Mortality among Navajo Uranium Miners

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Introduction

Radon progeny, both in the workplace and in the household, are a continuing cause of concern because of the well-established association between exposure to radon progeny and lung cancer. In addition to lung cancer, associations have been suggested between exposure to radon progeny and other cancers, including leukemia, non-Hodgkins lymphoma, malignant melanoma, and kidney cancer, in studies of uranium and other hard rock miners in Canada, Sweden, Czechoslovakia, and the United States. In the United States, excess mortality risks for lung cancer, pneumoconioses and other respiratory diseases, tuberculosis, and chronic nephritis have been reported for the heavy-smoking White uranium miners of the Colorado Plateau study group. Studies among various populations of Navajo uranium miners in the United States have demonstrated excess mortality from lung cancer. The most recent report on the Navajo uranium miners in the Colorado Plateau study group, published in 1976, indicated significant excess mortality from lung cancer and a trend toward excess mortality from tuberculosis, nonmalignant respiratory disease, and unintentional injuries. To provide updated information on lung cancer and other mortality risks for these light-smoking Navajo men exposed to radon progeny, we have now updated and reanalyzed the cohort mortality data on the Navajo uranium miners from the Colorado Plateau study group.

Methods

The cohort of Navajos was selected from the Colorado Plateau study group of 4,126 White and non-White men who

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and Hornung and Meinhardt have evaluated the magnitude of error. In our life table analysis, exposures to radon progeny were separated into four categories having approximately the same number of expected deaths. The lowest exposure category (less than 120 working level months) was chosen to coincide with exposures that would be allowable under the current limit of 4 working level months per year over a 30-year career; our other three categories were 120 to 400, 400 to 1000, and 1000 or more working level months.

Vital status was ascertained from January 1, 1960, through December 31, 1990, through the records of the Social Security Administration, Internal Revenue Service, National Death Index, Health Care Financing Administration, and Indian Health Service. Those whose vital status was unknown were considered lost to follow-up on the date last observed. Death certificates were obtained and coded by a qualified nosologist into the appropriate revision of the International Classification of Diseases (ICD); the rules in effect at the time of death were used in coding. Deceased individuals for whom no certificate was located were assumed dead on the date specified by the reporting agency with the cause of death unknown.

**Life Table Analysis**

A modified life table analysis system was used in analyzing the cohort. Time since first exposure and duration of exposure were calculated from the first date of employment underground in a uranium mine. Because we used state mortality rates, initiated in 1960, as our comparison, person-years at risk of dying were also calculated from 1960. The expected number of deaths was computed by multiplying cause- and time-specific mortality rates for non-Whites in New Mexico and Arizona by the corresponding person-years distribution in the study population. Standardized mortality ratios represented the ratio of observed to expected deaths. The combined New Mexico and Arizona rates for mortality among non-White men (all except Whites and Hispanics) were chosen as the comparison rates because Navajos, Zunis, and other American Indians composed a relatively large percentage of the non-White population.

Direct standardization of rates, used to calculate the standardized rate ratios for lung cancer in Table 3, provided for internal comparisons between the higher exposure categories (i.e., 120 to 400, 400 to 1000, and >1000 working level months) and the lowest exposure category (<120 working level months). Our procedure for direct standardization calculated weighted rates for each of the four exposure categories; the age, race, sex, and calendar time stratum-specific sums of person-years across all exposure categories for the entire cohort were used as weights. The 95% confidence interval (CI) for the standardized rate ratio was calculated on the basis of a Taylor series approximation of the variance.

In calculating standardized mortality and standardized rate ratios by exposure categories in Table 3, we lagged exposure to radon progeny by 5 years under the assumption that 5 years was a reasonable estimate of a minimum induction period necessary before exposure could cause disease.

