Cancer Occurrence in Shipyard Workers Exposed to Asbestos in Hawaii

Laurence N. Kolonel, Carl N. Yoshizawa, Tomio Hirohata, and Beth C. Myers

Epidemiology Program, Cancer Research Center, University of Hawaii, Honolulu, Hawaii 96813 [L. N. K., C. N. Y., B. C. M.], and Department of Public Health, School of Medicine, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka City, Japan [T. H.]

ABSTRACT

Because large numbers of persons were employed in United States shipyards during World War II, the long-term risks for cancer associated with asbestos exposure in this setting are of great concern. We report here on the mortality findings after up to 29 years of follow-up on a retrospective cohort of 7971 male Pearl Harbor Naval Shipyard workers, which included more than 3000 men whose employment period spanned the World War II years. Compared with the general population of Hawaii, workers in the shipyard cohort had no increase in total mortality or in total cancer mortality irrespective of the duration of their exposure. However, the risk ratio for lung cancer among workers with at least 15 years of asbestos exposure was 1.4 overall (95% confidence interval, 1.0 to 2.0) and 1.7 for those with a latency interval of 30 or more years (95% confidence interval, 1.0 to 2.5). In addition, seven mesotheliomas occurred between 1977 and 1982 in a subset of the cohort, consisting of 7029 Hawaii residents who are being followed prospectively for cancer incidence. This represented an incidence of 67.3 per million men per year, compared with a rate of 5.8 for the state as a whole. These results suggest that the long-term relative increase in risk for mesothelioma may be even greater than that for bronchogenic carcinoma in this and other cohorts of United States shipyard workers exposed to asbestos.

INTRODUCTION

The relationship of asbestos exposure to cancer risk has been the subject of much epidemiological research. Of special concern in recent years has been the extent to which workers in naval shipyards, in particular, are at increased risk. One reason for this interest is the very large size of shipyard work forces, particularly at certain periods in the past [an estimated 4.5 million United States men worked in shipyards during World War II, for example (20)]. We have been following a retrospective cohort of nearly 9000 shipyard workers at the PHNS. In an earlier analysis, we reported on the results of up to 24 years of follow-up, in which we showed a maximum relative risk for lung cancer of 1.7 in those workers with 20 to 24 years of follow-up (14). As we noted in that report, the full extent of the risk for lung or other cancers would not be known until the cohort could be followed for a longer period of time.

We now have an additional 5 years of follow-up on this cohort and report here the results of our extended analysis.

MATERIALS AND METHODS

In 1975, we reviewed the personnel rosters at the PHNS and assembled a cohort consisting of all male employees registered on January 1, 1950, or hired subsequently through December 31, 1969. Although it was not possible to establish a full roster of hires prior to 1950 (these records were no longer available), a large proportion of the workers on the rolls at the start of the study period had been employed during World War II or earlier. Indeed, as a proportion of the total cohort included in the final analyses for this report, they comprised a substantial proportion (39.4%). A few groups of employees were excluded, namely, workers whose only trade at PHNS was sandblasting, rubber working, or radiation work, since employment in these occupations in other settings has been associated with a significant increase in risk for pulmonary disease, including cancer. About 800 men were excluded initially for this reason, leaving a study group of 9570 men.

For each employee in the cohort, we recorded the date of hire, the trade, and all subsequent movements within the shipyard, i.e., dates of entry into or departure from specific trades. (Many workers shifted trades several times during their employment at PHNS.) In addition, each man's date of birth and any other useful data for surveillance were recorded.

It was necessary also to have racial information on the members of the cohort. Because race was not recorded in the personnel records in sufficient detail (only as "white," "black," "other"), we had to determine it by other means. Since place of birth was recorded, and most men in the problem category (i.e., "other") were born in Hawaii, we located birth certificates on these men and were able to assign a specific racial identity on this basis. For a residual 12% of the cohort, no racial information could be found, and for these men, we assigned race based on surnames.

