PLAINTIFF'S EXHIBIT

CAP-1354

A/C Pipe
Producers Association

Internal Correspondence

TO Executive Committee
International Affairs Committee

FROM J.F. Welch
J.F. Welch, Vice President

DATE September 7, 1982

SUBJECT U.S. Environmental Protection Agency (EPA) - Epidemiological Study

ACTION REQUIRED: Review for information

Enclosed is the study, "Cancer Incidence in Relation to Asbestos in Drinking Water in the Puget Sound Region," as published in the August, 1982 issue of American Journal of Epidemiology. Like the Kanarek studies, an ecologic design was used in which the observational units were census tracts. Thus, the findings, however favorable, are subject to the same shortcomings. The study is somewhat less positive than the original work, "A Study of the Effects of Asbestos in Drinking Water on Cancer Incidence in the Puget Sound Region," by Richard Keith Severson of the University of Washington. In his Master of Science thesis, Severson concluded that imbibed asbestos in the Puget Sound Region was not related to alimentary tract cancer.

The enclosure concludes:

> Results of this study and prior studies of cancer in relation to waterborne asbestos are inconsistent, and provide little evidence that asbestos in community water supplies has altered the risk of any cancer.
>
> * * *
>
> Results of the present study confirm none of these findings (Ed. note: relationship between asbestos in water and neoplasms of rectum, stomach, pancreas, lung, peritoneum, esophagus and pleura)...and most of the findings in the various published studies probably represent chance phenomena resulting from large numbers of comparisons.
>
> * * *
>
> From the data we have analysed, we conclude that the cancer risk from asbestos in drinking water in the Sultan River area is, at most, very low, although there is suggestive evidence for some anatomic sites.

The sites to which the author refers are the small bowel and pancreas. The study observed consistent, but not statistically significant, associations between small bowel cancers and asbestos in drinking water. After noting these associations, the investigators give three reasons "...to doubt that the observed relationship is causal."

Polissar also suggests a possible link between pancreatic cancer and waterborne asbestos, referring to five previous studies that showed some association in one or both sexes. But again, he goes back to middle ground and states, "...our results neither confirm nor refute prior findings." As an aside, Staff is not aware of data demonstrating increased incidence of pancreatic cancer in highly exposed occupational groups. In the absence of such data, Polissar's suggestion of a waterborne asbestos-pancreatic cancer link is no more than speculation.

Polissar et al also are conducting a case control epidemiological study based on these exposure and cancer incidence/mortality data. A preliminary report will be finished for the EPA Summary Workshop on Ingested Asbestos.

Staff Analysis

This study is noteworthy in three respects. First, the findings are consistent with all other epidemiological studies (except Kanarek) of cancer and asbestos in drinking water. Second, putting aside the variable of population mobility, the exposure period (more than 50 years) is longer than the latency period for asbestos-related disease. Third, the so-called "signal tumor" for asbestos exposure (mesothelioma) did not even appear in sufficient numbers for analysis. Although the scientific caveats of the Polissar study give it less positive impact than the Meigs or Wigle studies, the industry should be encouraged by EPA's summarization of the findings in the July 28, 1982 Drinking Water Research Progress Report:

> The descriptive epidemiology study does not show any relationship between asbestos in water and increased cancer risk.

Also enclosed is a news clipping from the Seattle Times that includes some very positive quotes about the Polissar study...moreso than the study itself.

If you have any questions, please do not hesitate to call.

JFW/ccw
Enclosure

cc: A. Kahn, Esq.
AIA
AIA/NA
W. Smith, M.D.
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CANCER INCIDENCE IN RELATION TO ASBESTOS IN DRINKING WATER IN THE PUGET SOUND REGION

LINCOLN POLISSAR,[1,2] RICHARD K. SEVERSON,[1,2] EDWIN S. BOATMAN[2] AND DAVID B. THOMAS[1,2]

Polissar, L. (Fred Hutchinson Cancer Research Center, Seattle, WA 98104), R. K. Severson, E. S. Boatman and D. B. Thomas. Cancer incidence in relation to asbestos in drinking water in the Puget Sound region. *Am J Epidemiol* 1982;116:314-28.

Population-based and proportional odds ratios for various cancers, based on incidence data from 1974–1977 and mortality data from 1955–1975 for western Washington State, were calculated in relation to three measures of exposure to asbestos in community water supplies. Six odds ratios were calculated for each neoplasm that occurred in sufficient numbers in each sex. About half of the 332 odds ratios calculated were above unity and half were below unity, and no more of them differed significantly from unity at the 5% level than would be expected by chance. Odds ratios for tumors of the small intestine were consistently elevated in both sexes, as were those for neoplasms of the thyroid, eye, testis, and prostate in males; however, odds ratios for brain tumors and leukemia were consistently less than one in both sexes. Chance is the most likely explanation for these findings. Results of this study and prior studies of cancer in relation to waterborne asbestos are inconsistent, and provide little evidence that asbestos in community water supplies has altered the risk of any cancer. However, all investigations conducted to date are correlational studies which have an inherently high probability of failing to detect actual increased risks associated with imbibed asbestos, and additional studies of individual exposures are warranted. Neoplasms of the pancreas and small intestine should be included in such studies.

asbestos; environmental exposure; environmental pollutants; neoplasms; water pollutants; water supply

Only within the last 11 years have investigators begun to check thoroughly for the presence of asbestos fibers in drinking water. In 1971, Cunningham and Pontefract (1) found asbestos fibers in tap water samples from eight different areas of Canada, including Ottawa, Toronto and Montreal. In 1973, Duluth, Minnesota, public water supplies derived from Lake Superior were found to be contaminated with amphibole asbestos fibers (2). Since then, asbestos has been found in a number of other public water supplies (3).

