

39 - Tacoma studies

INVESTIGATING POSSIBLE EFFECTS OF ASBESTOS IN CITY WATER: SURVEILLANCE OF GASTROINTESTINAL CANCER INCIDENCE IN DULUTH, MINNESOTA

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Levy, B. S. (Minnesota Dept. of Health, Minneapolis, MN 55440), E. Sigurdson, J. Mandel, E. Laudon and J. Pearson. Investigating possible effects of asbestos in city water: Surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. *Am J Epidemiol* 103:352-368, 1976.

The recent discovery of over one million asbestos-like fibers per liter of Duluth tap water and the suggestive evidence of a link between certain gastrointestinal (GI) cancers and work exposure to asbestos fibers in the air prompted this study. GI cancer incidence data were gathered for Duluth in the same manner as data previously gathered for comparison cities, Minneapolis and St. Paul. Although some differences in GI cancer incidence occurred among the three cities in 1969-1971, there was no consistent pattern of statistically significant differences observed. The number of GI cancers diagnosed in Duluth residents in 1972 was similar to that in each of the previous three years. This study represents the start of ongoing cancer surveillance in Duluth.

asbestos; cancer; gastrointestinal neoplasms; water pollution

Human exposure to asbestos has increased greatly in recent years, yet the

hazards of this exposure to human health are not understood completely. Occupational exposure to certain concentrations of asbestos fibers in the air has been linked with pulmonary asbestosis, lung cancer, and pleural and peritoneal mesotheliomas (1-6). There are also data suggestive of a link between such exposure and development of cancers of the stomach, large intestine, and rectum many years after initial exposure (1, 6-9). This possible link, presumably the result of swallowing initially inhaled fibers, raises the possibility that drinking water with high concentrations of asbestos or asbestos-like fibers for sufficiently long periods may lead to an increased incidence of gastrointestinal (GI) cancers among those who drink it.

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Abbreviation: GI, gastrointestinal.
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In June 1973, the US Environmental Protection Agency (EPA) reported the recent discovery of large amounts of asbestos in Lake Superior, the source of municipal water for Duluth, Minnesota (1970 population: 100,578), and five smaller communities on the lake shore. Subsequent electron

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microscope studies revealed one to 30 million amphibole asbestiform fibers per liter of Duluth tap water. the concentration varying with lake conditions and the length of time water is in the distribution system (10). A federal court ruling indicates that these fibers result from a mining company dumping, since 1955, large quantities of taconite tailing wastes directly into the lake at its iron ore processing plant in Silver Bay, 50 miles (80 km) northeast of Duluth (11). These tailings contain minerals in the cummingtonite-grunerite series, which includes amphibole asbestos. EPA data on Duluth water in the past, although limited, are consistent with the court's ruling. They show that samples from 1939-1940 and 1949-1950 contain trace amounts of amphibole. but all samples studied from 1964-1965 contain large amounts of amphibole (an average of 31 per cent of total inorganic solids) (10). It is not known any more specifically, however, when amphibole in Duluth water increased beyond these amounts.

The discovery of these fibers in the lake and Duluth tap water prompted many environmental, pathological, and epidemiologic studies. The present study is the only recent one of cancer incidence in the entire populations of Duluth and comparison cities, and represents the start of ongoing cancer surveillance for Duluth. Further cancer incidence studies must be done in the future, as well as studies to quantitate ingestion and inhalation of asbestos by residents of Duluth and comparison cities.

METHODS OF INVESTIGATION

Information was obtained on each Duluth resident with a GI cancer first diagnosed from January 1969 through December 1972. A GI cancer was defined as a malignancy of the digestive tract or peritoneum with one of the following primary anatomic sites: esophagus; stomach; small intestine; large intestine; rectum and rectosigmoid; liver; gall bladder and biliary tract; pancreas; peritoneum, retroperito-

neum, and abdomen, not otherwise specified; and GI tract, not otherwise specified.

GI cancer incidence data for 1969-1971 for Duluth residents were compared with those for Minneapolis and St. Paul residents. These latter data had previously been obtained by the Division of Epidemiology, University of Minnesota School of Public Health, as part of the Third National Cancer Survey (TNCS) of the National Cancer Institute. Duluth incidence data for 1972, the most recent period for which data were available, were also obtained. These were compared only with Duluth incidence data for 1969-1971 since 1972 data for Minneapolis and St. Paul were not readily available.

Minneapolis (1970 population: 434,400) and St. Paul (1970 population: 309,950) were considered suitable comparison cities because they have large populations that are relatively similar to Duluth in age, race, ethnic groups, and occupational exposures (table 1), and because they have access to relatively comparable medical care. In addition, their water supplies, directly and indirectly from the Mississippi River, are thought to contain few, if any, asbestos or asbestos-like fibers. It is likely to be a relatively long time, however, before this fact is definitely established.

Since almost all known GI cancer patients are hospitalized at some time and since Duluth residents are hospitalized primarily in Duluth, data were obtained primarily from inpatient records at the three Duluth hospitals. Almost all of the few Duluth residents with cancer who seek medical care elsewhere visit the Mayo Clinic in Rochester, Minnesota, or hospitals in the Minneapolis-St. Paul metropolitan area. Data were therefore also obtained from inpatient records at the Mayo Clinic for the four Duluth residents with GI cancer diagnosed there from January 1969 through December 1972, and from TNCS records for Twin Cities area hospitals for the six Duluth residents whose cases were diagnosed there from January 1969 through

