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New York State Department of Health

Investigation of Cancer Incidence among the Woodstock Asbestos Exposure Registry Population

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Executive Summary

Late in 1985, asbestos contamination was discovered in the public water supply of the Town of Woodstock, Ulster County, New York. Contamination resulted from asbestos-cement (AC) pipes installed in the town water system in the mid to late 1950s and the corrosiveness of the local water. The New York State (NYS) Department of Health established the Woodstock Asbestos Exposure Registry (WAER) in 1986 to monitor rates of cancer among individuals who lived on the water supply between 1960 and 1985. Demographic, health, and residential information were collected on 2,936 registrants. The follow-up period for observation of cancer was 1980-1998, consistent with the expected lag of 20-30+ years for development of asbestos-related cancers. The NYS Cancer Registry was used to confirm cancer diagnoses and to identify unreported cancers. Standardized incidence ratios (SIRs) for gastrointestinal, respiratory, and total cancers were all approximately 1.00 or less and all 95% confidence intervals (CIs) included 1.00. For individual types of the gastrointestinal cancers, only the SIR for pancreatic cancer was marginally statistically significant at 2.19 (95% CI=1.00-4.16), based on a total of nine observed cases. The excess in pancreatic cancer occurred primarily among men (SIR=3.08; 95% CI=1.13-6.70), and was only slightly elevated among women (SIR=1.39; 95% CI=0.29-4.06). This association may be related to factors other than asbestos exposure such as cigarette smoking, or to chance. No cases of mesothelioma were observed among WAER participants. There was no increase in incidence by latency or duration of residence on the water supply, but the ability to detect these trends is limited by small numbers and unknown dates of initial exposure. The general pattern of results did not demonstrate a likely link between exposure to asbestos in drinking water and cancer occurrence among participants in the WAER. This is the final report for this study; follow-up of the Woodstock Asbestos Exposure Registry has ended.

Background

In November 1985, residents of the Town of Woodstock, Ulster County, NY, reported a decrease in water pressure following a temporary interruption in water service. Town and New York State (NYS) Department of Health (DOH) staff determined that strainers on faucets and showerheads were clogged with asbestos fibers. The source of the asbestos fibers was asbestos-cement (AC) pipes installed in the town water system in the mid to late 1950s. Examination of the pipe showed significant deterioration of its interior, probably due to the high corrosivity of the local water. All water delivered by the public water supply traveled through AC pipes located near the pumping stations. The time frame during which asbestos fibers started leaching into the water is unknown but the entire town water supply system may have contained some level of asbestos fibers since around 1960. A 10-year-old sample of water drawn in 1976 (tested in 1986) contained asbestos, confirming that leaching of asbestos into the water supply began as early as 1976.

A 1982 survey of 47 NYS public water supply systems performed by the United States Environmental Protection Agency (USEPA), which did not include the Woodstock water supply, found only one water system with an asbestos level greater than 10 million fibers per liter (MFL). Ulster County officials collected five water samples from different locations on the Woodstock water supply in November 1985, following flushing of the water mains. Four of the samples had asbestos levels greater than 10 MFL, with the maximum equaling 304.5 MFL.

To address the asbestos contamination problem, a variety of actions were taken. In December 1985, a water advisory was issued, cautioning people against use of the town water supply for drinking, cooking, food preparation, or mist-type humidifiers. Emergency procedures also included distribution of uncontaminated water and replacement of the AC pipes with ductile iron pipes. The majority of the AC pipe replacement was completed in early March 1986, with a few remaining sections replaced in early June. Extensive flushing of the water supply

system, cleaning or replacement of water service meters, and repeated testing for asbestos were conducted before lifting the water use advisory in July of 1987.

Carcinogenicity of Asbestos

Asbestos has been classified as a human carcinogen by the USEPA and the International Agency for Research on Cancer (IARC) (USEPA, 1993), (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 1987). Numerous studies have linked occupational exposure to airborne asbestos with excess risk of lung cancer and mesotheliomas of the pleura and the peritoneum (Selikoff, 1965), (Peto et al., 1985), (Enterline et al., 1987). A weak association between occupational exposure to airborne asbestos and gastrointestinal cancer was first noted by Selikoff et al., (1964), and was supported by a meta-analysis of 31 occupational cohorts using a lung cancer standardized mortality ratio of 2.0 or greater as a proxy for asbestos exposure (Frumkin and Berlin, 1988). Investigators proposed that the increase in gastrointestinal cancers was the result of ingestion of airborne asbestos fibers that occurred following clearance from the airways. A more recent meta-analysis of 69 occupational cohorts does not support a causal relation (Goodman et al., 1999). Some investigators have speculated that the elevated rate of gastrointestinal cancer attributed to occupational asbestos exposure reflects misdiagnosis of mesothelioma and lung cancer (Peto, 1989), (Gamble, 1994). Peto (1989) noted that relative risks for cancers other than lung and gastrointestinal are correlated with relative risks for lung cancer in cohorts of male asbestos workers. An increase in risk for all other sites of cancer similar to that observed for gastrointestinal cancer supports the misdiagnosis theory.

Both human and animal studies have examined the relationship between ingestion of asbestos fibers and cancer. Animal studies of ingested asbestos carcinogenicity have generally produced negative results (Chouroulinkov, 1989). However, based on the results of genotoxicity studies, Varga et al., (1999) suggest that combined exposure to asbestos fibers and waterborne asbestos may have a carcinogenic effect. Cancer incidence or mortality has been studied in eight populations exposed to asbestos in drinking water in the United States, Canada, and Norway (see Table 1). In several instances, the same study population was the subject of two or three investigations. A later study extended the scope or study period of the original investigation. The results of the most recent study of each population are summarized in Table 1.

Erosion of rock containing asbestos was the source of asbestos in several study locations: the Bay Area of California; Quebec, Canada; and the Puget Sound region of the state of Washington. Dumping of mine tailing wastes into Lake Superior resulted in asbestos in the water supply in Duluth, Minnesota. AC pipes were the source of asbestos in selected water supplies in Connecticut and Escambia County, Florida. Weathering of AC roof tiles by rainwater runoff used for drinking water was the source of asbestos exposure among lighthouse keepers in Norway. In some cases, asbestos levels were relatively low. Concentrations were less than 0.1 MFL in town water supplies in Connecticut (Meigs et al., 1980); in Escambia County, Florida, seven census tracts with detectable levels measured 0.11-0.7 MFL (Millette et al., 1983). Much higher concentrations were measured in lighthouse keepers' cisterns (range=1,760-71,350 MFL) (Andersen et al., 1993), in water supplies in two cities in Quebec, Canada (> 100 MFL) (Toft et al., 1981), and in the Puget Sound area of Washington (200 MFL) (Polissar et al., 1984). Intermediate levels were reported in water supplies in the Bay Area of California (up to 36 MFL) (Conforti et al., 1981) and Duluth, Minnesota (range=2-64 MFL) (Sigurdson, 1983).