Despite the known carcinogenic effect of inhaled asbestos, few studies have examined the health effects of waterborne asbestos. In 1974, Mason et al. (4) compared the 1950–1969 mortality rates of 21 neoplasms for Duluth to rates for Hennepin County (including Minneapolis) and to rates for all of Minnesota. Rectal cancer showed the strongest and most

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consistent relationship to asbestos exposure from water; lesser associations were observed for stomach cancer and female pancreatic cancer. Levy et al. (5) compared Duluth's gastrointestinal cancer incidence rates for 1969–1971 with those for Minneapolis and St. Paul combined, and did not confirm the previously observed excess risk of rectal cancer in Duluth. However, an excess of stomach cancer in males, and of pancreatic cancer in both sexes, was observed in Duluth.

Wigle (6) grouped 22 municipalities in Quebec into three classes based on asbestos concentrations in their water supplies: known high, possible high, and probable low concentrations. The observed-to-expected mortality ratios for 1965–1967 and 1970–1972 were then calculated for each exposure group. The communities with known high concentrations had been using asbestos-bearing water since about 1880, which suggested that enough time had passed to show whether any strong associations existed. Statistically significant increased rates were observed for male stomach cancer and lung cancer, although these observations may have been due to occupational exposure to asbestos. An excess of female pancreatic cancer rates also was observed, which is consistent with results for Duluth, but there was no increase in male pancreatic cancer rates.

Harrington et al. (7) computed the incidence rates of cancers of the colon, rectum, and stomach in Connecticut towns in which water is delivered through asbestos-cement pipes and compared them to incidence rates in towns with other types of pipes. No consistent excess rates in the former type of communities were observed. More refined analyses, based on multiple indices of asbestos exposure in the same towns, and on rates for additional types of cancer (8), also showed no consistent associations. However, somewhat consistent with observations from Duluth and Quebec, of the many possible associations considered, more positive associations were found for pancreatic cancer in males than for cancers of any other site.

Conforti et al. (9) related 1969–1974 cancer incidence rates in census tracts of the San Francisco-Oakland Bay Area, California, to chrysotile asbestos concentrations in municipal water supplies. Statistically significant associations, or results that showed the same trends in both sexes, were found for cancers of the stomach, esophagus, and pancreas. An association also was found for neoplasms of the pleura and peritoneum in women but not in men.

Although the studies conducted to date have yielded inconsistent results, in the aggregate the findings are sufficiently disconcerting to be cause for concern. In each of the four areas in which studies have been conducted (Minnesota, Quebec, Connecticut, and California), some evidence has been published suggesting an association between asbestos in water and pancreatic cancer in at least one of the sexes; and from all of the areas except Connecticut, there are similar findings for stomach cancer, which is consistent with the results of some studies of industrial exposure to airborne asbestos (10). Further investigation obviously is warranted.

The Puget Sound region of western Washington presents an excellent setting in which to continue this effort. Its three largest metropolitan areas generally have used the same river sources of water since

TABLE 1

Study areas with high and low concentrations of asbestos in drinking water, Puget Sound region, Washington

Asbestos concentration	Source of water	Major cities	Counties
High	Sultan River	Everett	Snohomish
Low	Cedar River, Tolt River, Green River, Lakewood Wells	Seattle, Tacoma	King, Pierce

TABLE 2

Cancer incidence and mortality codes grouped for analysis

Site	ICD-O Incidence Codes*	ICD-8 Mortality Codes†
Buccal cavity and pharynx	140.0–149.9	140.0–149.9
Esophagus	150.0–150.9	150.0–150.9
Stomach	151.0–151.9	151.0–151.9
Small intestine	152.0–152.9	152.0–152.9, 197.4
Colon	153.0–153.9	153.0–153.9
Rectum	154.0–154.9	154.0–154.9
Liver	155.0–155.9	155.0, 197.7–197.8
Gallbladder	156.0–156.9	155.1, 156.0–156.9
Pancreas	157.0–157.9	157.0–157.9
Retroperitoneum	158.0–158.9	158.0–158.9, 197.6
Larynx	161.0–161.9	161.0–161.9
Respiratory system	160.0–160.9, 162.0–163.9, 165.0–165.9	160.0–160.9, 162.0–163.9, 197.0–197.3
Bones and joints	170.0–170.9	170.0–170.9, 198.5
Soft tissue	164.1, 171.0–171.9	171.0–171.9
Melanoma	173.0–173.9 (Type = 8720–8789)	172.0–172.9
Breast	174.0–175.9	174.0–174.9
Cervix	180.0–180.9	180.0–180.9
Corpus uteri	182.0–182.9	182.0
Uterus, NOS‡	179.0–179.9	182.9
Ovary	183.0	183.0
Female genital, residual	181.0–181.9, 183.1–184.9	181.0–181.9, 183.1–184.9
Prostate	185.0–185.9	185.0–185.9
Testis	186.0–186.9	186.0–186.9
Male genital, residual	187.1–187.9	187.0–187.9
Bladder	188.0–188.9, 189.1–189.9	188.0–188.9, 189.1–189.9, 198.0–198.1
Kidney	189.0	189.0
Eye and orbit	190.0–190.9	190.0–190.9
Brain, CNS§	191.0–192.9	191.0–192.9, 198.3, 198.4
Thyroid	193.0–193.9	193.0–193.9
Hodgkin's disease	Type = 9650–9669	201.0–201.9
Non-Hodgkin's lymphoma	Type = 9590–9649, 9690–9709, 9750–9759	196.0–196.9, 200.0–200.9, 202.0–202.9
Multiple myeloma	Type = 9730–9731	203.0–203.9
Leukemia	Type = 9800–9949	204.0–207.9

