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RETROSPECTIVE COHORT MORTALITY STUDY

OF

UNDERGROUND GOLD MINE WORKERS

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16. Abstract (Limit: 200 words)

An epidemiological study of mortality among underground gold mine workers (SIC-1041) was conducted. The cohort consisted of 3,328 white males who had been employed at the Homestake Mine, Lead, South Dakota for at least 1 year between January 1, 1940 and December 31, 1964. Total cohort exposure was 79,123 person years. The cohort was traced by reviewing company records, death certificates, Social Security and Internal Revenue data, and South Dakota state records. Mortality analyses were performed using a person year life table computer program. Previous surveys showed that the cohort was exposed to cummingtonite-grunerite, free silica (7631869), arsenic (7440382), and radon (10043922) daughters. No correlation of lung cancer mortality with underground exposures was observed (42 deaths versus 42.9 expected). Mortality due to tuberculosis and nonmalignant respiratory diseases was strongly correlated with exposure to silica (36 deaths observed versus 9.9 expected and 53 observed versus 19 deaths, respectively). A statistically insignificant increase in deaths from chronic kidney disease (8 observed versus 5 expected) that increased with length of employment was observed. The authors recommend further study of the incidence of chronic kidney disease mortality among underground miners, especially since other studies have found a similar correlation.

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ABSTRACT

In a retrospective cohort mortality study, underground gold mine workers (N = 3,328) were exposed to a noncommercial form of cummingtonite-grunerite (CG), free silica, arsenic (arsenopyrite), and radon daughters. The CG fibers found in airborne samples were relatively short (geometric mean length of 3.3 μ). In 1977, the geometric mean time weighted average (TWA) exposure for all miners to fibers greater than 5 μ in length was 0.44 fibers/cc (which was below the OSHA and MSHA asbestos 8-hour TWA standard of 2 fibers/cc). However, since total dust levels were higher in the past the exposure to fibers were also higher. Prior to 1952, exposure to free silica was generally above the accepted limit. In recent years, the arsenic and radon daughters levels have been below occupational exposure standards.

No association with exposures to underground gold mine workers and lung cancer mortality was demonstrated in this study (43 observed vs. 42.9 expected). Silica exposure, however, was strongly associated with mortality due to respiratory tuberculosis mostly, silicotuberculosis (36 observed vs. 9.9 expected) and nonmalignant respiratory disease mostly, silicosis (53 observed vs. 19 expected).

In gold mine workers first employed after 1951 when total dust levels decreased significantly, mortality from nonmalignant respiratory disease was still elevated (4 observed vs. 1.4 expected deaths). This analysis is limited by small numbers, and based on available information; none of the observed deaths were due to silicosis or respiratory tuberculosis.

As with other mining populations, mortality due to accidents (other than falls, poisonings, medical complications, and transportation), was significantly elevated in this study. The data demonstrates that those workers with short work experience are the most likely to die from accidents.

As has been observed in other underground mining populations, mortality due to chronic renal disease was elevated. Although not statistically significant, the risk increased with an increase in exposure.

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I. Introduction

Most commercial types of asbestos including those in the amphibole group have been shown to have the potential for causing lung cancer, asbestosis, and mesothelioma.[1] The pathogenicity of asbestos fibers may depend on the mineral type and physical characteristics of the fibers. It is thought that long thin fibers, with an aspect ratio of at least 3:1 (length/width), are the most pathogenic.[2]

There is considerable controversy over the definition of asbestos and whether certain noncommercial types of amphibole minerals should be designated as asbestos. According to the U.S. Bureau of Mines,[3] asbestos is "(1) a collective mineralogical term encompassing the asbestiform varieties of various minerals; (2) an industrial product obtained by mining and processing asbestiform minerals." Asbestiform minerals were further defined to be "a specific type of mineral fibrosity in which the fibers and fibrils possess high tensile strength and flexibility." Other definitions of asbestos such as those from the International Agency for Research on Cancer,[4] the National Academy of Sciences,[5] and Zoltai,[6] all require that the mineral originate from a fibrous habit, which can only be determined from bulk samples (megascopic properties). However, on a microscopic scale acicular or cleavage fragments that originate from nonfibrous amphibole minerals may be indistinquishable from those that are released from the fibrous habit form. Since it is unclear whether the pathogenicity of these

microscopic fibers differ, NIOSH[7] has recommended the following definition of asbestos for regulatory purposes; "(1) Asbestos is defined to be chrysotile, crocidolite, and fibrous cummingtonite-grunerite including amosite, fibrous tremolite, fibrous actinolite and fibrous anthophyllite. (2) The fibrosity of the above minerals is ascertained on a microscopic level with fibers defined to be particles with an aspect ratio of 3 to 1 or longer."

Most studies that have demonstrated an excess risk of lung cancer associated with asbestos have been among workers exposed to commercial forms of asbestos which orginated from a fibrous habit. Studies that examine the lung cancer risk associated with exposure to noncommercial asbestos, which do not originate from a fibrous habit and may exhibit different physical and chemical properties, are important in resolving the controversy surrounding the definition of asbestos. Aside from defining asbestos, it is important to determine the health effects from these mineral types because of the possibility of such exposures in industry and among the general population.

Public interest in the health effects from exposure to the amphibole mineral, cummingtonite-grunerite (CG) was heightened when it was discovered that CG had entered the drinking water of Duluth, Minnesota. This contamination occurred between 1955 and 1980, when tailings from a taconite mine were deposited in Lake Superior.[8] At the same time, it was known that CG was also present in the ore of the

Homestake Gold Mine which is located in Lead, South Dakota. Because of the difficulty in conducting an adequate epidemiologic study (with a long enough follow-up period) of Duluth residents, we decided to study the workers from the gold mine, which had been in operation since the late 1800's. However, since exposure of miners to CG is more likely to occur by inhalation rather than through ingestion, extrapolation of data for the Duluth drinking water situation is limited. This present study is the fourth reported mortality analysis of the Homestake population.

The first of the previous mortality studies was conducted by Gillam.[9] This study included 440 white males who had been examined in 1960 by the United States Public Health Service (USPHS)[10] during a silicosis survey and who, as of 1960, had worked for at least five years in underground mining at the Homestake gold mine and had never mined elsewhere. These men were followed from April 1, 1960, to December 31, 1973. South Dakota mortality rates were used as a standard in calculating expected deaths. A 3.7-fold increased risk of death for respiratory cancer was reported, as well as a 2.5-fold increase for nonmalignant respiratory disease (Table 1).

The second study, by McDonald, et al.,[11] was a cohort study of mortality, and included 1,321 workers who were members of the "Homestake Veterans Association" as of 1973 and who were employed for 21 years or more at the mining company. The workers were placed into five dust

exposure classifications from very low to very high. For the overall cohort study, the risk of mortality due to respiratory cancer was not increased. There was, however, an extremely high risk of death for pneumoconiosis and respiratory tuberculosis (Table 2) and a positive "dose-response" was observed for these causes, where dose or amount of exposure was estimated based on total dust.

The third study by Bell, et al.,[12] was also a cohort study of mortality. The cohort included 376 men who were on the active underground payroll as of January 1, 1946, and who had either already worked 5 years in underground jobs or subsequently completed 5 years in such work. Mortality rates of the United States were used to calculate the expected number of deaths. The SMR for lung cancer was 91 (8 observed vs 8.83 expected) and for those with at least 20 years of latency the SMR for lung cancer was 96 (8 observed vs 8.83 expected). The SMR for nonmalignant respiratory disease (other than bronchitis, pneumonia and influenza) was 650 (32 observed vs 4.9 expected).

