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CAMBRIDGE

TO: H.C. Duecker
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DATE: April 26, 1977

FROM: H.A. Eschenbach

SUBJECT: Attached

PLAINTIFF'S EXHIBIT WRG-1432A

CONFIDENTIAL

This article may be of value if people in the Enoree or Libby communities raise questions concerning health hazards associated with living in those areas. While the study just refers to "deposits" and does not indicate whether any of these deposits are mined and/or milled, the data is encouraging.

H.A. Eschenbach

HAE:bp

Attachment

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lesions.

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CANCER MORTALITY AND ASBESTOS DEPOSITS

THOMAS J. FEARS

Fears, T.R. (Office of Field Studies and Statistics, National Cancer Institute, 7910 Woodmont Avenue, Bethesda, MD 20014): Cancer mortality and asbestos deposits. *Am J Epidemiol* 104:523-526, 1976.

Asbestos deposits are found in many parts of the United States. In this paper the question is asked: Is there an increase in risk from cancer associated with naturally occurring asbestos? In an attempt to control for the urban effect, geographic gradient and socioeconomic class, each county in the United States with asbestos deposits was matched for percent of area that was urban and for median years in school with two nearby counties that did not have known asbestos deposits. The study of cancer mortality rates in these matched counties provides no evidence that naturally-occurring asbestos is a great hazard to the general population of counties with asbestos deposits.

asbestos; cancer; carcinogens; environment; mortality

Introduction

Epidemiologic studies have found that asbestos workers display an extremely high risk of dying from lung cancer. In addition, incidence of cancer of other sites, esophagus, stomach, colon and rectum, are higher than average (1). The production of tumors in animals by inhaled asbestos has been difficult to demonstrate (2) but there have been examples of experimental carcinogenesis following injection or implantation of asbestos. There is evidence that asbestos fed to animals enters the gut wall, lungs and other organs.

Asbestos in the form of chrysotile and amphibole deposits is found in many parts of the United States (3, 4). In this paper we ask: Is there an increase in risk from cancer associated with naturally-occurring asbestos deposits?

Materials and Methods

Locations of asbestos deposits in the United States are given in *Asbestos in the United States* by A. H. Chidester and A. F. Shride, published by the US Geological Survey of the Department of the Interior (5). Their text lists counties with asbestos deposits and distinguishes those deposits as to mineralogic type, name of the deposit or prospect and associated references. With this as a guide, each county was classified as to type of asbestos present—having chrysotile deposits or not, having amphibole (including anthophyllite and tremolite) or not. There were 99 counties with asbestos deposits; 54 with amphibole and 45 with chrysotile.

Information on cancer mortality has been gathered by the National Center for Health Statistics and analyzed geographically by the Epidemiology Branch of the National Cancer Institute. Age-adjusted death rates over the period 1950-1969 were obtained for counties in the contiguous forty-eight states by race, sex, and site of cancer (6). The standard for the rate calculations was the total US population in 1960.

Two asbestos deposit counties, each with amphibole, were eliminated from the study as the cancer mortality data from these counties were combined with other nearby counties.

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The author thanks Drs. Marvin A. Schneiderman and Thomas J. Mason for their useful suggestions.

throughout. Only sites for which there is their controls was interpreted to [illegible]

[illegible] evidence of asbestos-caused cancer are "effects" of asbestos entering into the envi[illegible]

Lied. Specifically, we use Junk Interna[illegible]ronment on cancer mortality rates. For

[illegible] Classifications of Diseases (ICD) each of the study sites, for males and for

[illegible] (ICD 150), stomach (ICD 151), [illegible] females, for chrysotile and amphibole, as

[illegible] (ICD 153), rectum (ICD 154), [illegible] well as for all study counties the asbestos

[illegible] (ICD 155), and total for the gastro[illegible] effect was measured and tested using a

[illegible] as well as the total for the respira[illegible] paired t test. Normality of the distribution

[illegible] (ICD 160-163, 165, 193-194). [illegible] of differences is important for the validity

[illegible] for white males and females were [illegible] of the t test but moderate departures have

[illegible] studied since the black populations of the [illegible] been shown to have little effect (8). Wil[illegible]

[illegible] counties were, in general, small [illegible] coxon rank tests for each subgroup of coun[illegible]