Hodgkin's disease	Type = 9650–9669	201.0–201.9
Non-Hodgkin's lymphoma	Type = 9590–9649, 9690–9709, 9750–9759	196.0–196.9, 200.0–200.9, 202.0–202.9
Multiple myeloma	Type = 9730–9731	203.0–203.9
Leukemia	Type = 9800–9949	204.0–207.9
Other sites	159.0–159.9, 164.0, 164.2–164.9, 194.0–194.9, 173.0–173.9 (excluding Type = 8720–8789), Type = 9710–9722, 9950–9970, 195.0–195.9, 199.9, 169.0–169.9 (excluding types in specific sites above), 196.0–196.9 (excluding types in specific sites above)	159.0–159.9, 173.0–173.9, 194.0–195.9, 197.9, 198.2, 198.9–199.9, 208.0–209.9
All sites	140.0–199.9	140.0–209.9

* ICD-O, International Classification of Diseases for Oncology (11).
† ICD-8, International Classification of Diseases, Eighth Revision (11).
‡ NOS, not otherwise specified.
§ CNS, central nervous system.

the early part of the century, and each receives water from a different river system. There are large variations in levels of asbestos among waters of the three systems. The asbestos derives from geologic features in each river watershed, and the concentrations have probably changed little over the past 60 years. This paper presents results of a correlational study of the relationship of variations in cancer incidence and mortality rates in the Puget Sound region to temporal and spacial variations in exposure to waterborne asbestos.

Materials and methods

Study population. Cancer incidence and mortality risk ratios were calculated for the population in the Seattle-Everett-Tacoma metropolitan area served by several water sources with varying levels of asbestos. Only those 1960 and 1970 census tracts having the Sultan River, Cedar River, Tolt River, Green River or Lakewood Wells as the source of their drinking water were included in the analyses. The specific census tract numbers are shown in the Appendix.

The census tracts were grouped by level of asbestos concentration, as shown in table 1. The two geographic areas indicated in table 1, the Sultan River, which has high asbestos concentration, and the other water sources, which have low asbestos concentrations, are referred to as "Sultan" and "Other" in succeeding tables.

Cancer incidence. Data on incident cancers occurring between 1974 and 1977 were obtained from the Cancer Surveillance System, a population-based cancer registry that covers the study area. All cancer sites were coded using the International Classification of Diseases for Oncology classification scheme (11) (table 2).

Cancer Mortality. We obtained death certificate information for all residents of the study area who died of cancer as a primary cause of death between 1955 and

1975 and who were ages 35–79 years at the time of death. Causes of death had been coded to the Sixth, Seventh and Eighth Revisions of the International Classification of Diseases (12) by the Washington State Bureau of Vital Statistics. For each revision, the mortality codes were grouped in the same categories as for cancer incidence. As an example, the groups for the Eighth Revision are shown in table 2.

Asbestos concentration. We obtained 95 grab samples of tap water from the study area. Samples were identified only by number, and the source of the water remained unidentified until all water analyses were completed.

The method used for identification and quantification of asbestos fibers was in accordance with the procedure of Chatfield et al. (13). The procedure involved the filtration of water samples through 47 mm diameter, 0.1 μm pore size Nuclepore® membrane filters (Nuclepore Co., Pleasanton, CA). The filters were carbon-coated and portions were placed on copper electron microscopy grids. The filter matrix was dissolved by use of the Jaffe wick procedure using chloroform as a solvent.

Examination of electron microscopy grids was conducted by use of a JEOL® 100S transmission electron microscope (Japanese Electron Optics, Ltd., Medford, MA) operating at 100 kV at a magnification of 21,000X. The identification of asbestos fibers was based on size, shape, and appearance, and on crystal structure defined by selected area electron diffraction. Control water samples consisting of membrane-filtered distilled water were examined at appropriate intervals, along with a UICC (Union Internationale Contre le Cancer, Canada) standard chrysotile preparation.

A number of characteristics were reported for each water sample, including chrysotile and amphibole fiber concentration and fiber size.

Census variables. The 1960 and 1970 US Censuses of Population and Housing (14, 15) provided census tract-level data that included population by age and sex, per cent high school graduates, median family income, per cent married, per cent working in construction and manufacturing (where asbestos exposure is likely), and per cent with income at least three times above the poverty level.

In analyzing the 1955–1975 mortality data, we assumed that the 1960 population counts and characteristics would be close to average figures for 1955–1964 and that the 1970 data would be close to average for 1965–1975. In analyzing the 1974–1977 incidence data, we estimated midperiod population counts but assumed that other census tract characteristics, such as socioeconomic status, were similar to those recorded in the 1970 census.

The average annual population at risk used in the various analyses is shown in tables 3 and 4.

Duration of exposure. In some analyses of data only from the high exposure Sultan River area, we calculated an estimated duration of exposure by two methods. First, using annually published city directories (e.g., 16), which are available from the end of the 19th century, we checked the length of residence in the Sultan area city of Everett during the previous 40 years for cancer cases diagnosed among Everett residents in 1974–1977. Over 90 per cent of the cases were listed in one or more of the directories. We checked the directories at five-year intervals preceding diagnosis and divided the cases into long-term and short-term exposure groups: $\geq$30 vs. <30 years appearance in the directories.

Second, for each of the water district areas receiving the Sultan River water, we obtained information on the year water distribution from the Sultan River began and imputed an indirect duration of exposure based on these data. We used exposure groups of $\geq$30 vs. <30 years of Sultan River water supply.