The partially conflicting results of these studies provided inconclusive evidence regarding the question of CG exposure and mortality due to respiratory cancer. Because this question remained unanswered, the present more comprehensive study was conducted.

The Homestake population lends itself well to the examination of several important epidemiologic considerations which have been addressed in this study:

A. Of primary concern and the reason for initiating this study is the hypothesis that exposure to the amphibole mineral fibers found in the ore of the Homestake gold mine is associated with asbestos-related disease, such as lung cancer, asbestosis and mesothelioma.

B. Since silica dust has been a significant exposure at the mine, questions relating to this exposure have also been addressed. In particular, it has recently been suggested that silica exposure is associated with an increased risk of lung cancer,[13] and that it may be related to chronic renal disease.[14] Silica related nonmalignant respiratory diseases such as silicosis and silicotuberculosis have also been examined in some detail.

C. As in most mining populations, mortality due to accidents is a potential problem. This was also examined in the study population.

D. Since the analytic program used in this study is capable of evaluating 89 different categories of mortality, data on other causes of death are presented but not analyzed in any detail.

II. Description of the Facility and Environmental Aspects

Since the early 1900's, the mining practices at Homestake have continually improved. The changes in in the Homestake mining practices have resulted in lower occupational exposures to total dust, which is probably directly or indirectly correlated with most other specific exposures (e.g., arsenic, silica, CG, etc.). Dry drilling techniques were employed until 1919 when wet drilling was initiated. Changes in blasting were introduced in 1939, and then again in 1950; these changes substantially reduced exposure to dust and smoke. Also in 1950, automatic water valves were added to all drills, a change which further reduced exposure to dust.

Ventilation in the mine, as measured by exhaust air rate, has also undergone vast improvements since the early 1900's. From 1918 to 1923, the ventilation rate was estimated to be 77,000 cubic feet per minute (cfm). By successive improvements, in 1975 the ventilation rate had been increased to 720,000 cfm. Additional improvements in dust control have resulted from using progressively larger fans to deliver clean air to work sites.[15]

Another factor which reduced miners' exposures to many of the contaminants in the mine was the reduction in the number of hours spent underground per week. The number of hours worked per week has decreased steadily from 52 in 1920, to the present low of 40. Prior to 1920, the number of hours worked per week was greater than 52.

Based on data collected during environmental surveys conducted at the Homestake Mine, the following contaminants were thought to have the greatest potential for causing adverse health effects: quartz (free silica), amphibole mineral *fibers (or fragments), arsenic (arsenopyrite), and radon daughters.[15-18] Other airborne contaminants such as trace metals, carbon monoxide, carbon dioxide, methane, nitrogen dioxide, total hydrocarbons, benzo(a)pyrene, and total aldehydes were identified in the mine, but only in negligible quantities.

Based on examination of historical survey data, it appears that mean exposures to arsenic and radon daughters were within the current occupational standard (OSHA PEL = 10 $\mu g/m^3$ TWA). During an industrial hygiene survey[15] conducted in 1977, the geometric mean arsenic exposure for all miners grouped together was 1.17 $\mu g/m^3$ (95% confidence limits = 0.77 and 1.77 $\mu g/m^3$). The arithmetic mean for all miners was 2.03 $\mu g/m^3$ which was half the average exposure found by Mining Enforcement and Safety Administration (MESA)[18] in 1974. Only one work site in the 1977 survey was found to exceed the Occupational Safety and Health (OSHA)[19] standard of 10 $\mu g/m^3$. The NIOSH[20] recommended arsenic standard of 2 $\mu g/m^3$ for any 15-minute period was exceeded in 16 of the 51 samples. These samples were taken

*Since the fibers conform with the NIOSH definition of asbestos fibers they will be refered to as "fibers" throughout the remainder of the report.

as part of the fiber sampling and may not represent full-shift exposure. Based on a recent quanitative risk assessment by OSHA (47 FR 15358, April 9, 1982; 29 CFR 1910.1018) the levels of arsenic exposure experienced by the Homestake workers may have resulted in a slight excess in lung cancer mortality.

In the 1977 survey,[15] radon daughter ranged from 0 to 0.169 working levels (WL). In a survey conducted by MESA[21] in 1977, levels were reported to range from 0.01 to 0.130 WL. In a U.S. Bureau of Mines[16] survey conducted in 1960 the average WL exposure was 0.006. In 1968 and 1975 MESA [22,23] found WL's at 0.01. Because there was no attempt to identify sources of exposure in relation to ventilation systems, the exposures found prior to 1977 were probably low . The standard[24] for radon daughters is four working level months (WLM)/year or 0.33 WL at any given time. Therefore, the levels found in these surveys at Homestake were considerably below the accepted standard.

Based on electron microscopy analysis of airborne samples collected during the 1977 survey,[15] 84 percent of the airborne fibers were identified as amphiboles, while the remaining 16 percent were either unidentified (ambiguous) or not amphibole minerals. Sixty-nine percent of the amphiboles were characterized as CG, 15 percent as tremolite-actinolite, with the remaining 16 percent identified as fibrous hornblende minerals. The CG fibers had a geometric mean diameter of 0.43 μ (95% confidence limits of 0.39 and 0.47) with a

geometric mean length of 3.3 μ (95% confidence limits of 3.0 and 3.6); the tremolite-actinolite fibers had a geometric mean diameter of 0.27 microns and a geometric mean length of 4.1 μ. Twenty-four percent of the CG fibers and 32 percent of the tremolite-actinolite fibers had lengths greater than 5 μ.

Based on optical microscopy, personal exposures, as measured during the 1977 survey, for most job categories were below the current OSHA and Mine Safety and Health Administration (MSHA) time-weighted averages (TWAs) asbestos standard of 2 fibers (greater than 5 μ in length) per cubic centimeter. The geometric mean TWA exposure for all miners was 0.44 fibers (greater than 5 μ in length) per cubic centimeter. A summary of these findings is given in Table 3.

Prior to the early 1950's the dust levels and free silica content of the dust (39 percent in settled dust and 13.1 percent in *respirable dust) were sufficiently high enough to cause silicosis. After the early 1950's the overall dust levels dropped substantially to a point where the exposure to free silica was less likely to cause pneumoconiosis. This conclusion is based on the various standards which have been adopted for occupational exposure to free silica. The American Conference of Governmental Industrial Hygienists (ACGIH)[25] threshold limit Value (TLV) in terms of millions of

*The silica content of respirable dust was not measured until gravimetric sampling was developed in the mid-1970's.

particles per cubic foot (mppcf) is $300/\%SiO_2 + 10$ mppcf. Since the percent free silica found in the settled dust is 39%, the TLV is equal to 300/39 + 10 = 6.1. Table 4 gives a summary of the average dust level as measured by midget impingers by year and job category. Prior to 1952, the levels of total dust were above the TLV for silica, whereas, beginning in 1952, most of the dust levels have been substantially below the TLV.