**Cox Regression Analysis**

To account for simultaneous risk factors for disease mortality and to address problems associated with the choice of a possibly inadequate external comparison group encountered in the life table analyses, we performed a Cox regression analysis of the entire Navajo cohort. The disease-specific age at death was the censored outcome variable. All comparisons were thus age adjusted. The analyses were also stratified on categories of year of birth (1883 to 1909, 1910 to 1916, 1917 to 1923, 1924 to 1943). The time-dependent regressors considered for inclusion in the models were cumulative exposure, log cumulative exposure, duration of exposure, log duration, exposure rate, log exposure rate, log cumulative pack-months of smoking (all lagged 5 years), time since first exposure, time since last exposure, and smoking status (yes/no), together with possible two-way interactions. Log cumulative exposure was taken to be the logarithm of cumulative exposure to radon progeny plus a background of 0.4 working level months per year of age. Duration of exposure was taken to be the number of months of underground uranium mining. This variable included all mining exposures and included but was not limited to radon progeny. Exposure rate was taken to be the ratio of cumulative exposure to radon progeny divided by months spent in underground mining. Log cumulative pack-months was the logarithm of cumulative pack-months of smoking plus a background of .005 packs per day. We considered log-linear models in the logarithm of cumulative dose (these models are sometimes called "power" function models), as follows:

\[ h(t, \text{dose}) = h_0(t)e^\beta \text{dose + background} \]

where \( h(t, \text{dose}) \) and \( h_0(t) \) are the hazard and baseline hazard functions, respectively. We also considered the linear excess relative risk function

\[ h(t, \text{dose}) = h_0(t) + \beta \text{dose}/100 \]

Plots of log cumulative hazards were used to check the proportional hazards assumption within categories of year of birth. The EPICE3 software package was used to perform all Cox regression analyses.

**Results**

Of the 779 non-Whites in the Colorado Plateau study group, 3 were Black and 1 was Asian; the remaining 775 were Navajos, including Hopi, Laguna, and Comanche. Although we included Blacks, Asian Americans, and other Native Americans in our cohort, just as they were included in the non-White state mortality rates that we used for comparison, we have called our study group a Navajo cohort because the vast majority were Navajos. Twenty-two Navajo miners were excluded because they did not meet the cohort eligibility criteria. The characteristics of the remaining cohort of 757 Navajo miners are given in Table 1.

We reviewed all of the records on smoking from the periodic surveys between 1950 and 1973 for the Navajo miners. All records reviewed were available at the time of previous smoking classifications. We reviewed the records to correct all smoking classifications without knowledge of disease status. This reclassification caused a small difference between the smoking data in this report and previous reports on the Navajo miners. The revised smoking classifications are reported in Table 1. All Navajo miners reporting that they smoked pipes or cigars also smoked cigarettes. The 38 Navajos who chewed tobacco or used snuff with no other use of tobacco were categorized as never having smoked.

**Cohort Mortality**

The 303 deaths observed are listed by their underlying causes in Table 2, along with the expected numbers of deaths and standardized mortality ratios. Heart, circulatory, and digestive diseases (specifically cirrhosis standardized mortality ratio = 0.5, 95% CI = 0.2, 0.7) were found
to involve lowered standardized mortality ratios. Kidney cancer (Table 2), leukemia (standardized mortality ratio = 0.0, 95% CI = 0.0, 2.8), non-Hodgkins lymphoma (standardized mortality ratio = 0.6, 95% CI = 0.01, 3.3), and melanoma (standardized mortality ratio = 0.0, 95% CI = 0.0, 18.4), suspected of being associated with exposure to radon progeny, were not found to be in excess. Other diseases associated with uranium mining, including chronic nephritis and renal sclerosis (standardized mortality ratio = 0.9, 95% CI = 0.2, 2.5) and unintentional injuries (Table 2), also were not found to be in excess. Lung cancer, pneumoconioses and other respiratory diseases, and tuberculosis, however, were found to be in excess (Table 2); further analyses focus on these three diseases.

For the 34 Navajos who died from lung cancer (Table 2), (1) the mean age at death was 53 years (range = 33 to 81 years), (2) the mean time since first exposure to radon progeny was 26.7 years (range = 12.5 to 44.7 years), and (3) the mean exposure to radon progeny was 1517 working level months (median = 1284 working level months), (range = 30 to 3896). Cigarette smoking status for the 34 Navajos who died from lung cancer was about the same as that reported for the entire cohort of Navajo miners (Table 1). Mortality risk ratios for lung cancer are reported in Table 3 by


Some ICD-9 codes in broad ranges were excluded and fall under "Other causes."

Duplicate deaths were not included in the calculations for each cause.