Each of the many trades at the shipyard was classified as exposed or nonexposed with respect to asbestos. This classification was made with the help of occupational health personnel at PHNS after a thorough tour of the shipyard by one of us (B. C. M.). Exposed trades included those where workers handled asbestos directly or were in close association with other workers who used asbestos; nonexposed trades included those where workers had no, or at most minimal, likelihood of asbestos exposure in their work. A list of the trades identified with asbestos exposure is given in Table 1. Although a more precise measurement of exposure would have been desirable, no routine monitoring for air levels of asbestos was carried out during the period covered by the study.

Follow-up on the cohort made use of a number of different sources of information on vital status. These included local driver's license computer tapes, voter's registration lists, death record files for Hawaii, obituary columns in newspapers, records of the Social Security and Veterans Administrations, and personal contacts (by phone or mail). Vital status for each man was determined as of January 1, 1979, and death certificates were obtained for each man who had died, so that the underlying cause of death could be confirmed and recorded. Workers who were lost to follow-up were included in the analyses up to the time they were lost and were credited with observation into the calendar month prior to the one in which they were withdrawn.

Workers whose total employment at PHNS was less than 1 year were excluded from the analysis. Men who did not belong to one of the 5 main ethnic groups in Hawaii (Caucasians, Japanese, Hawaiians, Filipi...
The analysis was designed to assess the joint effects of duration of exposure and time since first exposure, or latency. The difference between latency and exposure duration is, by definition, the period of time since first exposure during which an individual was not exposed. A modified life-table method was used in which person-years of observation were accumulated for each exposure duration by latency subgroup of the cohort in 5-year age groups and 5-year calendar intervals for each of the 5 main ethnic groups. These data were then used to generate expected numbers of deaths based on the age-, sex-, race-, and calendar-specific mortality rates of Hawaii. Observed and expected numbers of the 5 main ethnic groups. These data were then used to generate the cohort in 5-year age groups and 5-year calendar intervals for each of these exposure duration by latency subgroup of the cohort. Results are shown separately for those men with no asbestos exposure, with 1 to 14 years of exposure, and with 15 or more years of exposure. Within each of these exposure categories, the data are shown for 3 latency intervals (0 to 19, 10 to 29, and 30 or more years). For nonexposed workers, latency refers to the interval since first hire. The results show that overall mortality was not increased among the subjects in this cohort, including those exposed to asbestos. Indeed, all of the risk ratios are less than unity, and all but 2 are statistically significant.

Table 4 shows a similar analysis for all cancers as a single group. None of the risk ratios in this table is significantly greater than 1.0. We also examined specific cancer sites. For lung cancer, the results are shown in Table 5. There are no significant elevations (or decreases) in risk for the nonexposed and shorter duration-of-exposure categories, even for the longest latency interval. For the group with the longest exposure, however, the risk ratios are all greater than 1.0, and for both the 30+ latency interval and overall, the risk ratios (1.7 and 1.4, respectively) are of borderline statistical significance at the 95% level (lower confidence limit, 1.0).

There were no significant increases for any other cancer sites, although the numbers of observed cases were very small in most instances. The results for the gastrointestinal tract (esophagus, stomach, colon, and rectum combined) are shown in Table 6. There is no pattern of increasing risk with duration of exposure, although for the longest latency interval in the shorter-exposed category, the SMR is significantly elevated (2.2; 95% CI, 1.1 to 4.0).

Only one mesothelioma case had occurred in the cohort as of January 1, 1979, the date of last follow-up for this analysis. Since that time, however, we have continued to follow a subset of the cohort, consisting of all subjects alive and residing in Hawaii as of January 1, 1977. Among this group of 7029 men, we have identified (by means of active surveillance, including confirmation of histological coding in the state-wide Hawaii Tumor Registry) 7 mesothelioma deaths (all pleural) during the 6-year interval from 1977 to 1982. All 7 of these occurred in the exposed group (as did the single case which occurred prior to this period), for an age-adjusted incidence rate in the total group of 67.3 per million men per year (1970 United States population standard). Of course, the computed rate would be even greater if the denominator were restricted to the exposed group of workers. This compares with an overall rate for the State of Hawaii of 5.8 per million men per year for the period of 1970 to 1975, using the same population standard (12). The mean interval from first exposure to death for the 8 mesothelioma cases was 31 years (median, 34 years), with a range of 13 to 41 years. The mean duration of exposure was 14.5 years, with a range of 1 to 29 years (Table 7).