TABLE 3

Number of cancers and population at risk for cancer from waterborne asbestos used for analyses in table 7, males, Puget Sound region, Washington, 1955–1977

Site	Sultan vs. all others				Sultan older vs. newer water districts: Incidence‡ (1974–1977)		Sultan long-term vs. short-term Everett residence: Incidence§ (1974–1977)	
	Incidence* (1974–1977)		Mortality† (1955–1975)					
	Sultan	Other	Sultan	Other	Older districts	Newer districts	Long-term	Short-term
Buccal cavity and pharynx	42	399	33	472	30	14	9	7
Esophagus	7	105	23	326	5	4	1	0
Stomach	32	254	68	910	24	11	8	4
Small intestine	2	16	6	33	2	0	2	0
Colon	87	792	90	1068	58	44	28	9
Rectum	55	445	37	473	40	18	9	8
Liver	5	64	16	269	5	0	0	1
Gallbladder	4	43	10	106	3	1	0	0
Pancreas	27	237	50	802	19	13	7	2
Retroperitoneum	0	8	2	31	0	0	0	0
Larynx	20	188	9	193	16	5	6	2
Respiratory system	212	1792	294	4273	152	84	50	40
Bones and joints	2	21	7	49	2	0	0	0
Soft tissue	10	50	2	67	9	2	2	1
Melanoma	24	180	9	109	17	8	1	5
Prostate	202	1566	69	801	147	70	58	22
Testis	19	105	4	31	16	5	2	1
Male genital, residual	8	31	1	16	8	0	1	1
Bladder	66	667	37	437	48	22	10	6
Kidney	18	171	29	318	12	8	3	3
Eye and orbit	4	10	2	18	1	4	1	0
Brain, CNS¶	16	164	26	443	11	10	1	3
Thyroid	7	51	4	25	4	3	1	0
Hodgkin's disease	14	82	10	176	7	6	1	0
Non-Hodgkin's lymphoma	28	234	31	390	21	8	2	3
Multiple myeloma	25	93	11	181	18	7	3	4
Leukemia	20	253	37	493	12	9	4	5
All sites	1003	8420	950	13030	723	374	220	133
Average annual population at risk	77,462	556,077	22,487	221,844	56,157	35,717	¶	¶

* Table 7, cols. 1 and 3.
† Table 7, cols. 2 and 4.
‡ Table 7, col. 5.
§ Table 7, col. 6.
¶ CNS, central nervous system.
¶ Analysis is not population-based.

Statistical analysis. We calculated six different pooled odds ratios for each sex and site combination, using a case-control approach and the method of Mantel and Haenszel (17) for pooling. The six odds ratios differed in the type of data used (incidence or mortality), the definition of case and control groups, and the definition of exposure. These definitions are presented in table 5. For each odds ratio a specific cancer, such as stomach, was used as the case group. For three of the six pooled odds ratios, all persons in the general population constituted the control

TABLE 4

Number of cancers and population at risk for cancer from waterborne asbestos used for analyses in table 7, females, Puget Sound region, Washington, 1955–1977

Site	Sultan vs. all others				Sultan older vs. newer water districts: Incidence‡ (1974–1977)		Sultan long-term vs. short-term Everett residence: Incidence§ (1974–1977)	
	Incidence* (1974–1977)		Mortality† (1955–1975)					
	Sultan	Other	Sultan	Other	Older districts	Newer districts	Long-term	Short-term
Buccal cavity and pharynx	27	219	9	199	19	10	4	3
Esophagus	6	45	6	115	6	0	2	1
Stomach	11	169	30	464	8	8	5	2
Small intestine	2	13	4	27	2	0	1	0
Colon	101	948	94	1138	72	41	24	18
Rectum	33	399	30	281	29	4	8	6
Liver	3	32	13	190	3	0	0	0
Gallbladder	5	65	14	170	4	2	0	1
Pancreas	30	206	39	523	25	8	7	4
Retroperitoneum	2	18	3	41	1	1	0	0
Larynx	7	40	2	41	4	4	2	1
Respiratory system	82	734	53	926	63	31	17	14
Bones and joints	1	17	3	40	1	0	0	0
Soft tissue	7	60	2	46	6	1	2	0
Melanoma	26	194	3	82	22	7	0	3
Breast	245	2631	198	2508	179	91	56	31
Cervix	281	1658	48	647	207	120	7	20
Corpus	125	1318	21	259	92	44	23	24
Uterus, NOS¶	2	10	12	160	1	2	1	0
Ovary	43	386	64	913	33	16	10	12
Female genital, residual	14	166	11	61	7	9	1	4
Bladder	22	255	14	142	16	9	3	2
Kidney	6	84	16	153	3	4	2	4
Eye and orbit	6	16	1	19	4	2	2	0
Brain, CNS¶	13	140	17	295	8	7	1	3
Thyroid	8	118	3	41	7	5	0	0
Hodgkin's lymphoma	10	62	3	110	8	2	0	0
Non-Hodgkin's lymphoma	24	227	27	324	19	8	3	4
Multiple myeloma	4	110	23	142	4	0	2	1
Leukemia	14	201	24	341	10	7	1	0
All sites	1201	10910	818	10811	915	469	194	171
Average annual population at risk	78,637	588,791	22,785	236,363	57,338	34,912	‖	‖

* Table 7, cols. 1 and 3.
† Table 7, cols. 2 and 4.
‡ Table 7, col. 5.
§ Table 7, col. 6.
¶ NOS, not otherwise specified; CNS, central nervous system.
‖ Analysis is not population-based.

group (population-based odds ratios), and, for the other three odds ratios, all other cancers besides the designated site were the control group (proportional-based odds ratios).

The population-based and proportional-based odds ratios are calculated in exactly the same manner once the stratified 2 × 2 tables (case or control vs. dichotomous exposure) are set up.

TABLE 5

Definitions for odds ratio calculations used in table 7

Type of Data	Definition of controls*	Definition of exposure groups		Column in table 7
		High	Low	
Incidence	General population	Sultan River area	All other areas	(1)
Mortality	General population	Sultan River area	All other areas	(2)
Incidence	Other cancer sites	Sultan River area	All other areas	(3)
Mortality	Other cancer sites	Sultan River area	All other areas	(4)
Incidence	General population	Sultan River area, older water districts	Sultan River area, newer water districts	(5)
Incidence	Other cancer sites	Sultan River area, long-term Everett residents	Sultan River area, short-term Everett residents	(6)

* Case-specific cancer site.