TABLE 2—Mortality in Navajo Uranium Miners vs Non-Whites in New Mexico and Arizona: 1960 through 1990

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>ICD-9* Code(s)</th>
<th>No. of Observed Deaths</th>
<th>No. of Expected Deaths</th>
<th>Standardized Mortality Ratio</th>
<th>Exact 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cancers</td>
<td>140-208</td>
<td>56</td>
<td>42.2</td>
<td>1.3</td>
<td>1.0, 1.7</td>
</tr>
<tr>
<td>Stomach</td>
<td>151</td>
<td>7</td>
<td>4.2</td>
<td>1.7</td>
<td>0.7, 3.4</td>
</tr>
<tr>
<td>Intestine</td>
<td>152-153</td>
<td>2</td>
<td>2.5</td>
<td>0.8</td>
<td>0.1, 2.9</td>
</tr>
<tr>
<td>Rectum</td>
<td>154</td>
<td>1</td>
<td>1.0</td>
<td>1.0</td>
<td>0.02, 5.6</td>
</tr>
<tr>
<td>Liver</td>
<td>155-156</td>
<td>2</td>
<td>2.7</td>
<td>0.7</td>
<td>0.1, 2.4</td>
</tr>
<tr>
<td>Pancreas</td>
<td>157</td>
<td>1</td>
<td>2.3</td>
<td>0.4</td>
<td>0.01, 2.4</td>
</tr>
<tr>
<td>Lung</td>
<td>162</td>
<td>34</td>
<td>10.2</td>
<td>3.3</td>
<td>2.3, 4.6</td>
</tr>
<tr>
<td>Prostate</td>
<td>185</td>
<td>1</td>
<td>4.5</td>
<td>0.2</td>
<td>0.01, 1.2</td>
</tr>
<tr>
<td>Kidney</td>
<td>189.0-189.2</td>
<td>1</td>
<td>1.2</td>
<td>0.8</td>
<td>0.02, 4.7</td>
</tr>
<tr>
<td>Bladder</td>
<td>188, 189.3-189.9</td>
<td>1</td>
<td>0.7</td>
<td>1.5</td>
<td>0.04, 8.2</td>
</tr>
<tr>
<td>Unspecified</td>
<td>194-199</td>
<td>3</td>
<td>4.5</td>
<td>0.7</td>
<td>0.1, 2.0</td>
</tr>
<tr>
<td>Lymphatic/hematopoietic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonmalignant diseases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>001-018</td>
<td>12</td>
<td>4.6</td>
<td>2.6</td>
<td>1.4, 4.6</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>250</td>
<td>6</td>
<td>8.3</td>
<td>0.7</td>
<td>0.3, 1.6</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>303</td>
<td>4</td>
<td>9.6</td>
<td>0.4</td>
<td>0.1, 1.1</td>
</tr>
<tr>
<td>Nervous system</td>
<td>320-389</td>
<td>6</td>
<td>4.6</td>
<td>1.3</td>
<td>0.5, 2.8</td>
</tr>
<tr>
<td>Heart</td>
<td>390-429</td>
<td>39</td>
<td>65.3</td>
<td>0.6</td>
<td>0.4, 9.8</td>
</tr>
<tr>
<td>Circulatory system</td>
<td>415-417, 430-459</td>
<td>8</td>
<td>21.8</td>
<td>0.4</td>
<td>0.2, 0.7</td>
</tr>
<tr>
<td>All respiratory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumoconioses and</td>
<td>470-478, 494-519</td>
<td>20</td>
<td>7.7</td>
<td>2.6</td>
<td>1.6, 4.0</td>
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<tr>
<td>Other respiratory</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digestive</td>
<td>520-579</td>
<td>12</td>
<td>26.3</td>
<td>0.5</td>
<td>0.2, 0.8</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>580-620</td>
<td>6</td>
<td>8.0</td>
<td>0.8</td>
<td>0.3, 1.6</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>711-739</td>
<td>2</td>
<td>0.6</td>
<td>3.3</td>
<td>0.4, 11.9</td>
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<tr>
<td>Ill defined</td>
<td>780-793, 795</td>
<td>28</td>
<td>19.1</td>
<td>1.5</td>
<td>1.0, 2.1</td>
</tr>
<tr>
<td>Unintentional injuries</td>
<td>E800-E949</td>
<td>57</td>
<td>57.9</td>
<td>1.0</td>
<td>0.7, 1.3</td>
</tr>
<tr>
<td>Suicide</td>
<td>E950-E959</td>
<td>8</td>
<td>4.9</td>
<td>1.6</td>
<td>0.7, 3.2</td>
</tr>
<tr>
<td>Homicide</td>
<td>E960-E978</td>
<td>6</td>
<td>12.4</td>
<td>0.5</td>
<td>0.2, 1.0</td>
</tr>
<tr>
<td>Other causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All causes</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>303*</td>
<td>325.5*</td>
</tr>
</tbody>
</table>