TABLE I
 Characteristics of populations of Duluth and
 comparison cities*

	Duluth	Minneapolis	St. Paul
Total population (1970 Census)	100,578	434,400	309,980
Median age			
Males	27.8	27.8	26.3
Females	31.5	30.7	29.7
Birthplace			
% born in Minnesota	69.9	67.1	72.6
% foreign-born	5.2	4.8	3.9
% foreign stock (foreign-born individuals - natives of foreign or mixed parentage)	21.9	23.9	21.8
Norwegian and Swedish	10.4	9.4	4A
German and Austrian	2.5	3.4	5.2
Finnish	2.8	0.5	0.2
% white	98.3	95.6	95.4
% who lived in same county in 1965 (as in 1970)	82.1	75.9	79.7
Median So. of school years completed (for population \geq 25 years old)	12.3	12.3	12.2
% of males \geq 16 years old in civilian labor force unemployed	6.0	4.2	3.6
% families below poverty level	7.1	7.2	6.4
Median family size	3.48	3.26	3.52
Median family income	\$9,313	\$9,960	\$10,544
% in selected occupations			
Professional, technical, and kindred workers	16.4	16.5	16.5
Craftsmen, foremen, and kindred workers	13.0	10.7	11.7
Laborers except farmers	4.8	4.2	4.3
% in selected industries			
Mining	0.6	0.1	0.1
Construction	4.8	4.2	4.7
Manufacturing	16.4	20.5	25.1

* Based on 1970 US Census data.

December 1971. TNCS data were limited to 1969-1971. Data were not obtained from Twin Cities area hospitals for Duluth residents diagnosed there in 1972. By extrapolation, however, we estimate that only two cases were excluded by not obtaining these data.

Medical record administrators at each of the three Duluth hospitals and the Mayo Clinic provided inpatient records for all Duluth residents discharged during the study period and shortly thereafter with a GI cancer listed among their discharge diagnoses. In order to assure comparability between Duluth and comparison cities

data, the same protocol used during the TNCS to collect incidence data for Minneapolis, St. Paul, and elsewhere was used in this study to collect data for Duluth; two of the authors (E. S. and E. L.) who abstracted data during the TNCS abstracted the data for Duluth. From the hospital records of each Duluth resident with a GI cancer first diagnosed from January 1, 1969, through December 31, 1972, they abstracted information that included: name of patient, address at time of diagnosis, sex, race, date and place of birth, and, for each GI cancer, date of first diagnosis, primary anatomic site and histologic type, and most accurate method of diagnosis. Addresses of all "Duluth residents" were checked with census tract guides and those who lived beyond Duluth city limits were excluded from the study.

The definition of cancer, as in the TNCS, was based on confirmatory evidence of a malignant neoplasm or, in the relatively few instances when that was not available, indication by physicians' notes in the patient's hospital record of a 50 per cent or greater probability of a malignancy. The definition of the date of first diagnosis, as in the TNCS, was the date when it was first determined that a patient had a new cancer, even though primary anatomic site and histologic type may not have been known at that time. It was the earliest date for one of the following diagnostic criteria: (a) inpatient hospital admission with a discharge diagnosis (not necessarily the admitting or main diagnosis) of cancer; (b) microscopic diagnosis of cancer on surgical biopsy or cytological specimen; (c) palliative or curative treatment directed toward a cancer; (d) autopsy diagnosis of cancer (with microscopic confirmation) without prior diagnosis of that cancer known; or (e) death certificate diagnosis of cancer judged to be valid, without other information available. For (d) and (e), date of diagnosis was the date of death.

Following procedures identical to those of the TNCS, three other sets of records

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were reviewed in attempts to find cases not identified from individual patient hospital records: pathology records and radiotherapy records at the two major Duluth hospitals, and death certificates for all Duluth residents who died in Minnesota between 1969 and 1973 and had GI cancers noted on their death certificates as underlying causes of death. (Such records were the primary source of information for only five Duluth cases.)

Usual occupation of patient, date of death, and birthplace of each parent were abstracted from death certificates of patients who died, and were added to information obtained from hospital records.

Incidence data for Minneapolis and St. Paul were abstracted from computer tapes of TNCS data for the Twin Cities metropolitan area. Residents of these cities were identified by zip code. Addresses of those living in zip code areas that are included in more than one city were pinpointed on official city maps to determine their cities of residence. In addition, a 5 per cent random sample of all patients' addresses was checked, also by pinpointing addresses on city maps, to confirm that zip codes were accurately recorded.

Data for Duluth residents, initially abstracted onto pre-coded forms, were punched onto computer cards in the same format as data for Minneapolis and St. Paul residents, that were abstracted from computer tapes. Tabulations and calculations were done by computer. Crude incidence rates were computed using study and 1970 census data. Age-and-sex-specific incidence rates for Duluth for each type of GI cancer were compared with those for Minneapolis and St. Paul using the formula to test the difference between two sample proportions:

$$d = \frac{P_1 - P_2}{\sqrt{P(1-P) \left(\frac{1}{n_1} + \frac{1}{n_2} \right)}}$$

Age- and age-and-sex-adjusted incidence

rates were computed using the 1970 Minnesota population as the standard.

RESULTS

The average annual age-and-sex-adjusted incidence rates for each GI cancer for Duluth, Minneapolis, and St. Paul for 1969-1971 are shown in table 2. For all sites

TABLE 2

Gastrointestinal cancer incidence rates, residents of Duluth and comparison cities, 1969-1971

Primary anatomic site	Sex	Average annual adjusted rates per 100,000 population ¹		
		Duluth	Minneapolis	St. Paul
Esophagus	Males	5.32	7.02	5.89
	Females	1.61	2.26	1.97
	Both	3.43	4.59	3.89
Stomach	Males	18.92	17.21	14.04
	Females	9.37	8.97	11.57
	Both	14.05	13.00	12.78
Small intestine	Males	2.25	2.18	1.30
	Females	1.01	1.29	0.68
	Both	1.62	1.73	0.98
Large intestine	Males	31.62	40.66	42.71
	Females	30.11	38.42	37.37
	Both	30.85	39.52	39.99
Rectum/rectosigmoid	Males	15.14	18.77	23.40
	Females	11.29	113.98	12.85
	Both	13.18	16.30	16.02
Liver	Males	2.58	3.20	2.52
	Females	0.51	0.99	2.10
	Both	1.53	2.07	2.35
Gall bladder/biliary tract	Males	2.32	3.17	4.41
	Females	5.25	3.91	5.29
	Both	3.81	3.55	4.86
Pancreas	Males	15.20	14.23	12.55
	Females	13.77	8.08	9.77
	Both	14.17	11.09	11.13
Peritoneum, retroperitoneum, and abdomen, not otherwise specified	Males	1.74	1.14	1.45
	Females	0.52	1.26	2.35
	Both	1.12	1.20	1.91
GI tract, not otherwise specified	Males	2.21	0.27	0.21
	Females	1.55	0.58	0.00
	Both	1.87	0.43	0.10
Total	Males	97.29	107.86	108.47
	Females	74.99	79.68	84.06
	Both	85.91	93.49	96.02