As shown in Table 1, an excess of stomach cancer among males was observed in five study populations (Conforti et al., 1981), (Toft et al., 1981), (Sigurdson, 1983), (Polissar et al., 1984), (Andersen et al., 1993), and pancreatic cancer was associated with exposure among males in one population (Meigs et al., 1980) and among females in another (Conforti et al., 1981). A positive association was not reported for more than one study population for any other cancer site. Despite indications of increased risk in early investigations, the epidemiologic studies conducted to date generally do not provide strong evidence of an association between exposure to asbestos in drinking water and gastrointestinal or respiratory cancers. However, a number of study limitations prevent firm conclusions. Six of the eight studies employed an ecologic design. The most serious weakness of ecologic studies is the potential for bias in attempting to draw conclusions about individual-level risk based on group-level observations. The assignment of exposure status to all individuals in a region undoubtedly results in exposure misclassification. Geographic boundaries used to identify cases and controls often do not correspond perfectly with water system boundaries. In addition, migration can contribute to misclassification since residents who have recently moved into an "exposed" region would not be at risk of exposure-related cancer. Other considerations include inadequate latency (Meigs et al., 1980), (Sigurdson, 1983), (Howe et al., 1989) and small sample size.

The case-control study by Polissar et al. (1984) was able to avoid many of these limitations. Detailed information

on residential and occupational history was used to estimate asbestos exposure, data were collected on personal risk factors for cancer, and latency was taken into account. Statistically elevated risk of cancers of the stomach and pharynx were reported among men. In view of the large number of statistical comparisons (84) in conjunction with nonsignificant protective effects observed among women for cancers of the stomach and pharynx, the authors suggested that positive findings were due to chance. The study had power to detect relative risks as low as 1.4-1.6 for cancers of the colon, lung, gastrointestinal system, and respiratory system.

A preliminary study of cancer incidence for the years 1973-1983 among Woodstock residents living in the census blocks that include the water district, conducted by the NYS DOH, is included among the studies summarized in [Table 1](#) (Howe et al., 1989). No evidence of elevated gastrointestinal or respiratory cancer incidence was observed when rates of cancer for census blocks including the water district were compared to rates for NYS excluding New York City (NYC). Insufficient latency and the inclusion in the exposed population of individuals who did not live on the public water supply were limitations of the study.

Most of the community studies conducted to date did not have individual-level information on source of water and duration of residence. The exposure assessment for the present study improves on these studies. Only people who lived on the water supply were included and information on duration of residence was examined.

Study Subjects and Methods

Construction of Cohort

In 1986, the NYS DOH began a prospective cohort study to monitor cancer incidence among individuals who lived in homes serviced by the Woodstock water supply. Specifically, NYS DOH established the Woodstock Asbestos Exposure Registry (WAER) to collect exposure and health status information on individuals who, between 1960 and 1985, had resided for six months or more in a home serviced by the Town of Woodstock water supply. Information on demographics, smoking history, drinking habits, occupation, family history of cancer, and residential history was obtained through questionnaires and interviews. The registrants were followed through 1998. Address and health information was updated every two years. The objectives of the WAER were:

- To identify individuals who lived for six months or more in a residence served by the town water between January 1, 1960, and December 31, 1985.
- To notify these individuals of their exposure.
- To obtain demographic and medical history data on registered individuals and to periodically update this information.
- To calculate cancer incidence rates among the WAER population for total cancers, mesothelioma, respiratory cancers and gastrointestinal cancers and compare these to cancer rates for NYS excluding NYC.

An in-person registration week was held in the Woodstock Town offices in June 1986. The following month, introductory letters and questionnaires were mailed out to an additional 565 households currently serviced by the public water supply. A variety of sources were used to identify and trace former property owners and tenants: tax assessor's records, voter records, city directories, post office change-of-address records, neighbor referrals, and Department of Motor Vehicle files. Extensive efforts were made to contact and recruit current and former residents. If possible, individuals who did not respond to mailed questionnaires were contacted by telephone and offered a telephone interview or remailing of the questionnaire. Those with non-published telephone numbers or otherwise not reachable by telephone were mailed registered letters.

The person-years of observation contributed by each person in the study cohort began at the start date of the follow-up period (January 1, 1980), or on the date of first residence on the water supply if residence began after 1980 (exceptions are noted below for the analyses accounting for latency and exposure beginning in 1976). Follow-up ended at the date of cancer diagnosis, date of death, last date before loss-to-follow-up or the end of the study period (December 31, 1998), whichever came first. An individual diagnosed with cancer no longer contributed person-years after his or her date of diagnosis. Similarly, accrual of person-years stopped as of date of death or loss-to-follow-up. Registrants for whom information was missing on duration of residence or date of birth were excluded from the study cohort. Since individuals who had a cancer diagnosis prior to 1980 would not be at risk of a first diagnosis of a primary cancer during the study follow-up period, persons with a confirmed diagnosis of cancer prior to 1980 were excluded from the study population.

Reference population

The white population of NYS excluding NYC was used as the reference population, or comparison population, for our study. This population was selected since it is a relatively large population with stable rates. The sociodemographic characteristics of the NYC population differ substantially from those of Upstate New York, justifying exclusion of NYC from the comparison group. Although all races were included in the study cohort, the reference population was restricted to whites because the study cohort was approximately 97% white, whereas the 1990 population of NYS excluding NYC was about 90% white.

To evaluate whether the Woodstock cohort differs from the reference population on risk factors for cancer, smoking and alcohol use reported by cohort members 18 years and older were compared to Behavioral Risk Factor Surveillance System (BRFSS) estimates for white residents of NYS excluding NYC. For the years 1986-1988, the BRFSS survey included approximately 700 residents of NYS excluding NYC each year (2037 total). We compared aggregated BRFSS data for the years 1986-1988 to data on Woodstock residents registered between 1986 and 1988. The BRFSS question, "Have you smoked at least 100 cigarettes in your life?" was closest in content to our category of "ever smokers," obtained by aggregating current smokers and past smokers. We categorized average number of drinks per day as "greater than two" and "less than two" in order to match the BRFSS category of two or more drinks per day, categorized as "chronic drinking."

Health outcomes

The health outcome of the study is a first diagnosis of a primary cancer, with a focus on gastrointestinal, respiratory, and total cancers. The NYS Cancer Registry (NYSCR) was used to confirm cancer diagnoses and to identify unreported cancers among WAER participants. NYSCR data were also used to calculate cancer rates for the reference population. Since the entire town water supply may have contained some level of asbestos starting around 1960, the follow-up period for observation of outcomes was defined as January 1980 through December 1998. Starting follow-up in 1980 allowed 20 years of latency, which is consistent with the expected latency of 20-30 years or more for asbestos-related cancers (Mossman and Gee, 1989). Although the NYSCR has been in existence since 1940, a start date of 1980 also was of practical significance since NYSCR data were complete on a statewide basis and accessible beginning in 1980.