In order to determine whether or not cause-specific mortality among Homestake miners is associated with exposure to total dust, an index of exposure was calculated. This index is based on the yearly average total dust exposure for specific job categories (see Table 4) and on the yearly average time spent (per shift) underground. The average time spent underground was estimated for each year, where the maximum time spent underground occurred prior to 1920 and was reported to be 3,600 hours per year. This was assigned a weighting factor of 10. For each year thereafter, the average time spent underground has declined. Based on a factor of 10 for 3,600 hours/year, a factor was calculated for each year corresponding to the average number of hours worked for that year (e.g., for 1933, when the yearly average was 2,642 hours the weighting factor was calculated to be 2,624 hours/3,600 hours X 10 = 7.3). All full-time underground jobs were assembled into five major groups based on similarity in job function and exposure to total dust. One additional category was used to group all jobs not considered full-time underground. Table 5 lists the jobs included in each of

these groups, which are the same as those given in Table 4. The index of exposure was calculated for each job category for each year by multiplying the dust level by the weighting factor for time underground. The indices of exposure are given in Table 6 for each year and job grouping. For the analyses of this study, index of exposure for any given time represents an approximate measure of exposure so as to examine dose-response relationships.

III. Methods

The epidemiologic study was designed as a retrospective cohort mortality analysis. The study cohort was defined so as to include all white, male Homestake workers who were employed full-time underground in the mine department for one year or more between January 1, 1940 and December 31, 1964. Their employment could have begun prior to 1940. All of the mine records were initially screened to select the study cohort, and company personnel records were used to supplement the data when necessary.

A verification that all eligible workers were included in the cohort was conducted. The first part of this verification was accomplished by comparing the data in the study cohort with other existing files. These existing files included the data from a silicosis survey conducted by the U.S. Public Health Service,[10] a comparison of lung cancer deaths from the McDonald[11] study and data from the Bell[12]

study. Based on the results of these comparisons it was concluded that approximately 4 to 5 percent of the eligible cohort members were missing from the cohort. Therefore, a second screening of the mine records was conducted. This screening yielded an additional 198 miners (including 31 deceased) who were eligible for the study; this group represents 6 percent of the cohort.

All workers who were known to have had experience in mining uranium were excluded from the cohort; this procedure resulted in the exclusion of 68 workers.

For each member of the final cohort, the jobs held at the Homestake Mine were coded into the six categories described in Table 5.

An attempt was made to determine the vital status of each worker as of June 1, 1977. This effort was accomplished by searching (1) records of the company, (2) death certificates from the previous NIOSH (Gillam) study, (3) Social Security Administration (SSA) data, (4) Internal Revenue Service (IRS) data, (5) South Dakota Department of Welfare data, (6) South Dakota Department of Motor Vehicles data, and (7) the Post Office. For all those workers identified as deceased, the death certificates were requested from State Vital Statistics' Offices and the underlying cause of death was coded by a nosologist according to the International Classifications of Diseases (ICDa) revision in effect at the time of death. Workers with an unknown vital status were

considered alive as of June 1, 1977 for the purposes of this analysis. This procedure typically results in slight underestimation of the specific mortality risks. If a miner was known to be deceased but a death certificate was not obtained, he was considered dead, cause unknown. If a worker died subsequent to the closing date, he was considered alive in the analysis of this study.

The mortality analysis was conducted using a person-years life table computer program described by Waxweiler, et al.[26] In the analysis, person-years (PY) at risk of dying were calculated for each worker, with PYs starting to accumulate after the worker had worked one year in full-time underground mining jobs after January 1, 1940. The PYs for each worker were accumulated until the closing date (June 1, 1977) or until his date of death, whichever occurred first. The PYs for all workers were then distributed into 5-year age groups and 5-year calendar-time periods and were multiplied by the corresponding United States white male mortality rate to yield the number of deaths expected. Table 7 gives the distribution of PYs by age and calendar-time. For certain causes of death an analysis of mortality was conducted by latency (time since first employment in a full-time underground job), duration of employment in full-time underground jobs, and accumulated exposure or dose (dust-days).

For the analysis by latency and length of underground employment, time spent in jobs that were not full-time underground (job category 5) was ignored. When calculating latency and length of underground

employment, work history prior to 1940 was included. The PYs were further stratified into 5-year latency groups beginning with at least 15 years of latency and by 5-year length of underground employment groups beginning with less than 5 years. A minimum of 15 years latency was selected, because most occupationally related chronic diseases do not occur prior to this time period. Table 8 gives the distribution of PYs by latency and by length of underground employment.

For the analysis by dose, PYs were distributed into categories of cumulative dose. Again, for this analysis PYs did not begin to accumulate until the employee worked for at least one year after January 1, 1940, even though, cumulative dose was based on total work history including employment at the mine prior to 1940. Cumulative dose was calculated by multiplying the index of total dust exposure (see Table 6) by time worked underground, yielding dose units equal to the index of dust exposure multiplied by days (dust-days). Categories or strata for dose were then established by examining the distribution of PYs for the cohort by dose. Intervals containing approximately equal numbers of PYs were chosen as the dose strata. The strata chosen increases geometrically by the number of dust-days. This selection of dose strata was done prior to any examination of cause-specific mortality in relation to dose. The allocation of PYs into these strata for each worker was accomplished by calculating the cumulative dose for each worker for each day he worked and assigning that day into the appropriate dose stratum. Cumulative dose was calculated for each

worker for each day by linking his work history with the index of exposure. The time (in days) spent in each job by specific calendar year was multiplied by the appropriate index of exposure for that job and year. Cumulative dose for a worker increased either by his accumulating more time underground and/or by changing to a job with a higher index of exposure.

For each worker, PYs were allocated to the appropriate dose stratum until the cumulative dose was greater than the cut-off point of that stratum, at which time the subsequent PYs were allocated to the next highest stratum. Table 9 gives the distribution of PYs by latency and dose.

Estimates of risk for specific causes of death were calculated as standardized mortality ratios (SMRs) -- observed deaths/expected deaths x 100. The statistical test employed to determine whether the number of observed deaths is significantly different than those expected is based on the Poisson distribution. For those causes of death where there was an a priori hypothesis, a one-sided test of significance ($p < 0.05$) was used. The causes of death with an a priori hypothesis were, lung cancer, nonmalignant respiratory diseases, and accidents. All other causes were also examined using a two-sided test of significance. In studies where multiple causes of death are being examined, 5% of the results are expected to be positive due to chance alone. Therefore, the results for those causes of death other than

those included in the a priori hypotheses should be interpreted cautiously taking into account trend with exposure and latency, and biological plausibility.

IV. Results

A. Cohort Description

The total cohort consists of 3,328 white males and 79,123 person-years. The results of vital status follow-up are given in Table 10. Only 5.6 percent of the cohort was lost to follow-up (unknown vital status), with a fairly uniform distribution of unknowns by date first employed.

A distribution of the cohort by length of underground employment and by year first employed underground is shown in Table 11. From this distribution, it can be observed that a large influx of new workers began employment in the ten years after World War II (1945-1954), and that 64 percent of the cohort was first employed in underground jobs after 1944. This influx of workers was due to a resumption of full-scale operations after the War. From an epidemiological viewpoint, this observation is important because it yields two subcohorts: (1) first, employed before the War; and (2) first employed after the War. The former group is older and is generally a selected surviving cohort; their maximum latency is longer and their exposures were relatively higher than those of the latter group.