*Persons with unknown vital status had person-years accumulated until last date observed.

**The number of person-years was 19,185.

*Includes tobacco chewers and users of smoke.

**Median exposure = 403 working level months; median exposure rate = 9 working level months per month.

*Includes 6 deaths with missing death certificates.

**Includes 4.5 expected deaths for diseases not included in this table.

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cumulative exposure to radon progeny. No deaths from lung cancer were observed among Navajos within 10 years of their first exposure to radon progeny.

Cox Regression Analyses

Lung Cancer. For lung cancer, we considered both log-linear relative risk models and linear excess relative risk Cox regression models. The log-linear relative risk model using duration of exposure yielded the best fit. Increased duration of exposure was strongly related to increased risk of lung cancer mortality ($\beta = 0.2611, \text{SE} = 0.0383, P < .0001$). The estimated relative risk for a 5-year duration of exposure vs nonexposure was 3.7 (95% CI = 2.5, 5.4). When duration of exposure was included in the log-linear model, no other variables added significantly to the model fit. Increased log cumulative exposure to radon progeny was also strongly related to increased mortality from lung cancer in the log-linear model ($\beta = 0.9838, \text{SE} = 0.1902, P < .0001$).

With log cumulative exposure to radon progeny in the log-linear model, time since last exposure and exposure rate, when considered separately, added significantly to the model fit. For linear excess relative risk models, cumulative exposure to radon progeny was the best predictor variable, while time since last exposure added significantly to the model fit.

In order to use the Cox regression results to obtain estimates of relative risk comparable to the standardized rate ratios included in Table 3, we estimated the risk of each category relative to the same internal comparison group (exposure of less than 120 working level months) using the median exposure levels in the categories. For example, the linear excess relative risk model ($\beta = 1.986$) revealed that the estimated relative risk for the 400 to 1000 working level month category vs the less than 120 category was 6.9 (95% CI = 2.3, 13.0) (Table 3). A likelihood-based approach was used to obtain the confidence interval because of the unreliable standard errors in the linear excess relative risk model.

In the log-linear model including log cumulative exposure and time since last exposure, log cumulative exposure was positively related to mortality ($\beta = 0.9271, \text{SE} = 0.1992, P = .0001$), while time since last exposure was negatively related to mortality ($\beta = -0.0984, \text{SE} = 0.0384$, $P = .008$) ($\beta$ over $\beta$ indicates a model estimate). The estimated relative risk for 400 working level months relative to no exposure was 9.0 (95% CI = 4.6, 41.7). The estimated relative risk at 10 years since last exposure vs 0 years was 0.37 (95% CI = 0.18, 0.79). In the log-linear model including log cumulative exposure, rate of exposure to radon progeny was negatively related to mortality ($\beta = -0.0671, \text{SE} = 0.0269, P = .001$). The estimated relative risk due to an increase in the rate of 10 working levels was 0.51 (95% CI = 0.30, 0.87). No other variables contributed significantly to model fit when log cumulative exposure to radon progeny was included in the log-linear model. This was particularly true of all smoking variables and of all two-way interaction terms.

Pneumocoonioses and tuberculosis. Increased mortality due to pneumoconioses and other respiratory diseases ($P = .001$) and to tuberculosis ($P = .01$) was also significantly related to duration of exposure in the log-linear relative risk and linear excess risk Cox regression analyses. For the log-linear model, the coefficients of duration of exposure were .1519 (SE = .0441) for pneumoconioses and other respiratory diseases and .1426 (SE = .0658) for tuberculosis. Hence, the estimated relative risks for a 5-year duration of employment in underground uranium mining vs no such employment were 2.1 (95% CI = 1.4, 3.3) for pneumoconioses and other respiratory diseases and 2.0 (95% CI = 1.1, 3.9) for tuberculosis. No other variables contributed significantly to the model fit for these causes.