¹ Male and female rates were age-adjusted; combined rates for both sexes were both age- and sex-adjusted. Rates were adjusted using the 1970 Minnesota population as the standard.

combined, these rates were greater for Minneapolis and St. Paul. Cancers of the stomach, large intestine, rectum/rectosigmoid, and pancreas accounted for approximately 85 per cent of all GI cancers. For males and for females, Duluth incidence rates for cancer of the pancreas were higher than those of Minneapolis and St. Paul, and Duluth rates for cancer of the stomach were higher than those of Minneapolis. On the other hand, for males and for females, Duluth incidence rates for cancers of the large intestine and rectum/rectosigmoid were lower than those of Minneapolis and St. Paul. Statistically significant differences, based on tests which we felt were more appropriate to perform on crude incidence rates, are shown in table 3. (Since the age distributions of all three cities are similar, the crude incidence rates approximated the age-adjusted rates.) No other statistically significant differences existed among rates for all males, all females, or both sexes combined.

Among age-and-sex subgroups, there was no consistent pattern of statistically significant rate differences for any GI cancer

between Duluth and either of the comparison cities. The numbers of GI cancers that were diagnosed in Duluth residents in each year from 1969 through 1972 were tabulated (table 4) to examine for any trends over time.

Age-specific incidence rates increased progressively with age. For malignancies of all eight major primary anatomic sites except gall bladder and biliary tract, the age-adjusted incidence rates for males were higher than those for females. Analyses of data for whites only yielded results very similar to those for the entire population of the three cities; this was expected since more than 95 per cent of the population of each of these cities is white. Analysis of data on histologic types of malignancies did not yield any additional noteworthy information. with the vast majority of cancers identified as either "adenocarcinoma" or "carcinoma." There were no peritoneal mesotheliomas identified in residents of any of the three cities during 1969-1971 nor in Duluth residents in 1972. Approximately 90 per cent of the malignancies in this study were microscopically confirmed, and for each major type of GI cancer, the percentages microscopically confirmed in the three cities were similar.

TABLE 3

Statistically significant differences in crude GI cancer incidence rates for all ages combined among residents of Duluth, Minneapolis, and St. Paul, 1969-1971

Primary anatomic site	Sex	Statistically significant difference	p value
Stomach	Males	Duluth > St. Paul	<.05
	Males	Minneapolis > St. Paul	<.05
Large intestine	Females	Minneapolis > Duluth	<.01
	Both	Minneapolis > Duluth	<.01
	Both	St. Paul > Duluth	<.05
Pancreas	Both	Duluth > St. Paul	<.05
GI tract, not otherwise specified*	Males	Duluth > Minneapolis	<.01
	Males	Duluth > St. Paul	<.01
	Females	Duluth > St. Paul	<.01
	Females	Minneapolis > St. Paul	<.05
	Both	Duluth > Minneapolis	<.01
	Both	Duluth > St. Paul	<.001
Total	Females	Minneapolis > Duluth	<.05
	Both	Minneapolis > Duluth	<.05

* These differences are based on seven cases in Duluth, eight in Minneapolis, and one in St. Paul in 1969-1971.

TABLE 4

The number of gastrointestinal cancers diagnosed in Duluth residents during 1969-1972

Primary anatomic site	1969	1970	1971	1972
Esophagus	4	2	6	5
Stomach	17	21	14	15
Small intestine	1	4	1	1
Large intestine	34	35	44	43
Rectum/rectosigmoid	17	20	11	15
Liver	2	2	1	3
Gall bladder/biliary tract	2	7	5	5
Pancreas	17	22	15	13
Peritoneum, retroperitoneum, and abdomen, not otherwise specified	1	2	1	1
GI tract, not otherwise specified	2	0	5	2
Total	97	115	103	103

DISCUSSION

This study detected no excess in total GI cancer incidence among Duluth residents in 1969-1972 and therefore suggests no effect in this period possibly attributable to high concentrations of asbestos-like fibers in Duluth tap water, a finding consistent with two recent mortality studies regarding this situation (12, 13). It is impossible at present to know the significance of the differences in incidence rates of certain GI cancers that were observed.

Data in this incidence study are thought to be more reliable than data of the two previous mortality studies of cancer in Duluth in recent years because (a) data in these mortality studies were based on death certificate information, which is often inaccurate regarding cause of death, (b) many patients with GI cancer are cured, and (c) many others die of other illnesses and their active cancers are not recorded on their death certificates. Our findings are also thought to be reliable because Minneapolis and St. Paul are considered good comparison cities for Duluth and because the same methods were used to collect data for each of the three cities.

In attempting to determine the existence of a cause-and-effect relationship between an environmental factor and disease, the first step is often an examination of readily available data. This study may be considered one of several first steps in assessing the hazards of orally-ingested asbestos. Although large amounts of amphibole asbestos may have been present long before the EPA finding for 1964-1965, our data are probably best considered "baseline" data. They therefore do not rule out the possibility that there may be eventually demonstrated an association between drinking water with high concentrations of asbestos fibers and certain GI cancers. The long lag period between initial asbestos exposure and diagnosis of certain asbestos-associated cancers (1, 2, 6, 9) and the uncertainty of the exact length of time that

high concentrations of these fibers have been in Duluth water necessitates ongoing cancer surveillance for Duluth. We therefore plan to repeat this study and perform other cancer incidence studies in Duluth at regular intervals in the future. As part of such surveillance, we also plan to measure asbestos fiber concentrations in Minneapolis and St. Paul water to assure that these are appropriate comparison cities. If the water of these cities is eventually found to contain substantial amounts of asbestos, we will choose other comparison cities as cancer surveillance for Duluth continues.