International Classification of Diseases 9th revision (ICD-9) codes 150-159 were used to define gastrointestinal cancers and include cancers of the esophagus, stomach, small intestine, colon, rectum, liver, gall bladder, pancreas, and peritoneum. Respiratory cancers were defined as ICD-9 codes 161-163 and 164.2-165.9, which encompass cancers of the larynx, trachea, bronchus, lung, pleura, and mediastinum. Mesotheliomas were identified as malignant tumors reported to the NYSCR with a morphology code (based on ICD-02) between 9050 and 9055. ICD-9 codes 140-208 were included in the total cancers category.

Participants who moved out of NYS

It was not feasible for us to obtain comparable ascertainment of incident cancers for WAER participants living out of state. For this reason, the primary data analysis focuses on the subgroup of the cohort consisting of individuals who were NYS residents (referred to as "NYS residents") at the last date of follow-up and include only cancers confirmed by the NYSCR. In addition, all analyses were repeated for the entire study cohort ("whole cohort"). For analysis of the whole cohort, outcomes consisted of self-reported cancer diagnoses among residents who moved out of state and confirmed cancer diagnoses among NYS residents.

Exposure assessment

Timing and duration of residence in a home serviced by the Woodstock water supply were used as indirect measures of asbestos exposure. Duration and latency analyses were limited to gastrointestinal and respiratory cancer categories due to small numbers within duration and latency strata for individual cancer subsites.

Duration of residence

Duration of residence on the water supply was used as a proxy measure of cumulative exposure. Stratified analyses were performed based on duration of residence on the water supply from 1960 through 1985. Duration of residence was categorized as less than five years, five to 15 years, and greater than 15 years.

Latency

A time interval, or latency, of 20 to 30 years or more has been observed between first exposure to asbestos and diagnosis of asbestos-related cancers (Mossman and Gee, 1989). Latency was taken into account in the analysis by performing lagged analyses in which person-time at risk began after an interval following first exposure. The analysis was repeated for three "lag" periods: five, 10, and 20 years following first exposure. For the five-year lag, people who moved into a Woodstock residence during or before 1975 entered follow-up in 1980. People who moved onto the water district in subsequent years (i.e. 1976 through 1985) had a lag period of five years between first residence on the water supply and start of follow-up. The 10-year and 20-year lagged analyses were handled similarly, providing minimum intervals of 10 years and 20 years before start of person-time accrual.

Start of exposure

The earliest evidence of asbestos in the Woodstock drinking water dated to 1976. We have no measurements of drinking water asbestos levels during the first 15 years that the AC piping was in place. For this reason, in addition to analyses based on the assumption that exposure began in 1960 soon after the AC piping was first installed, we also repeated all analyses using a more conservative assignment of exposure with 1976 as the start date.

Water use patterns

We collected information by questionnaire on consumption of tap or bottled water, clothes dryer use and venting, and humidifier use. Response frequencies are reported as descriptive information. These data were not part of a detailed quantitative assessment because the small numbers of observations would not support comparisons based on these characteristics.

Statistical Analysis

Standardized incidence ratios (SIRs) and 95% confidence intervals (CIs) were calculated for individual cancer sites and the cancer groupings of interest: total, gastrointestinal, and respiratory cancers. An SIR is the observed number of cancers in the study population divided by the expected number of cases (Kelsey et al., 1996). The expected number of cases were calculated as the product of person-years of observation and cancer incidence rates from the reference population for each stratum of age (0-44, 45-49, ..., 80-84, >85 years), sex, and calendar year (1980-1984, 1985-1989, 1990-1994, 1995-1998). Ages <45 years were grouped since gastrointestinal and respiratory cancers are rare in younger age groups. Confidence intervals for the SIR were calculated based on the Poisson distribution.

Results

The 625 service connections on the Woodstock water supply included approximately 67 nonresidential connections, 14 additional connections to parcels with multiple family dwellings, five residences recently connected to the water supply in 1985, and three vacant dwellings. Although it was difficult to distinguish solely commercial properties and commercial properties with residential units, approximately 536 properties with residential units were serviced by the Woodstock water supply in 1985 (including about 48 parcels with multiple family dwellings). Of approximately 488 single-family dwellings, 394 households (80.7%) serviced by the public water supply in 1985 were represented in the WAER. We were unable to accurately estimate response rates for multiple family units. In all, 2,936 current or former Woodstock residents participated in the WAER.

A total of 247 WAER participants died or were diagnosed with cancer prior to 1980 and were therefore not included in the study cohort. An additional 66 registrants were excluded from analysis because of missing date of birth (52) or missing duration of residence on the water supply (14). The study cohort consisted of the remaining 2,623 WAER participants. [Table 2](#) shows the exclusions and follow-up status for the WAER. Fifty-nine individuals in the study cohort were lost to follow-up prior to 1998, 712 moved out of state, and 1,852 remained in NYS.

[Table 3](#) summarizes the gender, age, and race distribution of the cohort along with data on smoking and alcohol use among cohort members 18 years and older at registration. Race was not included on the questionnaire at the start of enrollment, resulting in a high number of missing observations for that variable. The person who completed the questionnaire will be referred to as the primary respondent. Unknown values for smoking and

alcohol use most often occurred for individuals who were not primary respondents. Unknown responses were recorded more than five times more frequently for individuals for whom information was provided by a family member or friend, compared to primary respondents. Among those for whom relationship to the respondent was known, information was provided by the participant himself or herself for 36.3% of cohort members, by an immediate family member for 54.7%, by an extended family member for 4.2%, and by an unrelated source for the remaining 4.8%.

Smoking and alcohol use reported by cohort members 18 years and older is compared to Behavioral Risk Factor Surveillance System (BRFSS) estimates for white residents of NYS excluding NYC in [Table 4](#). Exclusions based on age, interview date, and missing values reduced the Woodstock population to 1,586 and 1,408 for the smoking and alcohol use comparisons, respectively. Although the stratified estimates are based on small numbers for both the WAER and BRFSS populations and are therefore fairly unstable, the proportion of ever smokers and chronic drinkers was not consistently higher or lower among men and women in the Woodstock cohort compared to the BRFSS estimate. The differences in reported ever smoking and chronic drinking among males 18-24 years of age might be explained by proxy reporting since a parent provided information for 75% of Woodstock participants in this age group.

Water use patterns

Routine use of tap water for drinking and cooking was reported for 97.5% of the WAER study cohort. A clothes dryer vented indoors was reported for 6.9% of the study cohort and use of a humidifier in the home for 16.2%. Small numbers prohibited comparison of cancer incidence by water use patterns.