B. Overall Mortality

Table 12 lists the observed and expected deaths for many of the causes which were examined. The SMR for all causes was greater than normal and considerably greater than that seen for most working populations where the "healthy worker effect (HWE)" is seen. This increase in overall mortality is due mainly to increases over expectation in deaths from nonmalignant respiratory disease, tuberculosis, and accidents. In fact, examination of the mortality due to circulatory disease (SMR = 85; $p < 0.05$), which is the cause of death usually most affected by the HWE, indicates that the HWE is in fact a factor in the mortality experience of this population. These overall patterns of mortality are similar to those observed in other mining populations, where pneumoconiosis has been a major cause of death observed.[27,28] It should be noted that the SMRs are not corrected for unknown causes of death which, in this study, include deaths where the death certificate was not obtained and thus cause could not be assigned. As result of this conservative procedure, the reported SMRs may be slightly lower than the "actual" SMRs.

C. A Priori Hypotheses

Lung Cancer

The overall risk for lung cancer (including malignancies of the trachea, bronchus and lung) in this population is almost exactly

that expected, based on United States mortality rates 43 observed vs. 42.9 expected. When analyzed by latency and length of employment (see Table 13), there are no apparent trends in lung cancer mortality with increasing latency or with an increase in length of underground employment. There is a nonstatistically significant excess in mortality for those workers employed for 15-20 years; however, the risk decreases after 20 years of employment in full-time underground jobs. Therefore, this particular excess may be a chance fluctuation.

In Table 14, the mortality analysis is stratified by latency and dose. Again, there is no demonstration of an increase in lung cancer risk with an increase in dose as measured by dust-days.

Nonmalignant Repiratory Disease (NMRD) and Respiratory Tuberculosis (RTB)

Because of the heavy exposures to silica prior to 1952, it was hypothesized that mortality from nonmalignant respiratory disease (NMRD) and respiratory tuberculosis (RTB) would be associated with employment at the gold mine. Overall, there was a highly statistically significant risk ($p < 0.01$) for both NMRD (53 observed vs. 19.00 expected) and RTB (36 observed vs. 9.94 expected). Mortality for NMRD by latency and length of employment underground is given in Table 15. When examined by latency (column total) a statistically significant risk occurs after 25 years, and the risk for subsequent time periods

remains fairly constant -- at a SMR of approximately 300. There is an increase in risk up to 15 or more years and then appears to level off at an SMR of about 500.

Further evidence for a dose-response relationship is seen in Table 16 where the mortality for NMRD by latency and dose (measured by total dust-days) is given. Again, there is a general increase in risk with an increase in dose. It should be noted that dose in this analysis is heavily weighted by length of underground employment, so the results are not totally independent of those presented in Table 15. The parameters of dose and length of underground employment are also correlated with latency. The risk for NMRD measured independent of latency can be observed by examining single rows within the matrices of Tables 15 or 16. With few exceptions within each latency stratum, there still remains a general trend of increasing risk with an increase in exposure -- whether exposure is measured as length of employment underground or as dose. This type of examination of the data shows that even for periods of long latency there is no increase in risk at the low-dose levels (below 80,000 dust-days).

The mortality due to RTB by latency and length of underground employment is presented in Table 17. In contrast to NMRD, where the SMR leveled off at about 300 after 25 years of latency, there is an SMR of 258 (4 observed vs. 1.55 expected) for RTB during 20-25 years of latency but the SMR increases to 1,683 (17 observed vs. 1.01 expected) after 35 years. The risk by length of underground employment for RTB

is clearly parallel to that seen for NMRD. There is a general increase in risk with an increase in length of underground employment. The pattern of mortality for RTB by latency and dose is given in Table 18, where it can be seen that the excess risk is restricted to the last two highest dose categories (or at a dose greater than 320,000 dust-days).

Since NMRD and RTB both reflect silica and dust-related disease (silicosis and silicotuberculosis) and due to the fact that there may have been misdiagnosis between the two diseases, especially in the earlier years,[29] the pattern of mortality with these two causes combined were examined in Table 19 (latency and length of employment underground) and Table 20 (latency and dose). Considered as one disease, there is a doubling of risk beginning at 20 years of latency and the risk levels off at an SMR of approximately 400 after 25-years. There is a time-related gradient of risk with an increase in length of employment underground, where the statistically significant excess begins during the 10-15 year period. Except for one category (160,000-320,000 dust-days) there is also a dose-related gradient in risk.

Accidents*

The risk for accidents (excluding those due to falls, poisoning, medical complications, and transportation) was 331 (58 observed vs.

*Accidents, as reported throughout this paper are defined as those other than falls, poisoning, medical complications, and transportation.

17.53 expected; $p < 0.01$). The excess risk for accidents is primarily among those workers with less than 15 years of underground employment (53 observed vs 14.9 expected). For those with greater than 15 years underground employment, there is still an excess risk (5 observed vs. 2.6 expected).

Temporal Considerations

The mortality patterns for diseases relevant to the a priori hypotheses were examined by subcohorts defined by date first employed. The results of this analysis are given in Table 21. Much of the excess risk in the cohort for overall mortality occurred in the subcohort of workers who were first employed prior to 1930. In fact, when this subcohort is excluded, the resulting SMR for all causes is reduced to 102 (526 observed vs. 517 expected).

More specifically, 32 of the 36 deaths due to RTB occurred in the subcohort first employed prior to 1930, resulting in an SMR of 792. For NMRD, 33 of the 52 deaths (SMR = 510) occurred in this subcohort. Smaller, but still elevated risks for NMRD persisted in many of the subsequent subcohorts. For lung cancer there was an elevated risk (15 observed vs. 12.02 expected; SMR = 125) for the subcohort first employed prior to 1930, all other subcohorts are near or below normal. The risk for accidents among those first employed prior to 1930 is no higher than in subsequent years; in fact, the highest risks (SMRs greater than 400) occurred after 1945.

One additional analysis was performed on a subcohort of all workers first employed after 1951. The purpose of this analysis was to determine cause-specific mortality risks associated with a substantial decrease in total dust levels after 1951 (see Table 21). The results from this analysis are limited since there were only a total of 109 deaths, which was significantly more than expected (109 observed vs. 92.66 expected; SMR = 118, $p < 0.05$). In terms of specific diseases of interest, there were no deaths due to RTB (0.40 deaths were expected), there were 4 deaths due to NMRD - none of which mentioned specifically silicosis on the death certificate - (1.40 deaths were expected), there were 3 deaths due to lung cancer (4.76 deaths were expected) and there were 21 deaths due to accidents (4.23 deaths were expected).

Although the number of deaths is small, it appears that the pattern of mortality by latency and length of underground employment for NMRD is similar to that seen in the entire cohort. That is, there is an increase in risk after 20 years of latency (SMR = 667, 3 observed vs. 0.45 expected) and the risk for those with greater than 5 years of underground employment is elevated (SMR = 548, 4 observed vs. 0.73 expected). The mortality pattern for accidents was also similar to that seen in the entire cohort. Twenty accidental deaths occurred in those with less than 10 years of employment underground, whereas, 3.60 were expected giving an SMR of 556.