Further studies bearing on the possible association between asbestos or asbestos-like fibers in drinking water and GI cancer and other human disease should include examining other water supplies and commercially-sold beverages and other food products in the United States for the presence of such fibers and for any association between consumption of them and human disease. Studies in Canada have demonstrated high concentrations of asbestos fibers in commercially-sold beverages and the water supplies of Montreal, Ottawa, and Toronto (14, 15). So study, however, has yet demonstrated any association between consumption of water or beverages containing high concentrations of asbestos fibers and human disease.

Whatever future studies may show, efforts are currently underway to remove fibers from Duluth drinking water and to control the presumed source of these fibers by attempting to halt the dumping of asbestos-containing taconite wastes into Lake Superior at the iron ore processing plant in Silver Bay.

REFERENCES

1. Selikoff IJ, Churg J, Hammond EC: Asbestos exposure and neoplasia. *JAMA* 188:22-26, 1964
2. Enterline PE: Mortality among asbestos products workers in the United States. *Ann NY Acad Sci* 132:156-165, 1965
3. McDonald JC, McDonald AD, Gibbs GW, et al: Mortality in the chrysotile asbestos mines and mills of Quebec. *Arch Environ Health* 22:677-686, 1971

either of the comparisons of GI cancers that Duluth residents in each year through 1972 were tabulated to examine for any trends

incidence rates increased with age. For malignancies of many anatomic sites and biliary tract, the incidence rates for males were higher than for females. Analyses of the data yielded results very similar for the entire population of Duluth as was expected since it is predominantly white. Analysis of the different types of malignancies revealed additional noteworthy differences. In the vast majority of cases, the cancers were either "adenocarcinomas." There were no differences identified in residence in the three cities during the study period in Duluth residents in 1972. The percentage of the malignancies that were microscopically confirmed for each major type of GI cancer in the three cities were similar.

TABLE 4
Initial cancers diagnosed in Duluth during 1969-1972

	1969	1970	1971	1972
	4	2	6	5
If	21	14	14	15
1	4	1	1	1
34	35	44	43	
17	20	11	15	
2	2	1	3	
2	7	5	5	
17	22	15	13	
1	2	1	1	
-	2	0	5	2
97	115	103	103	

4. Enterline P, DeCoufle P, Henderson V: Mortality in relation to occupational exposure in the asbestos industry. *J Occup Med* 11:897-903, 1972
5. Selikoff IJ, Hammond EC, Churg J: Carcinogenicity of amosite asbestos. *Arch Environ Health* 25:183-186, 1972
6. Elmes PC, Simpson MJC: Insulation workers in Belfast: 3. Mortality 1940-1966. *Br J Ind Med* 28:226-236, 1971
7. Hammond EC, Selikoff IJ, Churg J: Neoplasia among insulation workers in the United States with special reference to intra-abdominal neoplasia. *Ann NY Acad Sci* 132:519-525, 1965
8. Mancuso TF: Asbestos and neoplasia: Epidemiology: Discussion. *Ann NY Acad Sci* 132:589-594, 1965
9. Selikoff IJ, Hammond EC, Seidman H: Cancer risk of insulation workers in the United States. *Insulation Hygiene Progress Reports* 6:1-8, 1971
10. Cook PM, Glass GE, Tucker JH: Asbestiform amphibole materials: Detection and measurement of high concentrations in municipal water supplies. *Science* 185:853-855, 1974
11. *United States of America v. Reserve Mining Company*, 5-72 Civil 19
12. Mason TJ, McKay FW, Miller RW: Asbestos-like fibers in Duluth water supply: Relation to cancer mortality. *JAMA* 228:1019-1020, 1974
13. Minnesota Department of Health: Gastrointestinal cancer mortality data, 1955-1971. (Unpublished data)
14. Cunningham HM, Pontefract R: Asbestos fibres in beverages and drinking water. *Sature* 232:332-333, 1971
15. Kay C: Ontario intensifies search for asbestos in drinking water. *Water and Pollution Control* 33-35, September, 1973

MORTALITY OF WORKERS IN TWO MINNESOTA TACONITE
MINING AND MILLING OPERATIONS

W. Clark Cooper, MD, Otto Wong, ScD, and Robert Graebner, MPH

SYNOPSIS

Mortality during the years 1947-1983 was studied in 3,444 men employed for at least 3 months in Minnesota taconite mining operations during the years 1947-1958. During 86,307 person-years of observation there were 801 deaths for a standardized mortality ratio (SMR) of 88 (U.S. white male rates) or 98 (Minnesota rates). The 41 deaths from respiratory cancer were fewer than expected, the SMR being 61 ($p < 0.01$) (U.S. rates) and 85 (Minnesota rates). There were 25 respiratory cancers 20 or more years after first taconite employment, for an SMR of 57 ($p < 0.01$) (U.S. rates). SMR's for colon cancer, kidney cancer and lymphopietic cancer were elevated, but below the level of statistical significance. There was one death from pleural mesothelioma, 11 years after first taconite employment, in a man with long prior employment as a locomotive operator. The pattern of deaths did not suggest asbestos-related disease in taconite miners and millers.

Footnote for page 1:

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INTRODUCTION

Taconite is a hard, fine-grained, iron-bearing rock which after World War II became the principal form of iron ore mined in the United States, as natural hematite reserves became depleted. By 1978 it provided nearly 90% of the iron ore used in U.S. iron and steel industries. Of this, over 60% came from the Mesabi Range in Minnesota, where a deposit about 110 miles long and 1 to 3 miles wide extends roughly east to west from Babbitt to Grand Rapids.