The population-based odds ratios have the advantage of describing the cancer risk in the entire population studied, but also have the disadvantage of being subject to estimation errors in the size of the population at risk. Our study includes estimated intercensal populations, and errors in these estimates can bias the odds ratios.

Proportional-based odds ratios are free of this kind of bias because they represent risk for a given cancer relative to the risk for all other cancers combined and do not require population estimates. The proportional-based odds ratios express a concept similar to standardized proportional mortality ratios. The two statistics involve different but roughly parallel calculations. A disadvantage of the proportional-based odds ratio is that cancer for many sites or for all sites combined may be linked to the exposure of interest, and, if so, the odds ratios for one site relative to other sites will be lower than the corresponding population-based odds ratio.

In the present study, potential errors in the population estimation may bias the population-based odds ratios while a multi-site effect of asbestos may bias the proportional-based odds ratios. There is some evidence that our population estimates are in error since the population-based odds ratios for most cancers tend to be either generally higher or generally lower than the corresponding proportional-based odds ratios, though the differences are small. Due to the systematic difference, it seems appropriate to present both types of odds ratios.

All cases and controls were stratified into a series of 2 × 2 tables (case-control status vs. dichotomous exposure). Each stratum was defined by a combination of specific levels of age and potentially confounding variables. The stratifying variables we used were age (four to five levels), and, as other variables which were census tract averages: per cent high

TABLE 6

Asbestos concentration and length of asbestos fibers in drinking water, Puget Sound region, Washington, 1978–1979

Tap water source	No. of samples	Chrysotile Concentration (10^6 fibers/liter)					Fiber length	
		Mean	Standard deviation	Median	Minimum	Maximum	% < 1μm	% < 5 μm
Sultan River	22	206.5	162.2	142.8	37.2	556.0	85.8	99.9
Other areas	73	7.3	12.4	2.0	0.0	77.6	82.5	99.4

TABLE 7

Mantel-Haenszel pooled odds ratios for cancer incidence risk (1974–1977) and cancer mortality risk (1955–1975) by sex and site, Puget Sound region, Washington

Site	Males						Females					
	Sultan vs. all others				Sultan high vs. Sultan low		Sultan vs. all others				Sultan high vs. Sultan low	
	Population-based incidence	Population-based mortality	Proportional incidence	Proportional mortality	Based on age of water district	Based on length of residence	Population-based incidence	Population-based mortality	Proportional incidence	Proportional mortality	Based on age of water district	Based on length of residence
	(1)	(2)	(3)	(4)	(5)	(6)	(1)	(2)	(3)	(4)	(5)	(6)
Buccal cavity and pharynx	0.92	0.73	0.85	0.95	1.23	1.06	1.22	0.47*	1.25	0.57	0.73	1.00
Esophagus	0.62	0.71	0.60	0.90	0.93	–†	1.56	0.61	1.69	0.73	–†	2.00
Stomach	1.15	0.73*	1.03	0.99	1.41	1.50	0.65	0.64*	0.66	0.77	0.35	1.93
Small intestine	1.31	1.89	1.51	2.42	–†	–†	1.55	1.49	1.65	1.89	–†	–†
Colon	1.08	0.85	0.96	1.23	0.78	1.84	1.05	0.87	1.08	1.13	1.03	0.90
Rectum	1.17	0.85	1.06	1.15	1.30	0.54	0.84	1.17	0.88	1.42	2.67	1.20
Liver	0.82	0.65	0.71	0.85	–†	0	1.03	0.75	0.95	0.95	–†	–†
Gallbladder	0.84	1.12	0.66	1.66	2.76	–‡	0.54	0.85	0.58	1.14	1.38	0
Pancreas	1.00	0.64§	0.95	0.90	1.22	2.22	1.33	0.79	1.42	1.00	1.23	1.53
Retroperitoneum	0	1.23	0	1.44	–‡	–‡	0.77	0.86	0.72	1.08	1.30	–‡
Larynx	0.96	0.46*	0.92	0.56	2.31	2.15	1.40	0.66	1.48	0.80	0.71	2.06
Respiratory system	1.03	0.67§	0.97	0.88	1.14	0.92	1.09	0.57§	1.16	0.76	1.36	1.23
Bones and joints	0.78	1.49	0.74	2.29	–†	–‡	0.60	0.61	0.55	0.91	–†	–‡
Soft tissue	2.41*	0.37	2.16	0.46	2.46	2.09	1.18	0.49	1.15	0.66	0.90	–†
Melanoma	1.30	0.70	1.18	1.20	1.33	0.25	1.22	0.37	1.20	0.49	1.59	0
Breast	–	–	–	–	–	–	0.88*	0.83*	0.88	1.12	1.20	1.77
Cervix	–	–	–	–	–	–	1.13	0.74	1.08	0.84	1.09	0.38
Corpus	–	–	–	–	–	–	0.96	0.91	1.00	1.05	1.34	0.89
Uterus, NOS	–	–	–	–	–	–	2.03	0.72	1.97	0.98	0.90	0
Ovary	–	–	–	–	–	–	1.11	0.74*	1.16	0.93	1.21	0.76