The mortality for those diseases relevant to the a priori hypotheses was also examined by year of death. The results of this analysis are shown in Table 22. The excess risk due to RTB peaked between 1955 and

1964. The excess deaths from NMRD remained at a high level with SMRs ranging from 309 to 1,176 until 1970 at which time the SMR dropped off to less than 200. It is interesting to note that no deaths due to lung cancer occurred until after 1955. According to the company, smoking was prohibited in the mine from the early 1900's until 1952. This rule was strictly enforced due to the fire hazard. In 1952 no-smoking areas were established and smoking was allowed elsewhere. Thus, the prevalence of smoking may have been reduced prior to 1952 resulting in a lower risk for lung cancer before 1960 as indicated in Table 22. For accidents, the excess deaths were greatest during the earlier periods prior to 1965, however, they have remained greater than normal throughout the study period except for the last period, 1975-77 (only a 2-year period) where no accidental deaths occurred.

D. Other Diseases of Interest

Chronic Renal Disease

During the course of this study, mortality due to chronic renal disease became of interest because of observations which indicated that mine workers may be experiencing an increased risk from this cause of death.[14] In this study, the cause of death category -- "Chronic and unspecified nephritis and other renal sclerosis" was examined in response to this interest. The results for this analysis can be found in Table 12. There is a nonstatistically

significant excess (SMR = 160, 8 observed vs. 5.0 expected). When examined by length of employment (see Table 23) there appears to be an increase in risk with an increase in length of employment, however, this is based on very small numbers of deaths in each employment category. The mortality for this cause of death by dose (dust-days) is shown in Table 24. Although the numbers are small, there appears to be an indication of a positive dose-response, and it appears that the increased risk is confined to the group of workers with 320,000 or more dust-days where there are 6 observed vs. 2.10 expected deaths.

Malignant Neoplasms of Other Parts of the Respiratory System and of the Peritoneum

Both of these disease categories are of interest in this study because an observed death due to the asbestos-related diseases, pleural or peritoneal mesothelioma, could have been coded to one of the causes included in these categories. The mortality for each of these categories can be found in Table 12. For malignant neoplasms of other parts of the respiratory system, there were 3 observed vs. 0.6 expected deaths and for malignant neoplasms of the peritoneum there were 2 observed vs. 0.7 expected deaths. The numbers of deaths in each of these categories are too small to conduct any further detailed analysis by latency or exposure, however, a description of these variables and other pertinent information regarding these deaths are given in Table 25.

Malignant Neoplasms of the Prostate

A nonstatistically significant excess of prostatic cancer was observed in this cohort (11 observed vs. 7.6 expected deaths). When this cause was analyzed by latency and length of employment, there was no positive trend with either variable.

Malignant Neoplasms of the Lymphatic and Hematopoietic System

There have been several case reports of malignant neoplasm of the lymphatic and hematopoietic system among workers exposed to asbestos.[30-31] Furthermore, an increase in lymphatic tumors has been noted in animals exposed to silica.[32] Therefore, an analysis of this category of disease was of interest in this study. For the whole disease category combined, a nonstatistically significant excess was observed (19 observed vs. 13.9 expected deaths). For leukemia and aleukemia in particular, the SMR was 170 (10 observed vs. 5.9 expected deaths).

V. Discussion

In at least two studies [33,34] of workers exposed to Amosite (a type of asbestos mined in South Africa) an excess risk of death was observed from lung cancer. Amosite is a commercial grade amphibole which falls mineralogically into the CG series. The CG in both of these studies

was different from that found at Homestake. It was considered fibrous, on a megascopic as well as microscopic scale, whereas the CG found at Homestake forms in a nonfibrous mineral formation, and through attrition by various mining processes, breaks into respirable fibers. In addition to its fibrous nature, the physical dimensions (short and thick) of the CG found at Homestake differentiate it from commercial grade Amosite. This point is very important, because it is widely held that the most pathogenic asbestos fibers are those that are long and thin.

In a fiber implantation study by Stanton,[35] it was concluded that fibers less than 0.25 μ in diameter and longer than 8.0 μ in length may be the most important for production of pleural sarcomas. The observation that the pathogenicity of fibers is correlated to their length is further supported in a study by Davis.[36] Most (76%) of the fibers at Homestake were less than 5 μ in length (76% of CG and 77% of total fibers). The geometric mean length for Homestake CG Fibers was 3.3 μ and for total fibers it was 3.2 μ. The geometric mean diameter for CG Fibers was 0.43 μ. In 1977[15], the fiber exposure (geometric mean) to underground miners was 0.44 fibers (> 5 μ in length) per cubic centimeter.

This study of mine workers at Homestake offers important epidemiologic information regarding the lung cancer risk from this type of fiber exposure. In comparison to other studies[37] of asbestos exposure

which demonstrate clearly an excess lung cancer risk associated with asbestos, this study indicates there exists only a slight excess in mortality after 30 years of latency; 29 deaths were observed in that category and 22.5 were expected. However, there is no apparent evidence of a positive dose-response for lung cancer, whether the dose is measured by length of employment or by dust-days.

When mortality for lung cancer is examined by date first employed there is a slight excess mortality in those workers first employed before 1930. Although exposures were generally higher during that time period, the higher exposures do not explain the higher risk, since this is not consistent with the results of the dose-response analyses. It should be noted that the risk for nonmalignant respiratory disease especially among those employed prior to 1930 is very high (65 observed vs. 10.5 expected, SMR = 620; for RTB and NMRD combined). It is possible that this high risk for nonmalignant respiratory disease could have competed with the risk for lung cancer; that is, those with heavy exposure to total dust and CG fibers) may have died prematurely from nonmalignant dust-related disease before the malignant disease became manifested. To eliminate this possibility, the analysis can be restricted to those first employed after 1930, at which time there is a reduction in risk for nonmalignant respiratory disease. However, for this subcohort and for the exposures associated with it, there still is no excess observed in lung cancer mortality (28 observed vs. 30.9 expected).

Goldsmith[13] has postulated that exposure to silica may be associated with an increase in lung cancer mortality. This topic was thoroughly debated at a recent symposium entitled, "Silica, Silicosis and Cancer, An International Symposium" held in Chapel Hill, North Carolina on April 3-5, 1984. The proceedings of this conference are in press.

As discussed above, there does not appear to be an association between length of underground employment or total dust exposure (a measure of silica exposure) at the mine and lung cancer mortality. Again, unless the risk for lung cancer is masked by competing risks from nonmalignant disease, this study provides no evidence that either exposure to CG fibers or to silica from the Homestake Mine is associated with an excess risk of lung cancer .

It should be noted that lung cancer mortality rates in the United States, which were used to generate the expected number of deaths in this study, have been higher than those of South Dakota, the state where the majority of the study population has resided. According to the National Cancer Institute,[38] for the years 1950 to 1969, which represents an approximate midpoint of the study, the age adjusted lung cancer mortality rates for white males in the United States were 37.98 deaths/100,000, whereas, the corresponding South Dakota rates were 23.71 deaths/100,000. Therefore, if South Dakota rates had been used to generate expected deaths, the SMRs could have been elevated by as much as 38%. However, these findings related to lung cancer should

be viewed as truly showing no association to CG and silica for three reasons: 1) The patterns regarding latency and dose are inconsistent with an etiology related to exposures experienced at the mine, 2) The number of expected deaths in this cohort allows for a very powerful analysis; we had a 90 percent probability of detecting a true relative risk of 1.5 for lung cancer, based on a one-sided test. When the cohort was restricted to those with at least 15 years latency there was still an 88 percent probability of detecting the same risk and when restricted to at least 15 years latency and at least 5 years employment underground, there was still a 76 percent probability of detecting the risk, and 3) Arsenic exposure may have been high enough especially in the years prior to 1952, to produce slight excesses in lung cancer mortality. These exposures may confound the analysis of effects from either amphibole fibers or silica.