NYS residents, 1960-1985

[Table 5](#) presents observed numbers of cancers and SIRs for the unlagged analysis based on residency on the water supply between 1960 and 1985 among NYS residents. The SIRs for total, gastrointestinal, and respiratory cancer groupings are all approximately 1.00 or slightly below 1.00 and all confidence intervals (CIs) include unity. For the gastrointestinal subsites, the SIR for pancreatic cancer was marginally statistically significant at 2.19 (95% CI=1.00-4.16). The excess in pancreatic cancer occurred primarily among men (SIR=3.08; 95% CI=1.13-6.70) and was only slightly elevated among women (SIR=1.39; 95% CI=0.29-4.06). The SIR for esophageal cancer was also elevated among men. However, the increase was not statistically significant and was based on a small number of cases. With one exception, all respiratory cancers were cancers of the lung and bronchus. Therefore respiratory cancers are not listed by subsite. No cases of mesothelioma were observed among WAER participants.

Duration of residence on the Woodstock water supply is examined in [Table 6](#). No discernable patterns are observed in the incidence of gastrointestinal cancers across the three categories of length of residency. While SIRs for respiratory cancer increased with duration among males, the SIR remained less than 1.00 even in the longest duration category. Confidence intervals for all duration categories overlapped and none excluded unity.

The results of the lagged analyses are shown in [Table 7](#). Increasing lag periods resulted in small decreases in the SIRs for gastrointestinal cancer among both men and women and small increases in the SIRs for respiratory cancer among women. The confidence intervals for the different lag periods overlapped and none excluded unity with the exception of a reduced risk of gastrointestinal cancer among women associated with a minimum of 10 years following first exposure. The numbers of observations were too small to analyze individual gastrointestinal sites by latency.

Although the number of individual gastrointestinal subsite cases was too small to permit formal analysis of latency and duration, duration and latency were examined for pancreatic cases to see if the length and timing of residence on the water supply were consistent with an influence of asbestos exposure on cancer risk. Duration of residence on the Woodstock water supply was less than ten years for five pancreatic cancer cases, 10 to <20 years for two cases, and 20 years or more for two cases. Latency between start of residence on the Woodstock water supply and cancer diagnosis was less than 10 years for three pancreatic cancer cases, 10-<20 years for two cases, and 20 years or more for four cases.

NYS residents, 1976-1985

The analyses reported above were repeated using 1976 as the date of first exposure. The results for total, gastrointestinal, and respiratory cancer incidence (Table 8) are generally similar to those assuming exposure started in 1960 (Table 5). Duration and latency analyses were also performed using the 1976 exposure start date. Again, the results were similar to those presented in Tables 6 and 7 which assumed exposure started in 1960 (data not shown).

Whole cohort, 1960-1985

Analysis of cancer incidence for the whole cohort (including residents who moved out of state), based on exposure from 1960 through 1985, is summarized in Table 9. The incidence of gastrointestinal cancer, respiratory cancer, and all cancers combined was very similar to that observed for the NYS residents cohort (Table 5).

Discussion

This prospective cohort study, with a retrospective component for the years 1980-1985, did not demonstrate an increased incidence of total gastrointestinal cancer, total respiratory cancer, or all cancers combined among individuals living on a water supply contaminated with asbestos. When individual gastrointestinal cancers were examined, only pancreatic cancer was significantly elevated and the excess occurred primarily among males.

In previous epidemiologic studies, an association has been observed between asbestos fibers in drinking water and incidence of stomach cancer and other gastrointestinal or respiratory cancers (Meigs et al., 1980), (Conforti et al., 1981), (Toft et al., 1981), (Sigurdson, 1983), (Polissar et al., 1984), (Andersen et al., 1993), while others do not support an association (Millette et al., 1983), (Howe et al., 1989). Our study does not confirm the increased risk of stomach cancer noted in some prior studies. The observation of an excess stomach cancer risk in these other studies was limited to men, suggesting that occupation or lifestyle characteristics that differ by gender may play a role in the findings. Sigurdson (1983) noted that stomach cancer mortality had been elevated in the Duluth population prior to the start of asbestos exposure, possibly due to ethnic dietary practices. In the Duluth population, stomach cancer mortality was also elevated among females but the difference was not statistically significant. Norwegian lighthouse keepers exposed to asbestos-contaminated drinking water also experienced higher than expected rates of stomach cancer. Andersen et al. (1993) speculated that lack of refrigeration equipment prior to the late 1960s and a diet high in dried, salted, and smoked foods, may have accounted for the elevated incidence of stomach cancer among lighthouse keepers.

Although we observed a statistically significant excess of pancreatic cancer among males, the incidence of other individual gastrointestinal cancer sites, respiratory cancer, and all cancers combined, was not elevated and most SIRs were less than 1.00. Of the studies summarized in Table 1, a significant association between asbestos exposure and pancreatic cancer was observed in studies by Meigs et al. (1980) and Conforti et al. (1981). Meigs et al. (1980) presented site- and sex-specific results for two study periods: 1955-1964 and 1965-1974. The regression coefficient for the estimated concentration of asbestos fibers in the water supply was statistically significant for pancreatic cancer among males for the 1955-1964 study period only. Since little AC pipe was reported to have been in place in Connecticut water supplies prior to 1950, a stronger association would instead have been expected for the 1965-1974 interval if pancreatic cancer was causally related to asbestos exposure (Meigs et al., 1980). In the study by Conforti et al. (1981), a positive association between asbestos and pancreatic cancer among females was based on significant correlation and regression coefficients. The authors noted that the fit of the regression model was poor and suggested that the "regression data must be viewed with caution." A test for trend to determine the presence of a dose-response relationship between asbestos exposure and pancreatic cancer was not significant. Also a nonsignificant excess of pancreatic cancers among females and a nonsignificant deficit of pancreatic cancers among males were observed in the case-control study by Polissar et al. (1984). Although a positive association between exposure to asbestos in drinking water and pancreatic cancer was noted in an early investigation of some of the other study populations cited in Table 1 (Masson et al., 1974), (Levy et al., 1976), (Wigle, 1977), the association did not persist in follow-up investigations of the same populations with an extended observation period and longer latency.

Several explanations other than a causal relation might account for the observed excess of pancreatic cancer among males in our study. Cigarette smoking is the risk factor most consistently associated with pancreatic cancer. In our study, a history of cigarette smoking was available for eight of nine pancreatic cancer cases. Seven of the eight cases were current or former smokers. Secondly, the observation of an increase in pancreatic cancer primarily among men, but not women, suggests the influence of occupational or lifestyle risk factors. If environmental exposure to asbestos via drinking water was responsible, elevations in pancreatic cancer among

both males and females might be expected. Also, due to the anatomic location of the pancreas and the usually poor prognosis at the time of diagnosis, the proportion of pancreatic cancers confirmed histologically is less than that for any other major cancer (Anderson et al., 1996). Problems with ascertainment and misclassification are therefore a concern in epidemiologic studies of pancreatic cancer. Lastly, a chance positive finding is possible, particularly when examining 14 sex-specific estimates.

Duration of residence on the water supply was used as a surrogate for cumulative exposure to asbestos in the current study in an effort to assess dose-response. Cancer incidence did not significantly increase with longer duration of residence. Uncertainty about timing of first exposure as well as small numbers of observations by category of duration limited our ability to more accurately and precisely examine the influence of duration of residence on the water supply.