Breast	—	—	—	—	—	—	0.88*	0.83*	0.88	1.12	1.20	1.77			
Cervix	—	—	—	—	—	—	1.13	0.74	1.08	0.84	1.09	0.38			
Corpus	—	—	—	—	—	—	0.96	0.91	1.00	1.05	1.34	0.89			
Uterus, NOS	—	—	—	—	—	—	2.03	0.72	1.97	0.98	0.90	0			
Ovary	—	—	—	—	—	—	1.11	0.74*	1.16	0.93	1.21	0.76			
Female genital, residual	—	—	—	—	—	—	0.78	1.99	0.78	2.39*	0.42*	0.30			
Prostate	1.31*	0.85	1.22*	1.14	1.36	1.32	—	—	—	—	—	—			
Testis	1.38	1.23	1.27	1.88	2.44	2.30	—	—	—	—	—	—			
Male genital, residual	2.51	0.47	2.22	0.59	—†	0.97	—	—	—	—	—	—			
Bladder	0.90	0.82	0.80	1.10	1.57	0.50	0.89	1.15	0.89	1.40	0.59	1.19			
Kidney	0.97	0.86	0.93	1.21	0.91	0.63	0.61	0.89	0.60	1.16	0.27	0.42			
Eye and orbit	3.08	1.53	2.83	2.17	0.00	—†	3.58*	0.52	3.59*	0.47	0.61	—†			
Brain, CNS	0.84	0.62*	0.76	0.97	0.56	0.22	0.80	0.61	0.79	0.76	0.83	0.36			
Thyroid	1.34	1.58	1.18	2.68	1.19	—†	0.54*	0.66	0.61	0.79	0.79	—‡			
Hodgkin's disease	1.15	0.49*	1.05	0.71	1.13	—†	1.33	0.27*	1.23	0.36	2.34	—‡			
Non-Hodgkin's lymphoma	1.07	0.77	0.98	1.13	1.97	0.27	1.07	0.94	1.10	1.18	1.43	0.70			
Multiple myeloma	2.06*	0.53	1.95§	0.75	1.01	0.62	0.33**	1.63	0.35*	2.20**	—†	1.42			
Leukemia	0.60*	0.70*	0.58*	1.03	0.89	0.45	0.61*	0.70	0.59	0.84	0.93	—†			
All sites	1.10*	0.71§	—*	—*	1.23	—*	0.99	0.78§	—*	—*	1.16	—*			

* $p < 0.05$.
§ $p < 0.01$.
‡ Numerator = denominator = 0 in pooled odds ratio.
† Numerator ≠ 0, denominator = 0, in pooled odds ratio. Evidence for an effect.
* *Proportional analyses do not permit odds ratio calculations for all sites.*

school graduates as a socioeconomic indicator (two levels), per cent of the population in industries possibly associated with asbestos exposure (two levels), median family income (two levels), and per cent married (two levels). The mortality data covered 22 years, hence we also stratified these cases and controls by calendar year (two to four levels). For pooled odds ratios based on length of residence in the Sultan area city of Everett, we used the per cent of census tract population with income at least three times above poverty level as a stratifying variable in place of the median family income. Also, due to the smaller number of cases available for this analysis, the per cent married was not used for stratification.

We did not use census tract population density in the analysis. Only urban areas were included in the study, hence population density of tracts would not be an indicator of urban vs. rural lifestyle or exposure.

Results

Asbestos concentration. Table 6 shows some characteristics of the tap water from the Sultan River and other areas. Only one amphibole fiber was found in all of the water samples, hence exposure was characterized by chrysotile fiber concentrations alone. Mean chrysotile fiber concentration in the Sultan River area differs from that in the other areas by a factor of more than 25.

We tested to see if the asbestos exposure varied geographically within the Sultan River distribution area. We found that there was no significant clustering of exposures, which allowed us to treat the population in the Sultan River area as receiving a uniform exposure to asbestos.

Cancer risk. A summary of numbers of cases and populations at risk is shown in tables 3 and 4 for males and females, respectively. Table 7 shows the pooled odds ratios of cancer risk by sex and site. For each cancer site, the population-based

and proportional incidence ratios are based on the same cases, as are the population-based and proportional mortality ratios. Therefore, the odds ratios in columns one and three, and those in columns two and four, of table 7 are not totally independent. For most cancer sites, the odds ratios in the methodologically related columns have about the same magnitude, which provides some grounds for confidence in the validity of the results.

Since a large number of comparisons are being made, it is likely that some of the results are significant by chance alone. Altogether, 332 odds ratios are presented, not counting those for "all sites" and for sites where the pooled odds ratio was indeterminate due to insufficient cases or controls. The 332 odds ratios are about evenly split above and below unity, suggesting a chance mechanism for most of the results. Among 332 independent odds ratios, 16.6 would be expected to differ significantly from unity at the conventional 5 per cent significance level. Thirty of the 332 odds ratios actually differed from unity at that level, which is not an excessive number because the odds ratios based on incidence and proportional incidence, and those based on mortality and proportional mortality, are not independent (same numerators). Furthermore, only six of the 30 "significant" odds ratios were greater than one, and there is no known biologic reason to expect that the 24 that were less than unity represent a protective effect of asbestos.

Consistent results among the various analyses and populations shown in table 7 may be more likely to represent real alterations in risk than single statistically significant odds ratios. There appears to be a consistent, or nearly consistent, positive association with asbestos exposure in all analyses for neoplasms of the small intestine in both males and females. On the other hand, cancers of the brain and central nervous system and leukemia show a reduced risk in both sexes in the high-

TABLE 8

Mantel-Haenszel pooled incidence odds ratios for exposure to Sultan River drinking water vs. all other sources of water for census tracts with lower migration rates, Puget Sound region, Washington, 1974–1977

