

OCCUPATIONAL EXPOSURES AND THE RISK OF COLON CANCER

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ABSTRACT

Persons performing various occupations have been reported as at-risk for colorectal cancer by prior investigators. Occupational exposures have been inferred as risk factors based upon assumed levels of exposure, particularly to asbestos. This population-based incident case (n=1449) – control (n=1763) study of colon cancer utilized participants from Utah, Minnesota, and northern California. Occupational exposures to asbestos, benzene, electromagnetic fields, formaldehyde, lead, and solvents were assigned using a job exposure matrix. Potential confounders were evaluated including tobacco, nonsteroidal anti-inflammatory drugs, physical activity, income, family history and dietary factors, age, and gender. Associations were estimated from logistic regression models with the final model adjusting for age, gender and other occupational exposures. Data were also explored for potential interaction between tobacco and asbestos.

INTRODUCTION

Colorectal cancer is the second leading cause of cancer death in the United States and is the third most common cancer in men and women. While diet, low physical activity and omission of non-steroidal anti-inflammatory drugs (NSAIDs) have been associated with increased risk, data are somewhat inconsistent regarding occupational exposures and apparent risk of colorectal cancer. Studies have reported excess colorectal cancer rates in industrial workers in several industries, and others studies have attributed increased risk for various occupational exposures, especially to asbestos. However, few of these studies have controlled for non-occupational risk factors, such as physical activity, family history, or diet.

This case-control study investigated the potential association between asbestos, electromagnetic fields (EMF), formaldehyde, lead and solvent exposure and colon cancer risk.

METHODS

We identified 1449 colon cancer cases from the metropolitan Minneapolis/St. Paul, Minnesota, the State of Utah, and the Kaiser Permanente Medical Care Program (KPMCP) in Northern

California from 1991-1994 who had complete data available. A total of 1763 randomly selected community controls were derived from these same populations who had a complete dataset. A structured personal interview was used to collect detailed data on occupation, diet, medical history, family history of cancer, lifestyle, physical activity, and NSAID/aspirin use. The intensity of occupational exposures to asbestos, EMF, formaldehyde, lead and solvents were calculated from the participants' occupational history using a National Cancer Institute (NCI) job exposure matrix (JEM), with a final numerical score between 0-9 (from none to high, respectively) assigned.

Adjusted odds ratios (OR) and 95% confidence intervals (CIs) were calculated using logistic regression. Potential confounders evaluated were: sex, age, race, education, income, family history, BMI, vigorous physical activity, regular use of NSAIDs or aspirin, smoking, and alcohol intake. However, inclusion of most of these potential confounders caused little alteration in the final point estimates. Thus, the final model included sex, age, and adjustment for other occupational exposures.

Effect modification between colon cancer risk and occupational exposure was evaluated, particularly between tobacco and asbestos.

RESULTS

Colon cancer cases are generally more likely than controls to be older and more likely to be current or former smokers (See Table 1).

The association between colon cancer risk and the intensity of occupational exposures is given in Table 2. After adjusting for age and gender, there is no evidence of an association between colon cancer risk and exposure(s) to asbestos, EMF, formaldehyde, lead or solvents.

The association between colon cancer risk and asbestos exposure stratified by cigarettes consumed per day (never vs. 1-19 vs. 20+) is in Table 3. There is no evidence that smoking modifies a relationship between colon cancer and occupational asbestos exposure.

DISCUSSION

In this population-based case-control study, we found no evidence that occupational exposures to asbestos, EMF, formaldehyde, lead or solvents are associated with colon cancer risk. We also found no evidence of interaction between asbestos and tobacco.

CONCLUSION

In this study, asbestos and other occupational exposures examined were not associated with colon cancer.

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Table 1. Selected Characteristics of Study Population Colon Cancer Cases and Controls

	Colon Cases (n=1449)		Controls (n=1763)	
	n	(%)	n	(%)
Gender				
Male	805	(55.6)	962	(54.6)
Female	644	(44.4)	801	(45.4)
Age				
30-54	249	(17.2)	320	(18.2)
55-59	164	(11.3)	180	(10.2)
60-64	222	(15.3)	263	(14.9)
65-69	289	(19.9)	351	(19.9)
70 +	525	(36.2)	649	(36.8)
Cigarettes Smoked per Day				
0	592	(40.9)	813	(46.1)
1-19	281	(19.4)	373	(21.2)
20+	569	(39.3)	572	(32.4)

Table 2. Occupational Exposure and Risk of Colon Cancer

	Colon Cases (n=1449)	Controls (n=1763)	Risk of Colon Cancer
	n	n	OR 95% CI
Asbestos			
None	1137	1434	1.0 (---)
Low	185	195	1.2 (0.9-1.4)
High	127	134	1.0 (0.8-1.3)
p-trend			0.498
Electromagnetic Fields			
None	1130	1358	1.0 (---)
Low	180	232	0.9 (0.7-1.1)
High	139	173	0.9 (0.7-1.2)
p-trend			0.353
Formaldehyde			
None	1218	1483	1.0 (---)
Low	82	101	0.9 (0.7-1.3)
High	149	179	1.0 (0.8-1.3)
p-trend			0.825

Table 2. (cont'd)

Lead			
None	1166	1448	1.0 (---)
Low	173	204	0.9 (0.7-1.2)
High	110	111	1.1 (0.8-1.4)
p-trend			0.729
Solvents			
None	1008	1237	1.0 (---)
Low	242	316	0.9 (0.7-1.1)
High	199	210	1.1 (0.8-1.4)
p-trend			0.850

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Table 3. Asbestos and Risk of Colon Cancer Stratified by Smoking Status

	Never Smoked OR (95% CI)	1-19 Cigarettes/day OR (95% CI)	20+ Cigarettes/day OR (95% CI)
Asbestos			
None	1.0 (---)	1.0 (0.8-1.3)	1.4 (1.2-1.7)
Low	1.2 (0.8-1.7)	0.9 (0.5-1.7)	1.1 (0.7-1.9)
High	1.3 (0.8-2.1)	1.0 (0.4-2.1)	0.8 (0.4-1.5)
p-trend	0.240	0.817	0.596
p-interaction			0.852

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