Since the latency period for asbestos-related cancers can be 20-30 years or more (Mossman and Gee, 1989), cancers observed in the early years following exposure to asbestos in drinking water might not be attributable to this exposure. A lagged analysis was conducted, in which individuals were not considered at risk and were not counted in the follow-up for an interval following first exposure. When a causal relationship exists and an appropriate lag period is used, a higher cancer incidence rate would be expected in lagged analyses since the estimated rates would not be diluted by observation time for individuals not yet at risk of exposure-related cancer. After allowing for latency, significantly higher cancer rates were not observed in the current study for gastrointestinal cancers, respiratory cancers, or all cancer sites combined. Uncertainty about timing of first exposure restricts our interpretation of lagged analyses. The longest interval between first exposure and the end of follow-up in 1998 was 39 years based on exposure starting in 1960 but would be only 23 years if exposure started in 1976. In addition, the small number of WAER participants with long latency limited statistical power of estimates stratified by latency.

Although the number of individual gastrointestinal subsite cases was too small to permit formal analysis of duration and latency, pancreatic cases were examined in more detail to see if duration and latency were consistent with exposure to asbestos in the water supply. Among the nine pancreatic cancer cases in our cohort, duration of residence on the Woodstock water supply was less than ten years for five pancreatic cancer cases, 10 to <20 years for two cases, and 20 years or more for two cases. Latency between start of residence on the Woodstock water supply and cancer diagnosis was less than 10 years for three pancreatic cancer cases, 10-<20 years for two cases, and 20 years or more for four cases. It is unlikely that cancers diagnosed within ten years of first exposure would be related to asbestos exposure.

An important limitation of this study is the lack of historical exposure data needed to establish when leaching of asbestos into the water supply first produced measurable exposure. The AC pipes were installed in the mid- to late-1950s, but asbestos contamination was first detected in 1985. A 10-year-old water sample that was tested in 1986 contained higher-than-normal levels of asbestos. This indicates that leaching had begun by 1976. The use of 1976 as the starting time of exposure produced similar results to the analysis based on a 1960 start date.

Other than the one water sample believed to have been collected in 1976, we have no evidence regarding the extent of the asbestos contamination in the Woodstock water supply prior to 1985, nor can we estimate changes in exposure over time or by location on the water supply. The water sampling that was performed in November 1985 occurred after the water mains were flushed (an attempt to remedy the water pressure problem). Deterioration of the pipe was probably aggravated by the forceful flow of water, both from turning the water on after it was turned off for repairs and from flushing the water mains. The asbestos levels measured may not have represented usual concentrations. Gradual leaching of asbestos may have resulted in generally low concentrations of asbestos. Due to limited information on the actual levels of exposure, this study's findings regarding risk associated with asbestos exposure cannot be generalized to other exposure experiences. The exposures experienced by the WAER cohort do not appear to have led to a detectable increased risk of cancer.

We could not directly control for potential confounding due to risk factors such as smoking, alcohol use, and socioeconomic status in our SIR estimates. However, we examined registry, survey, and census data to determine whether the WAER cohort was comparable to the population of NYS excluding NYC with respect to these risk factors. The frequency of cigarette smoking and alcohol use among the WAER participants did not differ substantially from survey estimates for the population of NYS excluding NYC. Education and income information were not available through the registry so census data were used to assess socioeconomic status. Based on 1990 U.S. Bureau of Census data, the Town of Woodstock population had a higher level of education than the population of NYS excluding NYC. Thirty-nine percent of Town of Woodstock residents had a bachelor's

degree or higher, compared to 23% of the population of Upstate NY excluding NYC. Stomach and lung cancer are associated with low socioeconomic status and colorectal cancer is associated with high socioeconomic status (Nomura, 1996), (Blot and Fraumeni, 1996), (Schottenfeld and Winawer, 1996). The greater proportion of residents with advanced education, indicative of higher socioeconomic status, could explain the somewhat lower than expected incidence of respiratory cancer among males in the study cohort. We did not have information on other risk factors for stomach and colon cancers such as diet, physical activity, and body mass (Nomura, 1996); (Tomeo et al., 1999).

Selection bias in cohort studies most often results from low and differential follow-up of the study population. This is unlikely in the current study, since only 2.2% of the study cohort was lost to follow-up. Selection bias due to differential participation at the beginning of the study is less of a concern when participant recruitment precedes disease diagnosis, as was the case for most registrants. It nevertheless is possible that individuals in the WAER differed from all eligible residents who lived on the Woodstock water supply. If registrants were generally more "health conscious" than non-participants, our estimates may be biased, possibly explaining the somewhat low incidence of gastrointestinal and respiratory cancers compared to NYS excluding NYC. However, it is unlikely that the bias would have been sufficient to mask a moderate positive association since only about 20% of eligible single family residences in 1985 were not represented in the WAER.

The study power for the cancer categories of interest was adequate to detect a modest increase in cancer risk. We had 80% power to identify a 50% increase in gastrointestinal cancer (SMR=1.50) and nearly 100% power to detect a doubling in the risk of gastrointestinal cancer (SMR=2.00). For respiratory cancer, the study power was 74% to identify a 50% increase in cancer incidence (SMR=1.50) and 99% to detect a doubling in risk (SMR=2.00). However, the study power for certain gastrointestinal subsites was relatively low. For example, power to identify an SMR of 2.00 or more for stomach cancer was only 43%.

Strengths of this study are that individual information was obtained from personal interviews of the participants or members of their household. The follow-up rate was high, with 59 persons lost to follow-up from 2,623 persons in the study cohort (2.2%). All the persons in the study population lived in homes that received water from the public water supply, unlike other studies in which exposed populations were defined by geographic boundaries. We were able to confirm incident cancers among individuals living in NYS using the NYS Cancer Registry. Analyses of the whole cohort, which included self-reported cancers among individuals who moved out-of-state, were similar to analyses based on confirmed cases only.

Conclusions

Previous studies of the cancer risks of asbestos in drinking water have been equivocal, with some showing a positive association and others not. This study is consistent with the latter in that individuals in this cohort study did not experience an increased incidence of respiratory cancer, gastrointestinal cancer, or all cancers combined. A significantly increased risk of pancreatic cancer was observed among males but not females. This association may be related to factors other than asbestos exposure such as cigarette smoking or chance.

Relative to earlier studies, this investigation had the advantage of a prospective cohort design, considerable latency, and the inclusion only of persons who lived on the public water supply. There was no increase in incidence by latency or duration of residence on the water supply, but the ability to detect these trends is limited by small numbers and unknown dates of initial exposure. This is the final report for this study; follow-up of the Woodstock Asbestos Exposure Registry has ended.

Preventive public health policy suggests that new AC pipes should not be installed to carry water, especially if the water is corrosive. Also, water supply systems using existing AC pipes should be monitored, especially in areas where the water has corrosive properties, and replaced if necessary.