Site	Males		Females	
	Total cases	Odds ratio	Total cases	Odds ratio
Buccal cavity and pharynx	196	0.92	111	1.25
Esophagus	53	0.26	23	0.70
Stomach	128	0.96	82	0.35
Small intestine	4	0	5	0
Colon	397	1.17	442	1.13
Rectum	218	0.97	198	1.08
Liver	30	0.42	18	0
Gallbladder	20	1.93	23	0
Pancreas	118	0.97	101	1.25
Retroperitoneum	2	0	5	0
Larynx	89	0.63	19	1.50
Respiratory system	905	1.10	334	1.17
Bones and joints	16	0.86	5	5.89
Soft tissue	34	3.41*	35	1.51
Melanoma	91	1.06	90	1.05
Breast	—	—	1316	0.91
Cervix	—	—	834	1.04
Corpus	—	—	720	0.94
Uterus, NOS	—	—	6	5.17
Ovary	—	—	191	1.41
Female genital, residual	—	—	75	0.75
Prostate	823	1.43*	—	—
Testis	51	1.16	—	—
Male genital, residual	15	4.53*	—	—
Bladder	332	0.90	130	1.11
Kidney	97	0.96	47	0.81
Eye and orbit	8	0	11	7.75*
Brain, CNS	79	0.91	67	0.37
Thyroid	22	3.70	49	0.28
Hodgkin's lymphoma	31	1.78	35	1.17
Non-Hodgkin's lymphoma	131	0.80	107	0.45
Multiple myeloma	55	1.55	55	0.50
Leukemia	141	0.87	99	0.65
All sites	4290	1.14*	5477	0.99

* $p < 0.05$.
† $p < 0.01$.

exposure area. In men, odds ratios tend to be consistently elevated for cancers of the prostate, testis, eye and orbit, and thyroid. Although consistent in direction, most of the odds ratios for these sites are not statistically significant.

To determine the possible effect of migration on our results, we calculated population-based odds ratios for cancer incidence using the census tracts with lower migration rates as recorded in the 1970 census. This analysis, which included about half the cases in the Sultan River distribution area, used tracts with at least 73 per cent of the population over

TABLE 9

*Summary of studies of cancer risk in relation to asbestos in water supply by site of neoplasm**

Area	First author (reference no.)	Site of neoplasm						
		Colon	Rectum	Stomach	Pancreas	Lung	Peritoneum	Esophagus
Duluth	Mason (4)	00	MF	MF	0F	–	–	00
	Levy (5)	00	00	M0	MF	–	00	00
Quebec	Wigle (6)	00	00	M0	0F	M0	–	–
Connecticut	Harrington (7)	00	00	00	–	–	–	–
	Meigs (8)	00	00	00	B	00	–	–
California	Conforti (9)	00	00	MF	MF	00	0F	MF
Present study		00	00	00	00	00	–	00

Area	First author (reference no.)	Site of neoplasm								
		Gallbladder	Pleura	Small intestine	Brain	Leukemia	Thyroid	Eye	Testis	Prostate
Duluth	Mason (4)	00	–	–	–	–	–	–		–
	Levy (5)	00	–	00	–	–	–	–		–
Quebec	Wigle (6)	–	–	–	00	00	–	–		M
Connecticut	Harrington (7)	–	–	–	–	–	–	–		–
	Meigs (8)	–	–	–	–	–	–	–		–
California	Conforti (9)	00	0F	00	00	00	00	–		00
Present study		00	–	MF	MF†	MF†	M0	M0		M

* M, association in males; F, association in females; B, association in both sexes combined; 0, no association; –, not studied.

† Inverse association with asbestos levels.

age five years living in the same house both in 1965 and 1970. Thus, a subset of the incident cases and population used to calculate the odds ratios in column one of table 7 was used. Results are shown in table 8. There were no cases of small intestine cancers in the low-migration census tracts with high levels of asbestos, so the odds ratios were reduced to zero. This also occurred for cancer of the eye and orbit in males. Odds ratios for leukemia, and for cancers of the prostate and male thyroid, were not altered appreciably. Of the 59 sex-specific odds ratios in table 8, 27 were larger than those in column one of table 7, and 29.5 would have been expected to be so due to chance. Except to help rule out associations between asbestos exposure and neoplasms of the small intestine, and of the eye and orbit, the estimated odds ratios in low-migration tracts do not materially alter the conclusions based on all tracts.

DISCUSSION

Table 9 presents a summary of results from this and previously published studies (4–9). Our interpretations of the data from these studies are somewhat subjective and are not always the authors'.

Of the seven types of neoplasms possibly related to asbestos in drinking water in this study, the associations were most consistently observed for cancers of the small bowel—the only neoplasms among the seven types that arise from tissue that comes in direct contact with imbibed asbestos. However, there are three reasons to doubt that the observed relationship is causal: 1) the increased risk was not confined to a single histologic type of small bowel tumor; 2) the association was not observed when analyses were confined to census tracts with relatively low migration rates; and 3) this relationship was

not found in the two previous studies (5, 9) that examined this site. Nevertheless, due to the consistency of our findings, additional studies of the relationship of neoplasms of the small bowel to asbestos exposure may be warranted.

Risks of cancers of the brain and central nervous system, and of leukemia, were inversely related to levels of asbestos in water. These probably represent chance observations. There is no known reason to expect asbestos to exert a protective effect against these neoplasms, and two previous studies (6, 9) found neither to be related to waterborne asbestos.

The observed increase in risks of tumors of the eye and thyroid also are most likely spurious findings. The odds ratios were elevated only in males; those for ocular tumors were not elevated when analyses were confined to census tracts with relatively low migration rates, and the observed association for thyroid tumors is not consistent with previous results from California (9).

Risk of testicular tumors has not previously been studied in relation to asbestos in water. Because the histologic types of testicular tumors did not differ in areas with relatively high and low water asbestos levels, this association also probably has no biologic significance.

An increased risk of prostate cancer was found in this study, as well as in Wigle (6), one of the two previous studies that included this site. The distribution of our cases by stage of disease at diagnosis was very similar in the high and low asbestos exposure areas, so the finding appears not to be an artifact due to a greater rate of urologic procedures (which tend to reveal small tumors) in the high exposure area.

As shown in table 9, one or more previous studies have shown some relationship to asbestos in water for neoplasms of the following sites: rectum (4), stomach (4–6, 9), pancreas (4–6, 8, 9), lung (6), peritoneum (9), esophagus (9) and pleura (9). Results of the present study confirm none of these findings (there were too few pleural and peritoneal tumors for analysis), and most of the findings in the various published studies probably represent chance phenomena resulting from large numbers of comparisons.