DISCUSSION

This study had a number of advantages for assessing the asbestos-cancer relationship in shipyard workers. (a) The cohort size was large (7971 men in the final analysis), and the observation interval was long (up to 29 years, with more than 40 years...
since first employment at the shipyard for many men). (b) Detailed job histories were available, so that men could be classified into exposure groups within the shipyard; furthermore, men were only credited with exposure for their actual time spent in exposed trades if they transferred into or out of nonexposed trades. Thus, the observed risks in this study should more precisely assess the effects of asbestos exposure than would the more usual analysis in which all employees are considered as exposed. (c) Follow-up was very complete (97.1%). (d) All deaths were confirmed, and underlying causes were checked on death certificates. (e) For computation of expected numbers, ethnic group-specific mortality rates for Hawaii were used rather than more general rates for the total United States populations as is often the case.

The major limitations of the data were the lack of true measurements of actual exposure to asbestos and the inability to control for smoking. However, as we showed in our earlier report (14), cigarette smoking rates differed little between the occupational cohort and the general population (64% ever-smokers among the exposed workers, 63% among the nonexposed workers, and 59% in the general population, age and race adjusted). Total lifetime cigarette use (pack-years) among smokers showed even less variation among the 3 groups.

Unfortunately, ethnic group-specific analyses were infeasible due to the small numbers of observed and expected deaths in each ethnic group. More stable SMRs were obtained by "pooling together" the observed and expected numbers of deaths across the 5 ethnic groups. Although we have no reason to suspect that the effects of asbestos exposure would differ among ethnic groups, such differences, were they to exist, would affect our pooled SMRs, since these measures are weighted averages of the ethnic group-specific SMRs, with weights equal to the numbers of deaths expected in each ethnic group. We think this is quite unlikely, however.

The results of the analysis on total mortality showed no increase in the risk ratios, even in the exposed group with 15 or more years of exposure. Indeed, all risk ratios were less than...
For insulation workers in particular, the risk ratio of 2.2 for lung cancer based on a life-table analysis on a group of intestinal cancers. In Genoa, Puntoni et al. (18) found a risk ratio of 3.9 from onset of employment; there was no increase in gastrointestinal cancers. In Genoa, Puntoni et al. (18) found a risk ratio of 2.2 for lung cancer based on a life-table analysis on a group of 2190 shipyard workers. For insulation workers in particular, the risk ratio was 5.3. Beaumont and Weiss (3) found a risk ratio of 1.4 for lung cancer mortality in an analysis of a retrospective cohort of metal trade union workers in Seattle, WA, but they found no excess of deaths among the subgroups of workers employed exclusively in shipyards. Blot et al. (4), on the other hand, used a case-control approach. They found an odds ratio of 1.7 for lung cancer mortality associated with shipyard employment, after adjustment for race and smoking. In this study, there was no evidence for an effect of exposure duration, since men employed only during World War II (median of 4 years) had a similar risk to career shipyard workers (median of 29 years in the industry). This differs from the present study which found an increased risk for lung cancer only after a long latency period in men with at least 15 years of exposure. Overall, these studies suggest that, in both the United States and Europe, shipyard workers do have an increased risk for lung cancer, with risk ratios around 2.0. No doubt, exposures and risks vary among the many different trades within shipyards, as suggested by the higher risks for insulators (18, 24). Unfortunately, we were not able to separately analyze our cohort for specific trades because of the small observed and expected numbers of deaths.