References Cited

1. Andersen, A., Glatte, E., and Johansen, B.V., 1993. Incidence of cancer among lighthouse keepers exposed to asbestos in drinking water. *Am. J. Epidemiol.* 138 (9), 682-687.
2. Anderson, K.E., Potter, J.D., and Mack, T.M. (1996). Pancreatic cancer. In *Cancer epidemiology and prevention*, D. Schottenfeld and J.F. Fraumeni, Jr., eds. (New York: Oxford University Press), pp. 725-771.

3. Blot, W.J. and Fraumeni, J.F. (1996). Cancers of the lung and pleura. In *Cancer epidemiology and prevention*, D. Schottenfeld and J.F. Fraumeni, eds. (New York: Oxford University Press), pp. 637-665.
4. Chouroulinkov, I., 1989. Experimental studies on ingested fibres. *IARC Sci. Publ.* (90), 112-126.
5. Conforti, P.M., Kanarek, M.S., Jackson, L.A., Cooper, R.C., and Murchio, J.C., 1981. Asbestos in drinking water and cancer in the San Francisco Bay Area: 1969-1974 incidence. *J. Chronic. Dis.* 34 (5), 211-224.
6. Enterline, P.E., Hartley, J., and Henderson, V., 1987. Asbestos and cancer: a cohort followed up to death. *Br. J Ind. Med.* 44 (6), 396-401.
7. Frumkin, H. and Berlin, J., 1988. Asbestos exposure and gastrointestinal malignancy review and meta-analysis. *Am. J. Ind. Med.* 14 (1), 79-95.
8. Gamble, J.F., 1994. Asbestos and colon cancer: a weight-of-the-evidence review. *Environ. Health Perspect.* 102 (12), 1038-1050.
9. Goodman, M., Morgan, R.W., Ray, R., Malloy, C.D., and Zhao, K., 1999. Cancer in asbestos-exposed occupational cohorts: a meta-analysis. *Cancer Causes Control* 10 (5), 453-465.
10. Howe, H.L., Wolfgang, P.E., Burnett, W.S., Nasca, P.C., and Youngblood, L., 1989. Cancer incidence following exposure to drinking water with asbestos leachate. *Public Health Rep.* 104 (3), 251-256.
11. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. IARC monographs on the evaluation of the carcinogenic risk of chemicals to man: asbestos. *Suppl. 7[14]*, 106-116. 1987.
Ref Type: Serial (Book,Monograph)
12. Kelsey, J.L., Thompson, W.D., and Evans, A.S. (1996). *Methods in observational epidemiology*. (New York: Oxford University Press), pp. 167-178.
13. Levy, B.S., Sigurdson, E., Mandel, J., Laudon, E., and Pearson, J., 1976. Investigating possible effects of asbestos in city water: surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. *Am. J. Epidemiol.* 103 (4), 362-368.
14. Masson, T.J., McKay, F.W., and Miller, R.W., 1974. Asbestos-like fibers in Duluth water supply. Relation to cancer mortality. *JAMA* 228 (8), 1019-1020.
15. Meigs, J.W., Walter, S.D., Heston, J.F., Millette, J.R., Craun, G.F., Woodhull, R.S., and Flannery, J.T., 1980. Asbestos cement pipe and cancer in Connecticut 1955-1974. *J Environ Health* 42 (4), 187-191.
16. Millette, J.R., Craun, G.F., Stober, J.A., Kraemer, D.F., Tousignant, H.G., Hildago, E., Duboise, R.L., and Benedict, J., 1983. Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. *Environ. Health Perspect.* 53, 91-98.
17. Mossman, B.T. and Gee, J.B., 1989. asbestos-related diseases. *N. Engl. J Med.* 320 (26), 1721-1730.
18. Nomura, A. (1996). Stomach cancer. In *Cancer epidemiology and prevention*, D. Schottenfeld and J.F. Fraumeni, eds. (New York: Oxford University Press), pp. 707-724.
19. Peto, J. (1989). Fibre carcinogenesis and environmental hazards. In *Non-Occupational Exposure to Mineral Fibres*, J. Bignon, J. Peto, and R. Saracci, eds. (Lyon: International Agency for Research on Cancer), pp. 457-470.
20. Peto, J., Doll, R., Hermon, C., Binns, W., Clayton, R., and Goffe, T., 1985. Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. *Ann. Occup. Hyg.* 29 (3), 305-355.
21. Polissar, L., Severson, R.K., and Boatman, E.S., 1984. A case-control study of asbestos in drinking water and cancer risk. *Am. J. Epidemiol.* 119 (3), 456-471.
22. Schottenfeld, D. and Winawer, S.J. (1996). Cancers of the large intestine. In *Cancer epidemiology and prevention*, D. Schottenfeld and J.F. Fraumeni, eds. (New York: Oxford University Press), pp. 813-840.
23. Selikoff, I.J., 1965. Relation between exposure to asbestos and mesothelioma. *N. Engl. J Med.* 272, 560-565.
24. Selikoff, I.J., Churg, J., and Hammond, E.C., 1964. Asbestos exposure and neoplasia. *JAMA* 188, 2-226.

25. Sigurdson, E.E., 1983. Observations of cancer incidence surveillance in Duluth, Minnesota. Environ. Health Perspect. 53, 61-67.
26. Toft, P., Wigle, D., Meranger, J.C., and Mao, Y., 1981. Asbestos and drinking water in Canada. Sci. Total Environ. 18, 77-89.
27. Tomeo, C.A., Colditz, G.A., Willett, W.C., Giovannucci, E., Platz, E., Rockhill, B., Dart, H., and Hunter, D.J., 1999. Harvard Report on Cancer Prevention. Volume 3: prevention of colon cancer in the United States. Cancer Causes Control 10 (3), 167-180.
28. USEPA. Asbestos (CASRN 1332-21-4). Integrated Risk Information System . 7-1-1993. Ref Type: Electronic Citation
29. Varga, C., Horvath, G., and Timbrell, V., 1999. On the mechanism of cogenotoxic action between ingested amphibole asbestos fibres and benzo[a]pyrene: II. Tissue specificity studies using comet assay. Cancer Lett. 139 (2), 173-176.
30. Wigle, D.T., 1977. Cancer mortality in relation to asbestos in municipal water supplies. Arch. Environ. Health 32 (4), 185-190.

Table 1. Epidemiologic studies of cancer occurrence and exposure to asbestos in drinking water.