An initial study of the data (table 1) [illegible] ties and for each study site were calculated

revealed that cancer mortality rates in the [illegible] as a precaution against distortions in prob[illegible]

asbestos deposit counties were substan[illegible] ability statements because of possible de[illegible]

tially below the rates of the total US. The [illegible] viations from normality.

asbestos counties are heavily rural, which [illegible] Table 2 gives the average annual age-

might account for the low rates. [illegible] adjusted (1960) cancer mortality rates for

In an attempt to control for the urban [illegible] 1950–1969 among white males and females

factor, geographic gradient, and socioeco[illegible] in each subgroup of the asbestos counties

nomic class, the following approach was [illegible] and in their respective matched controls.

used. For each asbestos deposit county, was [illegible] Using the t and Wilcoxon statistics, no

matched for percent of area that was urban, [illegible] significant differences (all $p > .10$) for

for median years in school with two asbestos county mortality rates compared

counties that did not contain known asbest[illegible] with control county mortality rates were

deposits. The matched counties were [illegible] found for males or for females. No signifi-

same or an adjacent state. An analysis [illegible] cant differences (all $p > .10$) for chrysotile

then made of the differences between [illegible] or amphibole were seen.

cancer rates of an asbestos deposit [illegible]

Multiple regression

A county was classified as a county with

ically and the average rate of its demo[illegible]

graphically matched counties. This match[illegible]

is particularly appropriate because of [illegible] asbestos of a given specific type if any

epidemiologic features of cancer of the [illegible] type. Since some counties contained both

Esophageal cancer mortality rates [illegible] chrysotile and amphibole the examination

of the counties by type is not sufficient for

a high degree of geographic variation [illegible] understanding. A multiple regression anal-

the incidence is higher in urban areas [illegible] ysis was made using a model with terms for

in adjacent rural areas. Stomach [illegible] type of deposit (amphibole, chrysotile and

rates are higher among lower socio[illegible] both) for white male and white females for

economic classes. Large bowel cancer rates [illegible] each study site. An F statistic with 3 and 91

higher for urban than rural residents, [illegible] degrees of freedom was used to test the

associated with increased risk of [illegible] significance of proportion of the total varia-

cancer include low socioeconomic sta[illegible] tion explained by the model by asbestos

and urban residence. [illegible] type. No significant increases in cancer

mortality were found for any study site for

either males or females. The individual

regression coefficients were tested with t-

The mean difference between cancer [illegible] tests. No coefficient in any model was

mortality rates of asbestos counties and [illegible] significant (all $p > .05$). The constant term

TABLE 1

Cancer mortality rates 1950–1969 for various sites—asbestos counties compared to US totals (deaths per 100,000)

Sex and location	Esophagus (ICD 150)	Stomach (ICD 151)	Large intestine (ICD 153)	Rectum (ICD 154)	Total gastro-intestinal tract (ICD 150–154)	Biliary passages and liver (ICD 155)	Bronchus and lung (ICD 162–163)
White Males							
Asbestos Counties	3.14	13.25	12.68	5.69	34.76	4.21	31.86
Total U.S.	4.10	15.22	16.54	7.65	43.51	5.16	37.98
White Females							
Asbestos Counties	.97	6.95	13.25	3.96	25.13	4.65	5.38
Total U.S.	1.03	7.70	16.25	4.82	29.80	5.34	6.29

TABLE 2

Mean age-adjusted cancer mortality rates 1950–1969 by cancer site and type of asbestos deposit for white males and white females in asbestos deposit counties and matched counties (deaths per 100,000)

Counties	No.	Esophagus (ICD 150)		Stomach (ICD 151)		Large intestine (ICD 153)		Rectum (ICD 154)		Total gastro-intestinal tract (ICD 150–154)		Biliary passages and liver (ICD 155)		Bronchus and lung (ICD 162–163)	
		Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Chrysotile counties	45	3.14	.95	14.18	7.09	13.37	13.42	5.95	4.31	36.64	25.77	4.26	4.74	31.97	5.77
Control counties	95	3.07	.96	14.57	6.91	13.19	13.66	6.34	4.43	37.17	25.96	4.08	4.61	32.35	5.29
Amphibole counties	52	3.13	.96	12.77	6.83	11.58	12.54	5.20	3.69	32.68	24.02	4.19	4.75	31.10	5.14
Control counties	104	2.79	.88	13.81	6.38	12.15	12.94	5.45	3.58	34.20	23.78	4.22	4.55	32.77	5.18
All asbestos counties	97	3.14	.97	13.25	6.95	12.68	13.25	5.69	3.96	34.76	25.13	4.21	4.65	31.86	5.38
All control counties	194	2.96	.99	13.75	6.64	12.59	13.50	5.72	3.96	35.02	25.09	4.20	4.54	32.59	5.23

Analysis by type

RESULTS

was not significant (all $p > .10$) in any tests. The per cent of varintion in the data explained by the model did not exceed 10 per cent for any site or sex.