The eastern end of the taconite ore body has been found to contain amphibole minerals in the cummingtonite-grunerite series, some of which are fibrous and similar to amosite, a type of commercial asbestos. When such fibers were found in the water supply of Duluth, 50 miles southwest from the point where mine tailings from one of the taconite processing plants were being deposited, there was concern as to possible effects on health. Concurrent with drastic measures to prevent

effluents from entering Lake Superior, studies were begun to determine if there was any evidence of effects on the health of Duluth residents, with concurrent studies of workers in the taconite mines and mills. So far such studies have been negative (1,2,4,5,6,7).

Two of the investigations of taconite workers involved analyses of mortality. The first, by Higgins (6), was based on 5,751 men employed by the Reserve Mining Company for one year or more in the period 1952 to 1976, and observed through 7/1/76. There were 298 deaths during 77,400 person-years of observation, with no significant excess deaths from any cause. Respiratory tract cancers were slightly fewer than expected, and no mesotheliomas were observed. The second study, by Cooper (7) involved 3,442 men who had worked in either the Erie or Minntac taconite operations for at least 3 months prior to January 1, 1959. They were observed through December 31, 1977, and there were 489 deaths during 65,306 person-years. There were no excess deaths from lung cancer or any other major cause. It was felt, however, that the results should be interpreted with caution, since only 8% of the person-years of observation were 20 or more years since first opportunities for exposure in the taconite industry, which did not begin to any major extent until 1947.

The present report is based on observation of the Erie-Minntac cohorts for an additional 6 years, through 1983, increasing the person-years of observation to 86,307 and the total number of deaths for analysis to 801.

MATERIALS AND METHODS

Choice of study population. When the study was being planned in 1978, descriptive data from all operating taconite mines in the U.S. was reviewed. There were only five which had begun operations before 1958. One of them, Reserve Mining Company, was already being studied independently. Two were relatively small and were located in Michigan. It was decided to limit the study to the Erie Mining Company (Erie) and U.S. Steel Corporation (Minntac), which would provide populations based in Minnesota, and which, when added to the Reserve mining population, would encompass a large proportion of those engaged in early taconite operations.

Data collection. Demographic and work history information from Erie was obtained from individual forms supplied by the company, which were based on Social Security Administration (SSA) and plant records. Four thousand forms were received giving name, social security numbers, hire dates, termination dates and successive job assignments for all former and current

employees (as of December 31, 1977) who had worked prior to January 1, 1959. After exclusion of female employees, those with missing birth or hire dates, and those having less than 90 days employment in a job with potential taconite exposure before 1/1/59, a population of 2,765 men qualified for the study. Four of these had also worked at Minntac.

Data from the Minntac mine and mill were collected in a more conventional way, being based on summary cards on file in the plant for all individuals employed since operations began. An experienced research analyst from EHA microfilmed 800 such cards for all individuals who appeared to meet the study criteria. From these, 68G ultimately qualified, including the four who had also worked for Erie.

Classification of jobs and exposures. The occupational exposures which were of most interest were those to particulates which might be classified as asbestiform, i.e. with 3 to 1 aspect ratios. However, there were no industrial hygiene data upon which to make direct estimates of past exposures to such particulates. There was quantitative information, however, on non-fibrous airborne dust which had been collected to evaluate quartz exposures. These provided a foundation for ranking the relative dustiness of work areas and jobs, when supplemented by subjective appraisals from plant personnel and industrial hygienists.

The work areas or departments in which employees worked were coded as follows: Mining - 01; coarse grinding - 02; fine grinding - 03; concentrating - 04; pelletizing - 05; general plant - 06; pellet handling - 07; maintenance - 08; service - 09; non-taconite operation - 10; non-study plants - 11; power plant - 12; preliminary taconite plant - 51; Erie's Hibbing laboratory - 52; taconite contracting company or TCC - 53.

Each job title within a work area was also assigned a two digit code. Each work area job title combination was characterized in terms of relative dustiness, 00 = non dusty; 01 = least, 02 = intermediate and 03 = highest. For analysis, it was therefore possible to describe each individual's periods of exposure both in terms of relative dust exposures and in terms of the stage in mine operations.

Followup. Initial followup through 1977 was based on determining cohort members known by the plants to be alive because they were still employed, receiving pensions, or living in the local communities. Rosters of those whose status was unknown to the plants were sent to the Social Security Administration (SSA) for followup through 1977 and again through 1983. SSA provided lists of those presumed (1) to be alive because of continued contributions or payment of benefits; (2) to be dead because of notification of death

having been received. There remained a residual group whose vital status was unknown to SSA or to plant personnel.

Determining and coding causes of death. For all who were reported dead, requests for death certificates were sent to the offices of vital statistics in the states of residence or death. The certificates which were obtained were filed and utilized with careful consideration of restrictions imposed by many of the states.

Causes of death were coded by a certified nosologist according to the 7th or 1955 Revision of the International Classification of Diseases. This involved translation, when necessary, of causes coded by the 8th (1965) and the 9th (1975) revisions to correspond to the rubrics in the earlier classification which had been the basis for the initial study of the cohort.

The numbers of dead workers for whom death certificates were not obtained is shown in individual tables. Based on the assumption that causes of deaths in these individuals had the same distribution as those that were certified, the cause-specific mortality ratios should be increased proportionately .

Analysis of data. Cohort members were considered as under observation from three months after their first exposure in a taconite operation until December 31, 1983, or the date of

death, whichever occurred first. Those whose vital status was unknown as of the study cut-off date of December 31, 1983 were considered under observation through the last date they were known to be alive, which could be the date of termination or, in those alive at the conclusion of the previous study, December 31, 1977. This is a compromise between dropping those of unknown status from the study, which would underestimate person-years of risk, or treating them as alive until 12/31/83, which would overestimate person-years.

The numbers of deaths observed were compared with the numbers expected, and expressed as standardized mortality ratios (SMR's) by use of the Occupational Cohort Mortality Analysis Program or OCMAP (8). The U.S. white male population was the basis for comparison, since it was known that there were very few non-whites in the study population. Limited comparisons were also made with the numbers of expected deaths based on Minnesota death rates.