Mortality over calendar time. The standardized mortality ratio analysis showed that the lung cancer rate increased over time from 2.4 (95% CI = 0.6, 6.5) in the 1960s to 3.6 (95% CI = 2.2, 5.5) in the 1980s. The rate of pneumoconioses and other respiratory diseases increased as well, from 1.5 (95% CI = 0.1, 7.2) in the 1960s to 2.9 (95% CI = 1.4, 4.5) in the 1980s. When the effects of duration of employment and cumulative exposure to radon progeny over calendar time were controlled in the Cox analysis, the results indicated that exposure was the primary factor in the observed risk trend.

Discussion

Recent analyses of lung cancer among the Colorado Plateau study group have focused on the White miners.1,2,5,14-16 This is the first analysis since 1976 of mortality among the Navajo uranium miners in the Colorado Plateau study group. In this analysis, we extended vital status follow-up (17 years) through 1990, assessed risk by cumulative exposure to radon progeny and by duration of exposure to underground uranium mining, reviewed smoking status, and provided rate ratios using both an external and an internal comparison group. We found (1) elevated standardized mortality ratios for lung cancer (3.3, 95% CI = 2.3, 4.6), pneumoconioses and other respiratory diseases (2.6, 95% CI = 1.6, 4.0), and tuberculosis
(2.6, 95% CI = 1.4, 4.6); (2) trends of increasing risk with increasing duration of exposure to uranium mining and with increasing cumulative exposure to radon progeny for lung cancer, pneumoconioses and other respiratory diseases, and tuberculosis; and (3) a relative risk of 6.9 (95% CI = 2.3, 13.0) for lung cancer among those exposed to the midrange of 400 to 1000 working level months.

Prior to our study, the most recent investigation of the Navajo uranium miners in the Colorado Plateau study group had been conducted by Archer et al. with mortality follow-up through 1973. When we extended that follow-up by 17 years and used the same external comparison population for our standardized mortality ratio estimates, we found that overall mortality risk patterns had not changed. For mortality from all causes, lung cancer, nonmalignant respiratory disease, tuberculosis, and unintentional injuries, our standardized mortality ratio findings were similar to Archer’s. Our analysis revealed that standardized mortality ratios for lung cancer and pneumoconioses and other respiratory diseases appeared to continue to increase over time and remained elevated over the period 1980 to 1990. It seems clear that both lung cancer and pneumoconioses and other respiratory diseases continue to be elevated among this cohort of very-light-smoking Navajos an average of 23 years since their last exposure to radon progeny. However, the results of the Cox analysis indicated that exposure was the primary factor in the observed risk trend.

For lung cancer, our standardized mortality ratio of 3.3 (95% CI = 2.3, 4.6) was lower than the ratio of 4.2 (95% CI = 2.1, 7.6) estimated for Navajos by Archer et al. and the ratio of 4.8 (95% CI = 4.2, 5.6) estimated for White uranium miners by Waxweiler et al. Using Cox regression, we estimated a relative risk of 6.9 (95% CI = 2.3, 13.0) for those exposed to the midrange of 400 to 1000 working level months in comparison with those exposed to less than 120 working level months; using an internal comparison in our life table analysis, we estimated a standardized rate ratio of 8.4 (95% CI = 1.2, 61.8) for the same comparison (Table 3). Our estimates of lung cancer risk using internal comparisons are probably better indications of the true risk ratio.