The findings for mesothelioma in this study are more striking than those for lung cancer. Many studies have reported on the occurrence of mesotheliomas among shipyard workers (1, 11, 16, 19, 25, 26); one of these studies found no increase in lung cancer mortality (19). Since mesotheliomas have frequently been reported in persons with low or indirect exposure to asbestos, particularly the crocidolite type (5, 13), the general cancer pattern in shipyard workers (moderately increased risks for bronchogenic carcinoma and very high rates of mesothelioma) suggests that asbestos exposure levels may be lower or the fiber types in predominant use different in this setting from certain other occupations where the reported risks for bronchogenic carcinoma have been much higher (8, 21, 22). This may also explain why gastrointestinal cancers have not been clearly found to be increased in the present or other studies in shipyard workers (19, 23). Detailed records on the fiber types used at PHNS during the period of this study have not been available. However, chrysotile and amosite are thought to be the major ones, although the proportion of each of these types is not known.

Selikoff and Hammond (24) followed a cohort of 440 shipyard insulators in the United States and found a somewhat higher risk ratio of 3.9 for lung cancer after a minimal interval of 20 years from onset of employment; there was no increase in gastrointestinal cancers. In Genoa, Puntoni et al. (18) found a risk ratio of 2.2 for lung cancer based on a life-table analysis on a group of 2190 shipyard workers. For insulation workers in particular, the risk ratio was 5.3. Beaumont and Weiss (3) found a risk ratio of 1.4 for lung cancer mortality in an analysis of a retrospective cohort of metal trade union workers in Seattle, WA, but they found no excess of deaths among the subgroups of workers employed exclusively in shipyards. Blot et al. (4), on the other hand, used a case-control approach. They found an odds ratio of 1.7 for lung cancer mortality associated with shipyard employment, after adjustment for race and smoking. In this study, there was no evidence for an effect of exposure duration, since men employed only during World War II (median of 4 years) had a similar risk to career shipyard workers (median of 29 years in the industry). This differs from the present study which found an increased risk for lung cancer only after a long latency period in shipyard workers. For insulation workers in particular, the risk ratio of 2.2 for lung cancer based on a life-table analysis on a group of intestinal cancers. In Genoa, Puntoni et al. (18) found a risk ratio of 3.9 from onset of employment; there was no increase in gastrointestinal cancers. In Genoa, Puntoni et al. (18) found a risk ratio of 2.2 for lung cancer based on a life-table analysis on a group of 2190 shipyard workers. For insulation workers in particular, the risk ratio was 5.3. Beaumont and Weiss (3) found a risk ratio of 1.4 for lung cancer mortality in an analysis of a retrospective cohort of metal trade union workers in Seattle, WA, but they found no excess of deaths among the subgroups of workers employed exclusively in shipyards. Blot et al. (4), on the other hand, used a case-control approach. They found an odds ratio of 1.7 for lung cancer mortality associated with shipyard employment, after adjustment for race and smoking. In this study, there was no evidence for an effect of exposure duration, since men employed only during World War II (median of 4 years) had a similar risk to career shipyard workers (median of 29 years in the industry). This differs from the present study which found an increased risk for lung cancer only after a long latency period in
associated with increased risk in this study. However, if the workers who were employed during World War II and who left the shipyard prior to 1950 had constituted a less healthy group than those who stayed, then the selection bias resulting from the exclusion of the former group from our cohort may have caused us to underestimate the risks associated with shipyard exposure in asbestos. Selikoff and Hammond (20) have stated that 20% of all deaths in asbestos workers are due to lung cancer. This high rate does not seem to apply to the PHNS cohort of shipyard workers, among whom only 8.1% of the deaths thus far (9.1% in the exposed group and 6.3% in the nonexposed group) have been due to lung cancer.

ACKNOWLEDGMENTS

The authors wish to acknowledge the substantial contributions of Christie Brotherton, Marilyn Vanderford, Carolina Lau, and Gordon Arakaki to this research project.

REFERENCES

Cancer Occurrence in Shipyard Workers Exposed to Asbestos in Hawaii

Laurence N. Kolonel, Carl N. Yoshizawa, Tomio Hirohata, et al.


Updated version
Access the most recent version of this article at:
http://cancerres.aacrjournals.org/content/45/8/3924

E-mail alerts
Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions
To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions
To request permission to re-use all or part of this article, use this link http://cancerres.aacrjournals.org/content/45/8/3924.
Click on "Request Permissions" which will take you to the Copyright Clearance Center's (CCC) Rightslink site.