The overall risk for nonmalignant respiratory disease coded as either RTB or NMRD is significantly elevated. Both of these categories reflect dust-related pneumoconiotic diseases, and in this case, reflect silicosis-related disease; where the excess risk in RTB is due to excess mortality in silicotuberculosis, and the excess risk in NMRD is due to excess mortality in silicosis. Based on pathological reports obtained from hospitals, at least two-thirds of the individuals who died from NMRD had a confirmed diagnosis of silicosis.

An analysis of RTB and NMRD by length of employment in underground jobs, by dose (dust-days), and by latency indicate that both of these causes of death are associated with exposures experienced at the mine. For both causes, a positive dose-response gradient is observed whether the dose is measured by length of employment or by dust-days; and the significant excess in both causes occurred after a latent period predictable for an occupationally-related chronic disease. The dose-response relationship for NMRD is not strictly linear a finding which may be due to two factors. First, the category, NMRD, includes diseases that are not specifically related to silica dust exposure, for example, emphysema. Secondly, the calculations of length of employment and dose in terms of dust-days for the cohort is underestimated, because jobs other than those classified as "full time underground" were not counted in these calculations and some of these jobs had dust exposure.

The excess in RTB occurred almost entirely among miners first employed prior to 1930 with only one death due to RTB occurring after 1969. In part, this is a reflection of a general decline in death due to tuberculosis, because of better standards of living and improvements in medical care, and because of improvements in the diagnosis of silicosis. Similarly, most of the excess risk due to NMRD is among those first employed prior to 1930. However, the risk remains above normal for miners first employed in subsequent time periods as well. For the subcohort first employed after 1951 when dust levels were

substantially reduced, the risk for NMRD was still high with an SMR of 308 (4 observed vs. 1.4 expected). It is not known whether or not this excess is due to silicosis. For the only two deaths where pathological information was available, there was no mention of silicosis and in none of these four cases was silicosis mentioned on the death certificate. The number of deaths in this subcohort is small, and continuing follow-up will evaluate the possibility of silicosis among miners exposed after 1951.

The findings for nonmalignant respiratory disease in this study agrees with the previous studies[9,11,12] of the Homestake population. The findings for lung cancer agree with those of McDonald[11] and Bell[12] but disagree with those of Gillam.[9] This difference in the lung cancer finding could have resulted from the following: (1) the Gillam Study population was based on volunteers who participated in a Silicosis Survey in 1960 and does not necessarily represent an unbiased sample or the true population at risk. (2) In the Gillam Study actual personnel records were not used to verify work history, questionnaire data was used. (3) Lung cancer mortality was not reported separately from all respiratory cancer in the Gillam Study. (4) South Dakota death rates were used in the Gillam Study rather than U.S. death rates.

The risk for "other" accidents which excludes transportation, poisoning, falls, and medical complications, is consistently high for the cohort independent of the year first employed. When examined by

length of employment underground, an interesting pattern of mortality is observed. There is a negative "dose-response," where the highest risk is seen for those with less than 5 years of employment. This pattern is consistent with occupationally-related accidents among workers with little experience who have not been trained well enough and/or who have not had enough experience to avoid dangerous situations.

Since asbestos-related diseases are of great interest in this study, mortality from mesothelioma was of particular concern. Upon review of all death certificates no mention of mesothelioma was noted. It is of interest that both disease categories -- "malignant neoplasms of other parts of the respiratory system" and "malignant neoplasms of the peritoneum" were elevated. Improperly diagnosed pleural or peritoneal mesotheliomas could have been coded into these categories. However, when the information presented in Table 25 is taken into consideration, it is unlikely that any of these deaths were actually due to mesothelioma.

There were several other causes of death that were of interest in this study. However, the interpretation of data regarding each cause is inconclusive because of the limited number of deaths and because of the a posteriori nature of the hypotheses. Chronic renal disease was examined because of its hypothesized association with mining. This study provides additional evidence to support this hypothesis.

Although there was only a slight overall excess in mortality (8 observed vs. 5.0 expected deaths) there was an increase in risk with an increase in exposure (defined by length of employment and by dust-days). This observation should be further evaluated in other studies, especially other mining populations and other populations exposed to silica.

The excess mortality observed for leukemia is of interest because the risk increased with an increase in exposure (length of time and dust-days). However, it is very difficult to interpret these results because of the small numbers of deaths within each exposure category. This finding has some importance because of the findings by Wagner, et al.[32] who found an excess in malignant lymphomas among rats exposed to silica.

Prostatic cancer was also observed to be in excess. When examined by latency and exposure, however, the pattern of risk does not appear to be associated with work at the mine.

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Table 1. Results of the Gillam et al. Study, 1976.

Cause of Death	Observed Deaths	Expected Deaths	SMRs
All Deaths	71	52.9	134*
All Cancers	15	9.7	155
Respiratory Cancer	10	2.7	370**
Vascular Lesions of CNS	6	3.2	188
Heart Disease	25	25.2	99
Nonmalignant Respiratory	8	3.2	250*
Accidents	8	5.2	154

* Significant at $p < 0.05$.
** Significant at $p < 0.01$.

Table 2. Selected results of the McDonald et al. Study 1978.

Cause of Death	Observed Deaths	Expected Deaths	SMRs
All Deaths	631	549.7	115
All Cancers	93	90.5	103
Respiratory Cancer	17	16.5	103
Vascular Lesions of CNS	64	63.0	102
Heart Disease	264	232.5	114
Pneumoconiosis	37	--	--
Respiratory Tuberculosis	39	3.6	1083
Accidents	19	28.3	67

Table 3. Time weighted average airborne exposure (Optical Microscopy) to fibers greater than 5 microns among Homestake workers.

Job Category	Range (fibers/cm^3)	Geometric Mean
Miners	0.08-4.01	0.44
Underground Jobs Excluding Miners	0.02-2.57	0.24
Crushing Department Workers (Surface)	0.12-5.34	1.16

From: Reference 12

Table 4. Average yearly dust concentration (in millions of particles per cubic foot by year by job grouping at the Homestake Gold Mine (1937-1975).

Year	JOB CATEGORY				
	1	2	4	6	7
1937	8.1	6.6	12.8	7.4	95.1
1938	9.3	34.7	13.8	22.0	61.1
1939	12.0	21.0	15.7	16.5	27.1
1940	9.1	13.0	12.5	11.0	11.1
1941	12.4	10.2	14.2	11.3	11.1
1942	12.8	49.8	16.9	31.3	11.1
*1943-1945	---	---	---	---	---
1946	12.3	26.9	9.6	19.6	23.6
1947	12.3	26.9	9.6	19.6	23.6
1948	11.3	11.6	12.2	11.4	23.6
1949	22.1	12.3	15.5	17.2	20.0
1950	4.1	14.0	7.1	9.0	16.5
1951	6.3	6.7	10.7	6.6	13.0

*Except for limited activity, the mine was closed from October 16, 1942 to July 1, 1945.

Table 4. Average yearly dust concentration (in millions of particles per cubic foot by year by job grouping at the Homestake Gold Mine (1937-1975).

