

Cancer Risk from Asbestos in Drinking Water: Summary of a Case-Control Study in Western Washington

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We conducted a case-control, interview-based study of the risk of developing cancer from asbestos in drinking water. An area that included Everett, Washington, was selected for the study because of the unusually high concentration of chrysotile asbestos in drinking water from the Sultan River.

Through a population-based tumor registry, 382 individuals with cancer of the buccal cavity, pharynx, respiratory system, digestive system, bladder, or kidneys, diagnosed between 1977 and 1980, were identified, and they or their next of kin were interviewed. We conducted validation checks of our interviews, including a comparison with secondary sources.

Data on asbestos exposure were collected based on residence and workplace history, and on individual water consumption. Logistic regression was used to estimate cancer risk. We found no convincing evidence for increased cancer risk from imbibed asbestos. Confidence intervals for relative risks for almost all sites included unity. There were significantly elevated risks only for male stomach and male pharyngeal cancer, but these sex-inconsistent results, based on small numbers of cases, are probably due to other factors.

Our interest in conducting a study of cancer incidence and waterborne asbestos was stimulated, as was that of previous researchers, by the known carcinogenic effect of inhaled asbestos (1,2) and the discovery of asbestos fibers in a number of public water supplies (3-5).

All previous studies of the potential carcinogenic effect of imbibed asbestos, however, have been ecologic ones (6-13). In almost all of the previous studies, asbestos exposure was imputed from residence at the particular moment of the study, with no measurement of the duration or degree of individual exposure, or the pattern of individual migration.

In contrast, our study, which is described more fully elsewhere (14), determined individual exposure through in-person interviews with cancer

cases or their next of kin and with members of a control group.

Materials and Methods

Study Area

We chose cases and controls from the Everett, WA, area, which has used the Sultan River as a source of drinking water since 1918. Sultan River tapwater has concentrations of chrysotile asbestos around 200 million fibers/L, among the highest in the United States (5,15).

Since a large volume of migration into the study area could produce a population with small cumulative exposures, we limited our study area to 1970 census tracts with lower than average migration rates.

Cases

Data on incident cancer cases were obtained through the Cancer Surveillance System (CSS), a population-based tumor registry that covers 13

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counties and a population of nearly 3 million in western Washington. The CSS is part of the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute (16).

All invasive or *in situ* cases of cancer of the buccal cavity (excluding the lip), pharynx, respiratory system, digestive system, bladder, or kidney newly diagnosed between November 1977 and December 1980 were identified. The sites were chosen primarily on the basis of preceding studies, particularly the reported positive findings of Kanarek et al. (12), sites previously implicated in studies of airborne asbestos, sites that might be involved in elimination of asbestos fibers, and sites along the alimentary tract.

Only those individuals between 40 and 79 yr of age who resided in the eligible census tracts at the time of diagnosis were included in this study; both living and deceased were included.

Controls

We chose unmatched population controls of the same age range (40–79) and from the same group of 25 census tracts as the cases, using standard area sampling methods. We attempted to schedule an interview with each person of eligible age in 649 households selected.

Questionnaire

In-person interviews with subjects or next of kin focused on residence and workplace history, and the amount of water consumed by each individual. Information on other risk factors was also collected.

Quality Control Procedures

Quality control procedures included interview validation, independent recoding, and comparison of interview information with secondary sources. Since extensive concern has been expressed over the use of surrogate interviews for deceased cases (17), we compared a sample of subjects' residence and occupation histories with listings in independent, annually published Everett City Directories (18) from 1930 to 1980.

Exposure to Imbided Asbestos

By meeting with representatives of water companies, we learned the history of water district boundaries and could determine the source of tapwater (and the asbestos concentration) for any given location and date.

We then calculated four separate variables expressing cumulative exposure to imbided asbestos

for each subject. The calculations were based on each subject's residence and workplace history and on typical water consumption from all sources "five years ago." The first variable was based only on residence history and workplace history. The second variable was obtained by multiplying the first variable by the total amount of water intake as determined from the water consumption questions.

The second pair of variables was calculated in a similar fashion, except that we ignored all residence and work locations during a presumed 10-yr latent period prior to diagnosis or interview.

Statistical Methods

We estimated cancer risk by fitting a logistic regression model (19). The dependent variable was a dichotomous indicator of a specific cancer site versus control status. Predictor variables always included age and cumulative exposure to asbestos in water. Other covariates included well-established risk factors such as smoking. For each site, each sex was analyzed separately except for a few sites in which small numbers required a joint analysis. In such cases, sex was included as a covariate. Cross tabulation and other methods were used to check the logistic regression results.