Author, date	Exposed population	Comparison population	Study design	Asbestos source	Outcomes measured	Positive associations*
Meigs et al. 1980	Higher exposure townships, Connecticut	Lower exposure townships, Connecticut	Ecologic	Asbestos cement (AC) pipes	Cancer incidence	Pancreatic cancer (males 1955-1964, not 1965-1974)
Conforti et al. 1981	Higher exposure census tracts, Bay Area, CA	Higher exposure census tracts, Bay Area, CA	Ecologic	Natural	Cancer incidence	×
Toft et al. 1981	Two cities with high asbestos levels, Canada	52 cities with low asbestos levels	Ecologic	Natural and AC pipes	Gastrointestinal cancer mortality	Large intestine (males) Stomach (males)
Millette et al. 1983	Census tracts with AC pipes (high/low % served by AC pipes) Escambia County, FL	Census tracts with no AC pipe use	Ecologic	AC pipes	Cancer mortality	---
Sigurdson 1983	Duluth, MN	Minneapolis/St. Paul and Iowa (later years)	Ecologic	Mine tailing wastes	Cancer incidence	Peritoneum (males, 1969-1971 only) Stomach (males 1979-1980 only)
Polissar et al. 1984	Exposure based on residential history, sampling, Puget Sound Area, WA	---	Case-control	Natural	Cancer incidence	Stomach (males) Pharynx (males)
Howe et	Town of	New York State	Ecologic	AC pipes	Cancer	

al. 1989	Woodstock, NY	excluding New York City			incidence	
Andersen et al. 1993	Lighthouse keepers (males), Norway	Rural population of Norway	Retrospective cohort	Rainwater run-off, AC roof tiles	Cancer incidence	Stomach (males only in study)

* Statistically significant associations for gastrointestinal or respiratory cancers are listed.

×The large study population permitted power to detect relative risks as low as 1.1 for all gastrointestinal cancers combined (Erdreich, 1983). Numerous statistically significant correlation and regression coefficients and trend tests were reported. Among males all three tests were significant for all sites, all digestive sites, esophagus, stomach, and colon; significant results on one or two of the statistical tests were observed for cancers of the pancreas, trachea/lung, and mediastinum. Among females all three tests were significant for all digestive sites, esophagus, and retroperitoneum; significant results on one or two of the statistical tests were observed for cancers of the stomach, colon, rectum, gall bladder, pancreas, trachea/lung, and pleura.

Table 2. Woodstock Asbestos Exposure Registry cohort: exclusions and follow-up status.

Participants in Woodstock Asbestos Exposure Registry 2936	
Exclusions from study cohort (follow-up 1980-1998)	
Deaths prior to 1980	217
Cancer diagnosed prior to 1980	30
Unknown date of birth	52
Unknown duration of residence	14
Study cohort	2623
Status of cohort in 1998	
Lost to follow-up	59
Moved out of state	712
Remained in NYS	1852

Table 3. Study cohort characteristics, Woodstock Asbestos Exposure Registry.

Characteristic	Number	Percent
Gender		
Male	1212	46.2
Female	1411	53.8
Age at recruitment*		
<45	1602	64.4
45-54	353	14.2
55-64	243	9.8
65-74	158	6.4
75-84	96	3.9
85+	34	1.4
Race		
White	1953	96.9
Black	27	1.3

Other	36	1.8
Unknown	607	x
Smoking history^l		
Never smoker	1067	47.9
Ever smoker	1159	52.1
Unknown	83	x
Alcoholic drinks per week^l		
Less than one	999	50.2
1-6	621	31.2
7-13	218	11.0
14+	150	7.5
Unknown	321	x
Relationship to respondent[§]		
Self	949	36.3
Spouse	443	16.9
Child	817	31.2
Sibling	69	2.6
Parent	103	3.9
Extended family member	110	4.2
Other	126	4.8
Unknown	6	x

* Age is not listed for 137 persons included in the WAER who were deceased at the time a relative provided registration information.

x Unknown values are not included in percents.

^l Smoking and alcohol use among individuals 18 years and older at registration.

[§] Respondent refers to the person who completed the questionnaire.

Table 4. Smoking and alcohol use, Woodstock cohort members 18 years and older at registration, registered 1986-1988, compared to Behavioral Risk Factor Surveillance System (BRFSS) estimates for white residents of NYS excluding NYC.

Age at registration	Males (percent)		Females (percent)	
	Woodstock*	BRFSS	Woodstock*	BRFSS
Ever smoker ^x				
18-24	25.2	47.9	31.0	28.7
25-34	40.3	56.5	46.4	56.8
35-44	60.7	66.4	56.7	57.6
45-54	71.3	60.0	61.4	65.0
55-64	67.9	68.5	59.3	44.1
65+	60.0	70.7	48.0	37.2
Chronic drinking ^l				
18-24	2.9	22.7	0.0	3.4
25-34	11.2	9.9	4.4	1.2
35-44	8.7	8.9	3.4	2.5
45-54	12.4	14.9	6.1	0.5

55-64	17.7	9.4	6.7	1.3
65+	11.6	5.5	2.9	2.6

*Following exclusions (less than 18 years of age, not interviewed in 1986-1988, unknown smoking history or drinking habits), 1586 and 1408 participants were included in the smoking and drinking analyses, respectively.

*A "yes" response to "Have you smoked at least 100 cigarettes in your life?" was used to identify ever smokers using BRFSS data.

†A report of two or more alcoholic drinks per day was classified as "chronic drinking" to be consistent with the categorization scheme used by the BRFSS.

Table 5. Standardized incidence ratios (SIRs) and 95% confidence intervals for cancers of selected sites for cohort participants who were New York State residents during follow-up and lived on the Woodstock water supply between 1960-1985.

Cancer site	Gender	Observed	Expected*	SIR*	95% CI †
Total cancers					
	Male	66	72.79	0.91	0.70-1.15
	Female	78	81.00	0.96	0.76-1.20
	Total	144	153.8	0.94	0.79-1.10
<i>Total gastrointestinal</i>					
	Male	17	16.66	1.02	0.59-1.63
	Female	12	16.67	0.72	0.37-1.26
	Total	29	33.33	0.87	0.58-1.25
<i>Gastrointestinal subsites</i>					
Esophagus					
	Male	3	1.03	2.91	0.60-8.51
	Female	0	0.45	0.00	--
	Total	3	1.48	2.03	0.42-5.93
Stomach					
	Male	2	1.88	1.06	0.13-3.84
	Female	0	1.24	0.00	--
	Total	2	3.12	0.64	0.08-2.31
Pancreas					
	Male	6	1.95	3.08	1.13-6.70
	Female	3	2.16	1.39	0.29-4.06
	Total	9	4.11	2.19	1.00-4.16
Colorectal					
	Male	5	10.31	0.48	0.16-1.13
	Female	9	11.15	0.81	0.37-1.53
	Total	14	21.47	0.65	0.36-1.09
<i>Respiratory</i>					
	Male	10	15.31	0.65	0.31-1.20
	Female	11	10.44	1.05	0.53-1.88
	Total	21	25.75	0.82	0.50-1.25

*Expected number is based on the Woodstock cohort population and cancer rates for residents of NYS excluding NYC.

* SIR = observed/expected.

‡ Poisson exact confidence interval.

Table 6. Standardized incidence ratios (SIRs) and 95% confidence intervals for cancers of selected sites for cohort participants who were New York State residents during follow-up and lived on the Woodstock water supply between 1960-1985, by duration of residence on the Woodstock water supply.