Year	JOB CATEGORY				
	1	2	4	6	7
1952	3.6	7.1	6.1	5.3	9.5
1953	1.5	5.5	7.3	3.5	0.7
1954	2.0	8.1	7.0	5.0	0.8
1955	3.2	5.4	5.5	4.3	0.9
1956	1.4	5.4	5.9	3.4	1.0
1957	2.6	5.0	4.4	3.8	1.1
1958	2.2	4.7	3.8	3.4	1.2
1959	2.6	4.3	4.2	3.5	1.3
1960	2.4	4.2	4.1	3.3	1.4
1961	2.1	4.0	3.8	3.2	1.5
1962	1.7	3.8	3.6	2.6	1.6
1963	1.3	3.6	3.4	3.0	1.7
1964	0.9	3.4	3.2	2.9	1.8
1965	0.9	4.5	3.2	3.6	2.8

Table 4. Average yearly dust concentration (in millions of particles per cubic foot by year by job grouping at the Homestake Gold Mine (1937-1975).

Year	JOB CATEGORY				
	1	2	4	6	7
1966	2.0	3.3	4.1	3.5	3.8
1967	1.7	3.1	3.6	2.8	7.9
1968	1.5	3.2	4.0	3.8	12.0
1969	1.5	2.9	3.6	2.9	9.8
1970	1.4	3.0	3.6	3.0	4.8
1971	0.7	2.5	3.6	3.4	4.1
1972	0.8	2.9	3.6	3.6	3.4
1973	0.8	2.9	3.7	3.8	2.7
1974	0.9	2.6	3.9	4.0	2.1
1975	0.9	2.6	3.9	4.0	2.1

FROM: May 27, 1976, memo from J. K. Waterland to D. T. Delicate - Homestake Mining Company, Lead, South Dakota.

Table 5. Job codes used in Homestake Mortality Study.

Code Group 1

Barman
Backfilling Sandman
Below Wall Builder
Car Oiler
Diamond Drill Operator
Diamond Drill Helper
Doorman
Head Pipeman
Laborer
Laborer Hi
Landman
Maintenance Man
Nipper
Pipeman
Sand Backfill Foreman
Sand Bk
Sand Bk Sandman
Sand Book Laborer
Sandman Backfilling
Sand Boss
Sand Boss Temp
Shoveler
Timberman
Timberman Helper
Tool Carrier
Trackman
(U.G.) Small Hoist
Underground Hoist Operator
Watchman in Mine (Underground)
Wall Builder

Code Group 2

Blockholer
Boring Machine Operator
Boring Machine Helper
Chute Drawer
Draw Chutes
Gunite Operator
Guniter
Lead Miner
Loader Operator
Miner (All Classes)
Miner Instructor
Power Shovel Operator
Sampler
Sample Supervisor
Shovel Operator

Code Group 4

Motorman
Tallyman
Team Trainman

*Code Group 5

Amicus Mill
Batteryman
Beltman
Beltman, Mine Surface
Bin Cleaner
Bucket Dumper
Cager, All Types
Canvas and Leatherman
Carpenter, All Classes
Chief Timekeeper
Clean-Up Man
Clerk, All Types
Coneman
Crusher Feeder
Crusherman
Double Rainbow-Prefixed Titles
Drill Repair Boss
Dryman
Dust Room Helper
Efficiency Expert
Engineer, All Types
Gas Patrol
Gateman, All Types
Golden Reward-Prefixed Titles
Instrument Man
Lampman
Master Pipeman
Mechanic, All Types
Mine Superintendent
Mine Surface Foreman
Mine Surveyor, and Helper
Mine Survey Rodman
New Cooling Tower
Oiler
Oil Man
Powderman
Reno Powderman
Repairman, All Classes
Research Technician
Safety Director
Saw Filer
Sawmill-Prefixed Titles
Sawyer
Shaftman
Sluicer
Spearfish Hydro Tunnel #2
Surface-Prefixed Titles
Testing Assistant
Timekeeper
Tool Sharpener
Tram, or Tramway
Truckman, All Types
Ventilation Technician
Water Pipe Tunnel

Code Group 6

Assistant Day Foreman
Assistant Night Foreman
Diamond Drill Foreman
Division Foreman
General Foreman
Shaft Boss
Shaft Foreman
Shift Boss
Skip Boss

Code Group 7

Skiploader
Skipper
Skipper (ldgs)

*All Jobs not in Mining Department
*Leaves or Absences Greater than 31 days

Table 6. Index of dose by job category and year.

Year	Job Category				
	1	2	4	6	7
Pre-1920	100.0	100.0	100.0	100.0	100.0
1920-32	74.0	74.0	74.0	74.0	74.0
1933	73.0	73.0	73.0	73.0	73.0
1934	68.0	68.0	68.0	68.0	68.0
1935	65.7	65.7	65.7	65.7	65.7
1936	65.7	65.7	65.7	65.7	65.7
1937	53.5	43.6	84.5	48.8	627.7
1938	61.4	229.0	91.1	145.2	403.3
1939	78.0	136.5	102.1	107.3	176.2
1940	58.2	83.2	80.0	70.4	71.0
1941	70.7	58.1	80.9	64.4	63.3
1942	76.8	298.8	101.4	187.8	66.6
*1943	10.0	10.0	10.0	10.0	10.0
*1944	10.0	10.0	10.0	10.0	10.0
*1945	10.0	10.0	10.0	10.0	10.0
1946	66.4	145.3	51.8	105.8	127.4
1947	66.4	145.3	51.8	105.8	127.4
1948	59.9	61.5	64.7	60.4	125.1
1949	117.1	65.2	82.2	91.2	106.0
1950	23.0	78.4	39.8	50.4	92.4
1951	38.4	40.9	65.3	40.3	79.3
1952	20.2	39.8	34.2	29.7	53.2
1953	9.8	35.8	47.5	22.8	4.6
1954	13.0	52.7	45.5	32.5	5.2
1955	20.5	34.6	35.2	27.5	5.8
1956	9.1	35.1	38.4	22.1	6.5
1957	16.9	32.5	28.6	24.7	7.2
1958	14.5	30.6	24.7	22.1	7.8
1959	16.9	28.0	27.3	22.8	8.5
1960	15.6	27.3	26.7	21.5	9.1
1961	13.7	26.0	24.7	20.8	9.8
1962	11.1	24.7	23.4	16.9	10.4
1963	8.5	23.4	22.1	19.5	11.1
1964	5.9	22.1	20.8	18.9	11.7
1965	5.9	29.3	20.8	23.4	18.2
1966	12.8	21.1	26.2	22.4	24.3
1967	10.5	19.2	22.3	17.4	49.0
1968	9.0	19.2	24.0	22.8	72.0
1969	9.3	18.0	22.3	18.0	60.8
1970	8.8	18.9	22.7	18.9	30.2
1971	4.6	16.3	23.4	22.1	26.7
1972	4.5	16.2	20.2	20.2	19.0
1973	4.4	16.0	20.4	20.9	14.9
1974	5.0	14.3	21.5	22.0	11.6
1975	5.0	14.3	21.5	22.0	11.6

*For 1943-1945, the mine was closed but there was limited activity. An index of dust exposure equal to 10.0 was assigned for these years for all jobs. Prior to 1937, no dust counts were available, therefore, they were estimated by extrapolating from 1937-1952 data and from personal (Homestake) knowledge of conditions in mines with similar work practices.