Tests for statistical significance. The statistical significance of deviations of SMR's from 100 were tested by a method assuming a Poisson distribution (9,10,11); 95% (p 0.05) and 99% (p 0.01) confidence limits were calculated. Significantly high or low SMR's will be indicated in text and tables, by an asterisk (p 0.05) or double asterisk (p 0.01).

A two-tailed test of significance was used, as there was no a priori justification for assuming that the effect of working in a particular occupation could only have the effect of increasing an SMR.

RESULTS

Description of the study population. There were 3,444 men who met the study requirement of having been employed for 3 months or more in taconite operations at some time prior to January 1, 1959. Of these, 2,764 were Erie employees, 676 Minntac employees and 4 had worked in both plants. More than half were born before 1925, with the mean birth year being 1922. The average age at the beginning of followup was 33.2 years.

Table 1 summarizes the distribution by the year of first occupational exposure to taconite, from 1947 through 1958. By December 31, 1983, 25 years had elapsed for the 1958 hires and 36 years for the 1947 hires. For 19.8% of the population, those hired in 1953 or earlier, 30 years had elapsed. Thus there were ample opportunities for diseases with long latency to become manifest.

Prior occupational exposures. Only 1,968 cohort members had information on prior employment in their files. Of these,

1,223 had prior hematite mining experience. No separate analysis was made of this subcohort.

Followup and death certificate search. The vital status of 3,249 (95.8%) of the 3,444 cohort members was determined as of 12/31/83. As shown in table 3, 2,498 (72.5%) were known to be alive, 801 (23.3%) were dead, and the vital status of 145 (4.2%) was unknown. The latter fell into two categories: about 90 for whom neither SSA nor the plants could supply any information, and 55 in which there were discrepancies between names and social security numbers. Some of the latter might eventually be located.

The total number of person-years of observation was 86,307, of which 40,092 (46%) were after 1969 and 24,685 (29%) were after 1974. The 801 deaths represented 9.3 per 1000 person-years.

For the 801 deaths, certificates were obtained for 778, leaving 23 (2.9%) with unknown causes of death. This results in a slight understatement of cause specific mortality ratios. If one assumes that the unknown causes had the same distribution as those known, each cause specific SMR in the total cohort would be increased by 3.0% (23/778).

Standardized mortality for the total population. Table 3 summarizes deaths for major causes in all members of the cohort for the period 1959-1983, compared with deaths expected in U.S.

white males during the same years. The SMR for all causes was 88**, a highly significant difference from the U.S. average, but consistent with the healthy worker effect (i.e. those able to work usually have better than average health). There was a significant deficit in total deaths from malignancies (84*). Deaths from respiratory tract cancer (SMR = 61**), largely due to lung cancer (SMR = 59**), were strikingly low. There were elevations in SMR's for cancer of the large intestine (SMR = 128), kidney (SMR = 185) and the lymphopoietic system (SMR = 117), but none of these was statistically significant at the 95% level. Deaths from heart disease and other circulatory diseases were significantly fewer than expected (SMR = 89*). This largely represented a favorable pattern for arteriosclerotic heart disease (SMR = 85**), which is strongly influenced by the healthy worker effect. Deaths from non-malignant respiratory disease, which would include silicosis and asbestosis, were significantly below expected (SMR = 66*). As in the past, there was a non-significant excess of deaths from external causes, including accidents and suicide.

Deaths related to duration of employment and observation period. Table 4 summarizes the total deaths and SMR's based on the duration of employment in jobs with potential taconite exposures and periods of observation from the beginning of such

employment. Except for a slight upward trend in later observation periods, presumably attributable to the healthy worker effect, there was no striking pattern.

Table 5 similarly summarizes deaths from cancer of the respiratory tract, which showed no positive correlation with either length of exposure or time since exposure began. In the observation period 20 or more years since first exposure, the SMR was 57**, based on 25 deaths.

Deaths related to level and type of exposure. To determine if there was any suggestion of excess risk associated with level or type of exposure, the SMR's for a few causes of special interest were compared in several subcohorts. As can be seen in table 6, there was no evidence of any unusual pattern.

Occurrence of mesothelioma. There was one death attributed to pleural mesothelioma, in a 62-year-old white male. It had been classified as due to benign neoplasm, in conformity with the 1955 Revision of the ICD. Although the worker involved had been employed for 40 years, his exposures to taconite began only 11 years before his death. Prior employment had been principally as a locomotive fireman and engineer, in which there could have been exposures to asbestos from boiler insulation.

Standardized mortality ratios using Minnesota death rates.

Minnesota death rates are lower than U.S. rates for a number of causes. When they were used for determining expected deaths (table 7), the SMR's were higher for total deaths (98), total neoplasms (95), gastrointestinal cancer (97), respiratory cancer (85), and trauma (123*). Only the last reached a level of statistical significance at the 95% level. The SMR for lymphopietic cancer dropped to 108, as rates for this cause in Minnesota are higher than national rates. The available program did not separate kidney cancer from bladder cancer, but the two combined showed an SMR of 164, based on 15 cases; this was not a significant elevation. The corresponding combination in our study (not shown in tables using U.S. rates) had an SMR of 150.

When the analysis using Minnesota rates was limited to observation twenty years or more after first exposure, the findings were essentially the same, the SMR for all causes combined being 102, that for all neoplasm being 92, respiratory cancer 98 and lymphopietic neoplasms 111.

DISCUSSION

The original study and the current extension were carried out because of the possibility that cummingtonite-grunerite, an

asbestiform mineral found in some Mesabi Range taconite deposits, might cause asbestos-related disease, such as lung cancer. There is no evidence of such an effect. Respiratory tract cancer deaths were 39% fewer than expected, based on comparison with U.S. white males, and 15% fewer than expected when compared with Minnesota white males. Even when analysis was limited to deaths 20 or more years after first exposure, which provided ample opportunity for the leading edge of any excess in latent tumors to appear, there was no excess. The number of expected cases of respiratory tract cancer was sufficiently high to give the study considerable power. One can say with 80% probability of being correct ($\alpha = 0.05$ and $\beta = 0.2$) that the SMR for this cause in the total cohort could have been no greater than 132 using U.S. death rates, and no more than 139 using Minnesota death rates. The corresponding SMR, using U.S. rates, for those with 20 or more years potential latency could have been no more than 141.