We also estimated the statistical power of the logistic regression method to detect cancer risk.

Results

Interviewing Completion Rates

Of the total 445 eligible cases, the overall refusal rate was 13.5%. Of the total 549 eligible controls, the refusal rate was 11.7%.

After the interview and before analysis, we limited the study to white subjects, since we found only one nonwhite subject. Two additional interviews with the next of kin of subjects were also excluded because virtually all answers were "unknown." The final roster for analysis was 382 cases and 462 controls.

Quality Control Checks

Validation checks by supervisors showed that the data collected by the interviewers were highly reliable. A comparison of coding and independent recoding for a sample of subjects turned up an average of only one disagreement per lengthy questionnaire. Comparison of residence and employer histories with information from annual city directories also showed low disagreement rates that were similar across interviews with

living cases, next of kin of deceased cases, and controls.

Exposure Levels

The amount of exposure to chrysotile asbestos in drinking water was extremely similar between cases and controls. For example, 23% of the cases and 21% of the controls had 30 yr or more of exposure to Sultan River drinking water. If a 10-yr latent period is taken into account, exposure is still similar. The amount of water intake per week was also very similar between cases and controls.

Relative Risk

The validity of the data collected and the logistic regression analysis are supported by reconfirmation of other known risk factors. The logistic regression model, for example, produces coefficients that are generally positive for age (indicating increasing cancer risk with age) and smoking, and are in the right direction for other risk factors.

Summarizing our findings for imbibed asbestos, we found very few elevated risks of statistical significance. Considering the relative risk for each of the sites and for each of the four asbestos exposure variables, we found no instance in which the risk was elevated for both males and females. The only statistically significant ($p < 0.05$) elevated risks occurred for male pharynx and male stomach. The observed number of "significant" results is not surprising considering the number of comparisons made. As a summary measure, we calculated the relative risk from the logistic regression model for a 20-yr cumulative use of the Sultan River tapwater versus no exposure. On this basis, the male pharynx relative risk (RR) was 2.99 (lower 95% confidence bound = 1.43) with RR = 0.26 for females. The male stomach relative risk was 1.71 (lower 95% confidence bound = 1.06) with RR = 0.65 for females. These estimated risks were similar for each of the four asbestos exposure variables. For males, the stomach cancer risks are based on eight cases, and the pharyngeal cancer risks are based on four cases.

The pancreas, a site that we previously noted was most consistently implicated by the various ecologic studies (13), appears in our study with a sex-inconsistent negative risk for males and a positive risk for females. None of the risks was statistically significant at the 5% level.

Sometimes risks become evident only at very high levels of exposure. To test this, we calculated

relative risks for persons with 30 yr or more of Sultan River exposure and compared them with the risks for persons with 5 yr or fewer of exposure. This analysis included only the digestive system of males and females and all study sites grouped for both sexes. The calculated risks were nonsignificant and were similar to those based on all exposure.

Power Considerations

For single-sex analyses, the minimum risk that could be detected at the 5% significance level with 80% probability was under 2.0 for each sex for the following sites or site groups: all study sites combined, digestive system, respiratory system, colon and lung.

Discussion

Some limitations of the study are worth noting. We lack numbers of cases to make really solid judgment about risk for the rarer sites, such as the kidneys and gallbladder. It does appear, however, that the risk is unlikely to be large for any of the sites we have studied, due to low calculated risks or to sex-inconsistent results.

Another limitation is the effect of a possible long latent period. The latent period for inhaled asbestos appears to be about 30 yr (20). Exposure prior to the latent period would increase the power of the study. We note that about 25% of the cases and controls had an onset of exposure beginning at least 40 yr ago, even though we chose the more stable census tracts in our area. The migration habits of U.S. residents generally result in smaller exposures to geographically fixed sources compared to such exposures in more stable populations.

Finally, the use of next of kin for deceased cases, plus a few proxies for the eligible controls who were unavailable for interview, may have introduced some bias unknown to us, despite our verification checks.

Balancing these limitations of the study, however, are a number of strengths. The exposures are individually based and appear to be accurately measured. The case and control refusal rates were low, and the cases, from a population-based tumor registry, are representative of all cases in a community that does not have atypical features. The controls, which had been carefully drawn from the same population that contributed the cases, were interviewed during the same period. The community chosen for study has a very high level of asbestos in water and has apparently had this level for at least 60 yr.

We would suggest that the next step in this line of research should be to discuss the feasibility and advisability of attempting to detect very low risks or to detect risks for rare sites. We propose that additional studies not be undertaken hastily, since the ability to detect the risks involved may not be available in current epidemiologic methods except at great effort or expense.

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