Cancer site	Gender	Observed	Expected*	SIR*	95% CI ‡
<i>Gastrointestinal</i>					
Duration of residence < 5 yrs.					
	Male	5	4.67	1.07	0.35-2.50
	Female	4	4.77	0.84	0.23-2.15
	Total	9	9.43	0.95	0.44-1.81
Duration of residence 5-<15 yrs.					
	Male	5	5.29	0.95	0.31-2.21
	Female	5	5.29	0.94	0.31-2.20
	Total	10	10.58	0.95	0.45-1.74
Duration of residence 15+ yrs.					
	Male	7	6.74	1.04	0.42-2.14
	Female	3	6.61	0.45	0.09-1.33
	Total	10	13.35	0.75	0.36-1.38
<i>Respiratory</i>					
Duration of residence < 5 yrs.					
	Male	2	4.61	0.43	0.05-1.57
	Female	3	3.37	0.89	0.18-2.60
	Total	5	7.98	0.63	0.20-1.46
Duration of residence 5-<15 yrs.					
	Male	3	4.80	0.63	0.13-1.83
	Female	5	3.57	1.40	0.46-3.27
	Total	8	8.37	0.96	0.41-1.88
Duration of residence 15+ yrs.					
	Male	5	5.90	0.85	0.28-1.98
	Female	3	3.51	0.86	0.18-2.50
	Total	8	9.41	0.85	0.37-1.68

*Expected number is based on the Woodstock cohort population and cancer rates for residents of NYS excluding NYC.

× SIR = observed/expected.

‡ Poisson exact confidence interval.

Table 7. Standardized incidence ratios (SIRs) and 95% confidence intervals for cancers of selected sites for cohort participants who were New York State residents during follow-up and lived on the Woodstock water supply between 1960-1985, by increasing lag periods.

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Cancer site	Gender	Observed	Expected*	SIR*	95% CI [†]
<i>Gastrointestinal</i>					
No lag					
	Male	17	16.66	1.02	0.59-1.63
	Female	12	16.67	0.72	0.37-1.26
	Total	29	33.33	0.87	0.58-1.25
5-year lag					
	Male	17	15.83	1.07	0.63-1.72
	Female	8	15.38	0.52	0.22-1.02
	Total	25	31.20	0.80	0.52-1.18
10-year lag					
	Male	13	14.30	0.91	0.48-1.55
	Female	6	13.62	0.44	0.16-0.96
	Total	19	27.92	0.68	0.41-1.06
20-year lag					
	Male	8	9.54	0.84	0.36-1.65
	Female	4	8.77	0.46	0.12-1.17
	Total	12	18.32	0.66	0.34-1.14
<i>Respiratory</i>					
No lag					
	Male	10	15.31	0.65	0.31-1.20
	Female	11	10.44	1.05	0.53-1.88
	Total	21	25.75	0.82	0.50-1.25
5-year lag					
	Male	10	14.53	0.69	0.33-1.27
	Female	11	9.81	1.12	0.56-2.01
	Total	21	24.34	0.86	0.53-1.32
10-year lag					
	Male	8	13.16	0.61	0.26-1.20
	Female	10	8.79	1.14	0.55-2.09
	Total	18	21.94	0.82	0.49-1.30
20-year lag					
	Male	6	8.78	0.68	0.25-1.49
	Female	7	5.69	1.23	0.49-2.53
	Total	13	14.48	0.90	0.48-1.54

*Expected number is based on the Woodstock cohort population and cancer rates for residents of NYS excluding NYC.

* SIR = observed/expected.

† Poisson exact confidence interval.

Table 8. Standardized incidence ratios (SIRs) and 95% confidence intervals for cancers of selected sites for cohort participants who were New York State residents during follow-up and lived on the Woodstock water supply between 1976-1985.

Cancer site	Gender	Observed	Expected*	SIR*	95% CI [†]
Total cancers					
	Male	56	53.33	1.05	0.79-1.36
	Female	63	61.39	1.03	0.79-1.31
	Total	119	114.72	1.04	0.86-1.24
<i>Total gastrointestinal</i>					
	Male	14	12.31	1.14	0.62-1.91
	Female	10	13.02	0.77	0.37-1.41
	Total	24	25.33	0.95	0.61-1.41
<i>Gastrointestinal subsites</i>					
Esophagus					
	Male	3	0.74	4.06	0.84-11.87
	Female	0	0.35	0.00	--
	Total	3	1.08	2.77	0.57-8.09
Stomach					
	Male	2	1.39	1.44	0.17-5.20
	Female	0	0.98	0.00	--
	Total	2	2.37	0.84	0.10-3.05
Pancreas					
	Male	4	1.43	2.80	0.76-7.17
	Female	3	1.68	1.78	0.37-5.21
	Total	7	3.11	2.25	0.90-4.63
Colorectal					
	Male	5	7.64	0.65	0.21-1.53
	Female	7	8.71	0.80	0.32-1.65
	Total	12	16.36	0.73	0.38-1.28
<i>Respiratory</i>					
	Male	8	11.06	0.72	0.31-1.42
	Female	6	7.77	0.77	0.28-1.68
	Total	14	18.83	0.74	0.41-1.25

*Expected number is based on the Woodstock cohort population and cancer rates for residents of NYS excluding NYC.

* SIR = observed/expected.

† Poisson exact confidence interval.

Table 9. Standardized incidence ratios (SIRs) and 95% confidence intervals for cancers of selected sites for the whole cohort.

Cancer site	Gender	Observed	Expected*	SIR*	95% CI [†]
Total cancers					
	Male	92	95.62	0.96	0.78-1.18
	Female	98	103.8	0.94	0.77-1.15
	Total	190	199.5	0.95	0.82-1.10
<i>Total gastrointestinal</i>					
	Male	22	21.88	1.01	0.63-1.52
	Female	17	21.17	0.80	0.47-1.29
	Total	39	43.05	0.91	0.64-1.24
<i>Gastrointestinal subsites</i>					
Esophagus					
	Male	4	1.34	2.98	0.81-7.62
	Female	0	0.57	0.00	--
	Total	4	1.91	2.09	0.57-5.36
Stomach					
	Male	3	2.47	1.21	0.25-3.55
	Female	1	1.58	0.63	0.02-3.53
	Total	4	4.05	0.99	0.27-2.53
Pancreas					
	Male	6	2.55	2.35	0.86-5.11
	Female	3	2.74	1.10	0.23-3.20
	Total	9	5.29	1.70	0.78-3.23
Colorectal					
	Male	6	13.52	0.44	0.16-0.97
	Female	11	14.16	0.78	0.39-1.39
	Total	17	27.67	0.61	0.36-0.98
<i>Respiratory</i>					
	Male	12	20.01	0.60	0.31-1.05
	Female	12	13.21	0.91	0.47-1.59
	Total	24	33.21	0.72	0.46-1.08

*Expected number is based on the Woodstock cohort population and cancer rates for residents of NYS excluding NYC.

× SIR = observed/expected.

† Poisson exact confidence interval.