectal cancer, but that serpentine asbestos is not. But, Weiss [1995] after examining

summary SMRs of 30 cohorts published before 1993 concluded that neither a causal relationship between asbestos and colorectal cancer exists nor could the type of asbestos support causality. Because of these contradictory results, it is currently very difficult to justify a conclusion that GI cancer is related to asbestos exposure.

We analyzed the NOMS death certificate data to look at the relationship between asbestos exposure and GI cancer. Since the NOMS database includes information on the usual industry and occupation of the decedent, we were able to examine PMRs for GI cancer among occupations which are likely to have work-related asbestos exposure. If exposure to asbestos is related to GI cancer, the occupations with elevated ratios of mesothelioma should show elevated PMRs for GI cancer.

Most cases of mesothelioma are caused by asbestos exposure. Spirtas et al. [1994] calculated the attributable risk of pleural and peritoneal mesothelioma from exposure to asbestos in males as 88% and 58%, respectively using clinical records. For this reason, we used the elevated ratios of malignant mesothelioma as our indicator of asbestos exposure, while some studies used lung cancer mortality which has many different causal factors [Doll and Peto, 1987; Frumkin and Berlin, 1988; Weiss, 1990]. The finding of high PMRs of malignant mesothelioma for occupations of manager, construction painter supervisor, and supervisor in production occupations was unexpected. However, it is plausible that many experienced workers' final occupation which might be on the death certificate could be described as manager or supervisor.

Insulation workers, the group that was first reported [Selikoff et al., 1964] as having increased GI cancer and that had the highest PMR for mesothelioma in our study, did not show elevated PMRs for GI cancer. The insulation workers' result makes the causal relationship questionable although PMRs for GI cancer of the Group and Subgroup were statistically elevated. For specific sites, our study showed that PMRs for the Group and Subgroup were statistically significantly elevated for esophageal, gastric, and colorectal cancer even though the PMRs for some GI cancers in the occupations which had high PMRs for mesothelioma were not elevated. These results were similar to those reports by Selikoff et al. [1979] that gastric and colorectal cancer were elevated in insulation workers, and by Morgan et al. [1985] that GI cancer except esophageal cancer might be caused by asbestos exposure.

The major advantage of our study is that it included all white male deaths for the reported states, so it had a sufficient number of cases of GI cancer to provide substantial statistical power. However, this study shares the limitations of other PMR studies using death certificates.

There are problems of misclassification for usual industry and occupation since this information is collected by funeral directors from the next-of-kin [Selikoff, 1992]. This may cause a bias that can give variable results, especially for workers whose exposures occurred in the distant past, who had multiple occupations, or whose family reported an elevated occupational status for the decedent. Some workers might be classified to asbestos non-exposure occupations even though they worked in an environment with asbestos exposure and vice versa.

Another bias may arise from the PMR method of analysis. The PMRs could be biased because the PMRs for GI cancer might be affected by increases in other diseases caused by asbestos exposure. Therefore, to reduce the bias from the PMR method, which could be affected by asbestos exposure, we excluded mesothelioma, lung cancer, and non-malignant respiratory diseases in calculating PMR for GI cancer. However, there is still a possibility that the PMRs for GI cancer in these groups could be overestimated because the workers in these occupations may be less affected by diseases which are common in less healthy people. [Decoufle et al., 1980].

Another potential limitation of the study is misclassification of the disease reported on the death certificate. However, the information on cancer on death certificates has been shown to be more accurate than for other causes of death [Gittelsohn and Senning, 1979; Percy et al., 1981]. Gittelsohn and Senning [1979] found that gastric and esophageal cancer showed a high agreement between death certificates and hospital records. Selikoff and Seidman [1992] found that death certificate information was not different from the best evidence in the case of gastric cancer.

The ICD-9 does not have a specific code for malignant mesothelioma. The correct classification of mesothelioma in the ICD-9 is to the categories of malignant neoplasm of peritoneum (ICD-9: 158.8, 158.9) or pleura (ICD-9: 163). Death certificates often lack the requisite information to correctly code mesothelioma. It is often misclassified as either lung cancer or a malignancy of unspecific site (ICD-9: 199.1) [Lilienfeld and Gunderson, 1986]. In a Massachusetts study, Davis et al. [1992] reported that ICD 158.8 code did not capture mesothelioma cases and 158.9 captured only 31% of mesothlioma cases. In our study, there are relatively few cases of 158.8 and 9, and most cases came from insulation workers, who were exposed to high level of asbestos. Therefore, although it is not possible to select all deaths due to malignant mesothelioma using ICD 158.8, 9 and 163 code, these codes can be used to identify the occupations potentially having many workers exposed to asbestos until the ICD 10 code is available. Any missing cases of mesothelioma on the death certificate would not affect the identification of high risk occupations, unless this misclassification of mesothelioma occurred selectively.

Death certificates do not provide information on diet, exercise, drinking, smoking, socioeconomic status, and other possible confounding factors for GI cancer. Therefore, we were unable to control for their potential confounders. The results of a study by Ward et al. [1994] indicate that high socioeconomic status may be related to gastric cancer. Those

occupations except managers which had presumed high asbestos exposure in our study can be classified as low socioeconomic status, which may tend to lower the PMRs for GI cancer. Drinking and smoking, for which there is no information, may related to esophageal cancer [Weiss, 1990].

In conclusion, the PMR for GI cancer was statistically significantly elevated when high asbestos exposure occupations were analyzed as a group. However, a consistently high elevated PMR for GI cancer did not appear for each occupation group, especially insulation workers, which showed the highest PMR for malignant mesothelioma. This death certificate study supports on association between asbestos exposure and some GI cancer, however the magnitude of this effect is very small.

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