Discussion

While the results of this study are negative, the interpretation is not easily made. Problems which complicate the interpretation are: the lack of exposure information; the use of populations as the unit of study; migration within the US; other sources of asbestos; and the presence of epidemiologic features other than asbestos which could modify cancer mortality.

No measurements of asbestos exposure to individuals have been taken by us in the study or in control counties. We assumed that if there were asbestos deposits present, then there would be an elevated exposure. If in fact the presence of asbestos deposits does not lead to elevated exposure to asbestos, then no excess mortality should be seen.

We have used as our unit of study the population of counties with asbestos deposits and concerned ourselves with population mortality rates. There are many problems in making inferences about the risks to an individual in a county. For example, the general exposure of the population in the study counties can be very small, but a few individuals can have increased exposure, in which case the population hazard need not reflect individual hazard. The elevated individual hazard would in this case not necessarily be detected.

Finally, other factors of etiological significance have not been included in this study. It is possible that adjustment for these factors might [illegible] detection of asbestos' effect [illegible] sufficiently detailed information on smoking, alcohol, food, air, water, etc., are not available.

This study provides no evidence that naturally occurring asbestos deposits provide a great risk to the general population of counties with asbestos deposits.

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SEX, BIRTH ORDER, AND MATERNAL AGE CHARACTERISTICS OF INFANTS WITH CONGENITAL HEART DEFECTS

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Rothman, K. J. and Fyler, D. C. (Department of Epidemiology, Harvard School of Public Health, Boston, MA 02115). Sex, birth order and maternal age characteristics of infants with congenital heart defects. *Am J Epidemiol* 104:527-534, 1976.

The records of the New England Regional Infant Cardiac Program, a service program covering all of New England, provide a useful source of information about the characteristics of children born with congenital heart defects. Data were analyzed on more than 2000 children born in New England who were diagnosed as having a congenital heart defect before the first birthday. Children with arterio-venous fistula, aortic stenosis, transposition of the great arteries or hypoplastic left ventricle were predominantly male; children with persistent ductus arteriosus and endocardial cushion defect were predominantly female. Positive trends in risk with increasing birth order were present for pulmonic stenosis and transposition of the great arteries, and a negative trend was seen for persistent ductus arteriosus. What evidence there was for associations with maternal age was greatly reduced after controlling for confounding by birth order.

aortic diseases; aortic valve stenosis; arterio-venous fistula; heart defects, congenital

The New England Regional Infant Cardiac Program (NERICP) is a service program designed to provide medical care for all infants with serious congenital heart disease born in New England (1). The population coverage and centralized record-keeping provide the equivalent of a regional registry of congenital heart disease. In a previous report (2) we used these data to describe the associations of congenital heart defects with season and with population density. In this paper we present the sex, birth order and maternal age characteristics of infants with congenital heart defects.

Subjects and Methods

Enrollment of patients into the NERICP began July 1, 1968, and 2336 children under one year of age born in New England with serious cardiac defects were enrolled by June 30, 1974. When the program

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Abbreviations: NERICP, New England Regional Infant Cardiac Program; TOA, transposition of the great arteries.

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The authors express their gratitude to the many individuals whose cooperative efforts make NERICP successful. Participating centers in NERICP are the pediatric divisions of the following hospitals: Hartford Hospital and St. Francis Hospital, Hartford; Yale-New Haven Hospital, New Haven; Maine Medical Center, Portland; Boston City Hospital, Boston Floating Hospital for Infants and Children, Children's Hospital Medical Center, Massachusetts General Hospital, Boston; Dartmouth-Hitchcock Medical Center, Hanover; Rhode Island Hospital-Brown University, Providence; Medical Center Hospital of Vermont (Mary Fletcher Unit), Burlington.

Exhibit 1432