Comparison of the Cox regression analysis of lung cancer in the present Navajo study with the previous Cox regression analysis of the White uranium miners revealed that increased exposure to radon progeny was strongly associated with increased risk of death due to lung cancer in both studies. The power function model was used in both studies, and similar coefficients were obtained for White and Navajo miners. In the Navajo study, duration of exposure was a better predictor than logged cumulative exposure to radon progeny (possibly as a result of measurement error in exposure). In the study of the White miners, cumulative exposure was a better predictor of lung cancer than duration of exposure (R. W. Hornung, J. A. Deddens, and R. J. Roscoe, written communication, 1993). In the Navajo study, the log-linear model in log cumulative exposure and the linear model in cumulative exposure both fit the data equally well (both had the same deviances), yielding estimated relative risks of 13.8 and 9.0 for 400 working level months relative to no exposure, respectively. Log cumulative pack-months of cigarette smoking was strongly associated with increased risk of death due to lung cancer in the White miners study but not in the present Navajo study, possibly because the Navajos smoked considerably less than their White counterparts. Of the Navajos, 58.9% had never smoked (Table 1), in comparison with about 18% of the White miners; moreover, among the Navajo miners categorized as light smokers, many were very light smokers (about 75% smoked five or fewer cigarettes per day). In both the Navajo and White studies, exposure rate and time since last exposure showed a negative association with risk of death due to lung cancer.

For all nonmalignant respiratory disease, our standardized mortality ratio of 1.4 (95% CI = 1.0, 1.9) was similar to the ratio of 1.3 (95% CI = 0.5, 1.8) for Navajos estimated by Archer et al. and lower than the ratio of 3.2 (95% CI = 2.6, 3.9) for White uranium miners estimated by Waxweiler et al. Differences in smoking habits are probably the explanation for the difference in standardized mortality ratios for Navajos and Whites. The excess mortality in our cohort was mainly due to pneumoconioses and other chronic respiratory diseases. Silica and other mining exposures, rather than radon progeny, are the likely explanations for the increased risk.

Our estimate of tuberculosis mortality (standardized mortality ratio = 2.6, 95% CI = 1.4, 4.6) was similar to the mortality ratio for Navajos estimated by Archer et al. (2.2, 95% CI = 0.9, 4.3) and lower than that for White uranium miners estimated by Waxweiler et al. (4.1, 95% CI = 2.2, 6.9). Silicosis and silicotuberculosis were mentioned on 4 of the 12 death certificates, indicating that silica, rather than radon progeny, is the likely explanation for the increased risk. Associations have been suggested between exposure to radon progeny and other cancers, including leukemia, non-Hodgkin’s lymphoma, malignant melanoma, and kidney cancer. 12-13 We did not observe elevated mortality from these cancers. This result is consistent with the findings of Darby et al. 27

An important factor that could affect the standardized mortality ratios in our life table analysis is the choice of mortality rates to be used for comparison. The mortality pattern for male American Indians in the Southwest is quite different from that of US White or non-White men. While rates of death due to tuberculosis, injuries, alcoholism, and diabetes are higher among American Indians, rates of death from ischemic heart disease, cancer (particularly lung cancer), and respiratory disease are lower. 26 The deficits in heart and lung disease are thought to be due, in large part, to a low prevalence of cigarette smoking among American Indians. 28 We chose the non-White mortality rates for New Mexico and Arizona as the comparison because no national mortality rates for American Indians were available and because the non-White population in these two states included a relatively high percentage of American Indians. However, because the non-White comparison rates include other races that have mortality patterns different from that of the Navajo, some standardized mortality ratios in this study may have been biased. Potentially confounding any bias were the changing percentages of American Indians among the non-Whites in the two states over the period of the study. To address this problem, we performed Cox regression analyses to provide for internal comparisons.

In conclusion, we found excess mortality for lung cancer, pneumoconioses and other respiratory diseases, and tuberculosis for Navajo uranium miners. Increasing duration of exposure to underground uranium mining was associated with increased mortality risk for all three diseases. Increasing cumulative exposure to radon progeny, decreasing exposure rate, and decreasing time since last exposure to radon progeny were associated with increased mortality risk for lung cancer. The pattern of risk ratios among the light-smoking Navajo uranium miners
was consistent with the risk ratios found in the previous analyses of Navajo and heavy-smoking White uranium miners. The most important long-term mortality risks for the Navajo uranium miners continue to be lung cancer and pneumoconioses and other nonmalignant respiratory diseases.

Acknowledgments

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References


7. 30 CFR 57.5037-57.5047 Mining Safety and Health Administration Radiation Standards for Workers in Underground Metal and Nonmetal Mines.