There were no deaths from asbestosis. The only death from mesothelioma was in a man whose exposure to taconite began only 11 years before his death; he had worked as a locomotive fireman and engineer for thirty years previously. Mesotheliomas rarely occur sooner than 20 years after exposure to asbestos and commonly occur after 30 or more years. It is therefore plausible to hypothesize that asbestos from

locomotive boiler insulation was responsible for this tumor. One can be fairly certain that it was not due to taconite.

The small excess of kidney cancer deaths (9 vs. 4.9 expected) is probably not related to occupational exposures. Because of the small numbers, there is at least a 1-in-20 likelihood that the excess occurred by chance. There was no apparent correlation with either type or duration of exposure. Higgins (6) reported 3 urinary tract cancers (bladder plus kidney) with 2.97 expected, for an SMR of 101, in men employed by Reserve Mining Company for at least one year. Although Lawler et al (7) found 15 kidney cancer deaths with 9.3 expected (SMR 161) in above-ground hematite miners in Minnesota, underground hematite miners had no excess (11 with 11.1 expected).

The pattern of mortality observed in these cohorts does not suggest that dust exposures associated with taconite mining and milling are having any adverse effects on health. The continued deficit in deaths from lung cancer is especially reassuring. If asbestiform minerals were initiating such tumors, one would expect some excess cases to have become apparent, since all members of the cohort had begun employment in the industry at least 25 years before the end of the observation period. Studies of workers exposed to commercial asbestos have shown that excess deaths from lung cancer usually

become apparent within 20 to 25 years after first exposure, although delayed effects may continue for many more years. If there were a positive association between taconite exposures and lung cancer, sufficient time had elapsed in the present study for it to have become apparent.

REFERENCES

1. Cook PM, Glass GE, Tucker JH. Asbestiform amphibole minerals: detection and measurement of high concentrations in municipal water supplies. *Science* 1974;185-853-5.
2. Masson TJ, McKay FW, Miller RW. Asbestos-like fibers in the Duluth water supply: relation to mortality. *JAMA* 1974;228:1019-20.
3. Levy RS, Sigurdson E, Mandel J, taudan E, and Pearson J. Investigating possible effects of asbestos in city water: surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. *Am J Epidemiology* 1976;362-368.
4. Sigurdson EE, Levy BS, Mandel J, McHigh R, Michienzi LJ, Jagger H and Pearson J. Cancer morbidity investigations: lessons from the Duluth Study of Possible effects of asbestos in drinking water. *Env Res* 1981; 25:50-61.

5. Clark TC, Harrington VA, Asta J, Morgan WKC and Sargent EN. Respiratory effects of exposure to dust in taconite mining and processing. *Am Rev Resp Dis* 1980;121:959-966
6. Higgins ITT, Glassman JH, Oh MS, and Cornell RG. Mortality of Reserve Mining Company employees in relation to taconite dust exposure. *Am J Epidemiology* 1983;118:710-719.
7. Cooper WC: An epidemiologic study of workers in the taconite mining and milling industry. Report prepared for American Iron Ore Association, March 26, 1984.
8. Marsh GM and Preininger M. OCMAP: a user-oriented mortality analysis program. *Am Statistician* 1980;34:245-246.
9. Mantel N and Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. *J Nat Cancer Inst* 1959; 32:719-748.
10. Mantel N. Appendix C in Haenszel W, Loveland DB and Sirken MG. Lung cancer mortality as related to residence and

smoking histories in whites males. J Nat Cancer Inst
1962;23:947-997.

11. Bailer JC and Ederer F. Significant factors for the ratio of a Poisson variable to its expectation. Biometrics 1964;20:639-643.
12. Lawler, AB, Mandel JS, Schuman LM, and Lubin JH. A retrospective cohort mortality study of iron ore (hematite) miners in Minnesota. JOM 1985;27:507-517.

TABLE 1. DISTRIBUTION OF STUDY POPULATION BY
YEAR OF FIRST EXPOSURE TO TACONITE

Year	Number	Percent	Cumulative number	Cumulative percent
1947	2	0.1	2	0.1
1948	122	3.5	124	3.6
1949	41	1.2	165	4.8
1950	61	1.8	226	6.6
1951	57	1.7	283	8.2
1952	103	3.0	386	11.2
1953	295	8.6	681	19.8
1954	283	8.2	964	28.0
1955	425	12.3	1389	40.3
1956	308	8.9	1697	49.3
1957	1283	37.3	2980	56.5
1958	464	13.5	3444	100.0

TABLE 2. STATUS OF STUDY POPULATION AS OF
DECEMBER 31, 1933

Total	3,444 (100%)
Alive	2,498 (72.5%)
Unknown	145 (4.2%)
Dead	801 (23.3%)

With certificate 778 (97.1%)

No certificate 23 (2.9%)

TABLE 3. OBSERVED AND EXPECTED DEATHS BY MAJOR CAUSES, 1948-1983 IN TACONITE MINERS AND MILL WORKERS EMPLOYED IN TACONITE-EXPOSED JOBS FOR 3 MONTHS OR MORE PRIOR TO JANUARY 1, 1959, COMPARED WITH U.S. WHITE MALES