An exception to this might be pancreatic cancer. All of the five previous studies that included this neoplasm (4–6, 8, 9) showed some association between water asbestos levels and risk in one or both sexes. In the present study, results of the various analyses presented in tables 7 and 8 are not consistent for pancreatic cancer. Some odds ratios are above and some are below unity, and our results neither confirm nor refute prior findings. Because there are so few leads as to the cause of pancreatic cancer, this possible association with imbibed asbestos observed in five out of six studies that included the site should be the subject of a more intensive study.

In addition to chance, the discrepant results in table 9 may, in part, be due to differences in characteristics of asbestos exposure in the various study populations. These differences are summarized in table 10. The paucity of positive findings from Connecticut (7, 8) could be due both to the low concentration of asbestos in the water and the relatively short duration of exposure in some areas of the state (18). Short duration of exposure also could be an explanation for some of the nonassociations in Duluth (4, 5), as could asbestos type (amphibole), which is different from the type of asbestos in other study areas (chrysotile). In addition, the concentration of other possibly carcinogenic contaminants of water, as well as asbestos fiber length, may vary among the various studies cited.

Another limitation of all of the studies to date is that specific asbestos exposures are imputed to the population in an entire geographic region. There may be a confounding effect from other factors that

TABLE 10

Characteristics of asbestos in drinking water in various study populations

Study population (reference no.)	Type of asbestos	Range of no. of fibers/liter	Duration of community exposure (years)
Duluth (4,5)	Amphibole	$1-30 \times 10^6$	< 20
Quebec (6)	Chrysotile	$1.1-1300 \times 10^6$	> 50
Connecticut (7, 8, 17)	Chrysotile	BDL* -0.7×10^6	> 15
California (9)	Chrysotile	$0.02-36 \times 10^6$	> 50
Present study	Chrysotile	$7-207 \times 10^6$	>50

* BDL, below detectable limits.

vary geographically, and there may be misclassification of exposures due to recent in-migration into the study area (19).

Exposure to chrysotile asbestos is of long duration (>50 years) in the present study, and in Quebec (6) and California (9). These three study areas differ in that the concentrations of asbestos are lowest in California, intermediate in the present study area, and highest in Quebec. However, the associations summarized in table 9 do not appear to vary in accordance with the differences in asbestos concentration among the three study areas, which suggests that the observed associations do not have a biologic basis.

From the data we have analyzed, we conclude that the cancer risk from asbestos in drinking water in the Sultan River area is, at most, very low, although there is suggestive evidence for some anatomic sites.

All of the studies to date share an unknown but probably substantial misclassification of exposures, which causes a decrease in the power to detect excess risks. To remedy this shortcoming, we are currently conducting a case-control study of cancer risk in relation to imbibed asbestos in which exposures will be determined from in-person interviews. We anticipate that this technique will greatly reduce the amount of exposure misclassification and will allow a more accurate determination of cancer risk from waterborne asbestos.

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Appendix

Census tracts included in study of cancer and waterborne asbestos, by year and county, Puget Sound region, Washington

Year	County	Census tracts
1970	King	1–121, 201–228, 230–250, 260–276, 278–291, 298, 300, 323
	Snohomish	401–415, 417–420, 501–520*
	Pierce	601–635, 718.01–722, 723.01†, 723.02†, 735
1960	King	A1–5, B1–6, BU1–3, C1–4, D1–12, E1–4, F1A–2, G1–6, H1–3, I1–3, J1–3, K1–5, KC4, KC11, KC23, KC31–112, K11, L1–5, M1–5, N1–4, NP109, O1–4B, P1–3, Q1–3, R1A–5B, S1A–3, T1–23, TG41, U1–2
	Snohomish	EM1, EV1–12, LY1, MT1, SC113–119*
	Pierce	1–35, PC18–22, PC23†, PC35

* 1970 tracts 505–509 and 1960 tract SC11S received Tolt or Cedar River water (low asbestos concentration).

† Included in mortality odds ratios only.

Asbestos in local water is not dangerous, study finds

It means Seattleites "can relax and drink all they want."

That was the reaction of John Courchene, Seattle's director of water quality, to a study showing local water does not contain dangerous levels of asbestos as had been feared.

Investigators for the Fred Hutchinson Cancer Research Center have announced that a four-year study concluded "asbestos in drinking water has not led to an increased cancer incidence or death rate for people living in the Puget Sound region."

The study of the water was commissioned by the Environmental Protection Agency after naturally occurring asbestos was found in the Sultan River, which supplies water to Everett and other South Snohomish communities, and the Tolt River, which supplies a large part of Seattle's water.

Dr. David B. Thomas, an associate member of the Hutchinson Center and a professor of epidemiology at the University of Washington, said the study compared cancer rates in people served by the Sultan River, which has the highest asbestos content, with rates in others in the Puget Sound region who have fewer or no traces of asbestos in their drinking water.

"We didn't find any significant differences," he said.

Nearly 45,000 cancer cases in the Seattle-Tacoma-Everett area were studied during the research, headed by Lincoln Polissar, an assistant member of the Hutchinson Center and a research associate professor of biostatistics at the UW.

The study's results have little bearing on a $30 million water-filtration project under construction by Everett whose water comes from the Sultan River.

Clair Olivers, an Everett utility engineer supervising the filtration project, said it was turbidity — suspended particles of clay that promote bacterial growth in the water — not asbestos that created the need for filtering.

Although the study does not resolve the argument about how much asbestos is safe, Thomas said, the doses are sufficiently low not to create a measurable health hazard.

The study ends 7½ years of concern about asbestos in the water. Courchene said, "This is the best piece of good news we've had in a long time."

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