CAUSE OF DEATH (ICD 7th Rev. 1955)	Deaths			Confidence limits	
	OBS	EXP	SMR	95%	99%
All causes (001-998)	801	914.0	88**	82-94	80-96
All malignant neoplasms (140-205)	158	188.2	84*	71-98	68-103
Digestive organs and peritoneum (150-159)	48	49.6	97	71-128	64-139
Stomach (151)	5	0.7	58	19-135	13-164
Large intestine (153)	21	16.4	128	79-196	68-220
Respiratory system (160-164)	41	67.7	61**	43-82	39- 89
Bronchus, trachea, lung (162-163)	38	64.3	59**	42-81	37- 89
Kidney (180)	9	4.9	185	85-351	64-411
Lymphopoietic system (200-205)	22	18.9	117	73-177	62-197
All diseases circulatory system (400-468)	360	406.4	89*	80-98	77-101
Arteriosclerotic heart disease (420)	289	339.6	85**	76-96	73- 99
Non-malignant respiratory disease (470-527)	34	51.7	66*	45-92	40-101
Cirrhosis of liver (581)	22	27.5	80	50-121	43-135
All external causes of death (800-998)	105	94.3	111	91-135	86-143
All accidents (800-962)	73	62.9	116	91-146	84-156
Motor vehicle accidents (810-835)	30	29.3	102	69-146	61-161
Suicides (963, 970-979)	29	22.6	128	86-184	75-203
Cause unknown	23	---	---	-----	-----
Number of workers			3,444		
Number of person-years			86,341		
Deaths per 1,000 person-years			9.3		
Adjustment of cause-specific SMR's for missing certificates			+3.0%		

* Significant at the 5% level, i.e. $p = (0.05)$

** Significant at the 1% level, i.e. $p = < 0.01$

TABLE 4. DISTRIBUTION OF TOTAL DEATHS, WITH STANDARDIZED MORTALITY RATIOS (SMR's) BY DURATION OF EMPLOYMENT AND OBSERVATION PERIODS (COMPARISON WITH U. S. RATES)

Duration of Employment (Years)	Observation period (years from first taconite exposures)							
	10		10-19		20		Total	
	No.	SMR	No.	SMR	No.	SMR	No.	SMR
0.25 - 0.99	24	81	56	126	83	110	163	109
1 - 4.99	37	76	47	74*	132	85	216	81**
5 - 9.99	28	77	21	66	75	89	124	81*
10 or more	--	--	77	69***	221	95	298	87"
Total	89	77*	201	80**	511	93	801	88**

TABLE 5. DISTRIBUTION OF DEATHS FROM RESPIRATORY CANCER
(ICD 160-65) STANDARDIZED MORTALITY RATIOS (SMR's)
BY DURATION OF EMPLOYMENT AND OBSERVATION PERIODS
(COMPARISON WITH U.S. RATES)

Duration of Employment (Years)	Observation periods (years from first taconite exposures)							
	10		10-19		20		Total	
	No.	SMR	No.	SMR	No.	SMR	No.	SMR
0.25 - 0.99	1	87	3	97	8	50	7	68
1 - 4.99	2	100	4	93	7	65	13	76
5 - 9.99	2	103	1	46	2	32	5	48
10 or more	--	---	3	34*	13	62	16	53**
Total	5	98	11	59	25	57**	41	61**

Table 6. OBSERVED AND EXPECTED DEATHS 1948-1983 FOR SELECTED CAUSES IN SUBCOHORTS OF TACONITE MINERS AND MILL WORKERS WHO EVER OR NEVER WORKED IN INDICATED AREAS (U.S. Rates)

Subcohort (work area code)	OBS	All deaths		Respiratory cancer			Kidney cancer		
		EXP	SMR	OBS ¹	EXP	SMR	OBS	EXP	SMR
Mining (01)									
Ever 398	398	462	86**	24	34	70	5	2.4	205
Never 403	403	452	89**	17	33	51**	4	2.3	172
Grinding & Conc (02-04)									
Ever 243	243	297	82**	12	23	52*	4	1.7	241
Never 558	558	617	91*	29	45	65*	5	2.2	156
Pelletizing (05)									
Ever 245	245	281	87*	12	21	56*	3	1.5	198
Never 556	556	633	88**	29	46	62*	6	3.4	179
In any of above									
Ever 589	589	695	85**	30	52	58**	8	3.7	217
Never 212	212	219	97	11	16	69	1	1.2	84
In high or medium dust area									
Ever 539	539	571	94	27	43	63*	6	3.1	195
Never 262	262	343	77	14	25	56	3	1.8	170
In high dust area									
Ever 270	270	282	96	12	21	57*	0	1.5	---
Never 531	531	632	84	29	47	62*	9	3.4	265
Total population	801	914	88**	41	68	61**	9	4.9	185

* Significant at the 5% level, i.e. $p < 0.05$

** Significant at the 1% level, i.e. $p < 0.01$

TABLE 7. OBSERVED AND EXPECTED DEATHS BY MAJOR CAUSES IN TACONITE MINERS AND MILL WORKERS WHO WORKED AT LEAST 3 MONTHS IN TACONITE-EXPOSED JOBS PRIOR TO JANUARY 1, 1959 COMPARED WITH MINNESOTA WHITE MALES

CAUSE OF DEATH (ICD No., 7th Rev 1955)	Deaths		SMR	Confidence
	Observed	Expected		limits
				95%
All causes (1-999)	801	820.5	98	91-105
All neoplasms (140-205)	158	166.0	95	81-111
All gastrointestinal (150-159)	48	49.5	97	71-129
Respiratory (160-165)	41	48.0	85	61-116
Lymphopoietic (200-205)	22	20.4	108	67-163
Kidney and bladder (180-181)	15	9.1	164	92-271
Cardiovascular (400-443)	342	345.0	99	89-110
Selected respiratory (241, 480-83, 490-493, 500-502)	20	21.4	94	57-145
Most trauma (800-965, 970-979, 980-985)	105	85.3	123*	101-149
Motor vehicle accidents (810-835)	30	29.9	101	68-144
Other causes	153		---	----
		202.0		
Cause unknown	23	---		----
Number of workers		86,342		
		3,444		
Number of person-years				
Adjustment of cause-specific SMR's for missing certificates		+3.0%		

* Significant at the 5% level, i.e. $p = < .05$

Significant at the 1% level, i.e. $p < 0.01$