Table 7. Distribution of white, male person years at risk of dying by age and calendar time period for Homestake workers (January 1941 - June 1977).

Ages	1941-1944	1945-1949	1950-1954	1955-1959	1960-1964	1965-1969	1970-1974	1975-1977	Total
15-19	0.33	1.87	0.87	0.00	0.01	0.00	0.00	0.00	3.09
20-24	218.98	188.08	579.25	483.62	495.63	279.39	0.00	0.00	2,244.95
25-29	558.92	690.77	1,395.24	1,607.21	1,146.83	969.53	276.55	0.00	6,645.05
30-34	780.81	991.78	1,513.60	2,236.68	2,026.25	1,400.22	960.15	227.81	10,137.31
35-39	980.30	1,170.12	1,564.84	1,997.81	2,475.44	2,161.98	1,382.71	494.52	12,227.73
40-44	748.44	1,232.47	1,353.00	1,789.18	2,120.18	2,552.38	2,123.88	734.08	12,653.62
45-49	434.08	910.45	1,201.32	1,335.78	1,752.09	2,099.20	2,491.21	1,137.52	11,361.65
50-54	282.72	483.74	858.84	1,131.20	1,272.73	1,651.25	1,999.92	1,091.66	8,772.04
55-59	184.00	328.47	457.46	798.85	1,045.91	1,182.57	1,550.48	884.21	6,431.95
60-64	61.92	170.20	281.29	384.93	688.62	922.65	1,064.67	609.54	4,183.81
65-69	21.71	60.03	132.35	213.79	300.42	583.80	786.08	433.28	2,531.46
70-74	0.00	14.78	41.23	101.94	155.54	221.20	429.31	292.70	1,256.71
75-79	2.20	0.00	10.15	21.02	70.57	105.93	143.51	129.52	482.90
80-84	1.00	0.00	0.00	3.84	7.59	36.98	60.79	25.67	135.88
85+	0.00	0.00	0.00	0.00	1.98	5.46	25.13	22.02	54.60
Total	4,275.40	6,242.76	9,389.45	12,105.86	13,559.80	14,172.56	13,294.39	6,082.54	79,122.77

Table 8. Person years matrix by latency and length of employment underground for Homestake workers.

Latency	Length of Employment Underground (Years)							
	1 - 4	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	30 Years & Over	Total
Under 15 Years	24940.47	7831.61	2844.76	0.00	0.00	0.00	0.00	35616.85
15-19 yrs	6378.55	2728.50	2360.23	1788.11	0.00	0.00	0.00	13255.41
20-24 yrs	5023.12	2175.54	1529.61	1350.18	1034.04	0.00	0.00	11112.51
25-29 yrs	3125.85	1612.62	1144.72	667.49	806.96	416.62	0.00	7774.28
30-34 yrs	1344.22	1184.29	958.00	467.10	382.73	432.36	154.93	4923.67
35-39 yrs	598.44	889.89	772.93	396.56	229.44	228.24	262.34	3377.87
40 Years & Over	90.56	678.97	866.81	535.79	318.95	220.88	350.16	3062.14
Total	41501.25	17101.44	10477.09	5205.25	2772.14	1298.12	767.43	79122.75

Table 9. Person years matrix by latency and dose for Homestake workers.

Latency	Dosage (Dust-Days)							
	Under 20000	20000 - 40000	40000 - 80000	80000 - 160000	160000 - 320000	320000 - 640000	640000 & & Over	Total
Under 15 Years	8159.64	7743.70	9410.08	6125.61	3194.03	1184.42	0.00	35817.51
15-19 yrs	1602.45	1874.09	2734.75	2716.16	2266.25	2073.87	8.24	13275.86
20-24 yrs	844.23	1412.05	2127.68	1973.88	2252.99	2292.28	211.14	11114.28
25-29 yrs	135.61	784.05	1297.32	1277.68	1711.06	2132.68	436.10	7774.55
30-34 yrs	7.72	205.53	479.90	671.03	1173.30	1836.84	563.69	4938.03
35-39 yrs	1.96	42.21	188.42	357.36	817.45	1491.65	481.16	3380.24
40 Years & Over	0.00	6.71	18.52	94.62	555.77	1701.18	693.41	3070.23
Total	10751.64	12068.36	16256.37	13216.37	11970.88	12712.93	2393.77	*79370.68

* The number of person-years calculated by dose is slightly different than those calculated by length of employment. This will result in slight differences in the expected number of deaths for the two analyses.

Table 10. Vital status ascertainment for the Homestake Cohort.

Year First Employed	Alive (%)	Dead (%)	Unknown (%)	Total
<1930	106 (23)	335 (74)	11 (3)	452
1930 - < 1935	121 (45)	134 (50)	13 (5)	268
1935 - < 1940	173 (62)	96 (34)	12 (4)	281
1940 - < 1945	140 (67)	55 (26)	15 (7)	210
1945 - < 1950	547 (76)	113 (16)	55 (8)	715
1950 - < 1955	499 (82)	78 (13)	32 (5)	609
1955 - < 1960	379 (86)	29 (7)	32 (7)	440
1960 - < 1965	317 (90)	21 (6)	15 (4)	353
Total	2,282 (68.5)	861 (25.9)	185 (5.6)	3,328

Table 11. Distribution of workers by date first employed and length of employment in underground jobs among the Homestake Cohort.

Date First Employed	Length of Underground Employment										
	1 < 2	2 < 3	3 < 4	4 < 5	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	> 30	Total (%)
< 1920	1	0	0	2	2	4	10	19	27	23	88 (3)
≥ 1920 - 1924	0	1	0	1	15	18	45	19	12	9	120 (4)
≥ 1925 - 1929	0	3	2	1	38	111	47	22	9	11	244 (7)
≥1930 - 1934	2	3	2	2	125	80	19	11	6	18	268 (8)
≥1935 - 1939	13	27	57	57	82	15	6	7	8	9	281 (8)
≥1940 - 1944	96	24	13	9	30	10	5	12	8	3	210 (6)
≥1945 - 1949	283	126	51	30	78	30	25	34	48	10	715 (22)
≥1950 - 1954	160	76	39	41	98	70	37	71	17	0	609 (18)
≥1955 - 1959	93	60	44	26	94	41	46	36	0	0	440 (13)
≥1960 - 1964	94	61	56	35	58	34	15	0	0	0	353 (11)
Total	742	381	264	204	620	413	255	231	135	83	3328
Percent	(22)	(11)	(8)	(6)	(19)	(12)	(8)	(7)	(4)	(3)	

Table 12. Cause-specific mortality of the Homestake Cohort.

Cause	ICD Codes (7th Revision)	Observed Deaths	Expected Deaths [1]	SMR	95% Confidence Intervals for SMR [2]
Respiratory Tuberculosis	001-008	36	9.9	364	(270 ----)
Other Tuberculosis	010-019	3	0.6	500	- - - - -
All Malignant Neoplasms	140-205	137	141.0	97	(82 - 115)
MN of Buccal Cavity and Pharynx	140-148	6	4.8	125	(46 - 272)
MN of Esophagus	150	1	3.3	30	- - - - -
MN of Stomach	151	5	8.8	57	(18 - 133)
MN of Intestine	152,153	10	12.6	79	(38 - 146)
MN of Rectum	154	6	5.0	120	(44 - 261)
MN of Biliary Passages and Liver	155	3	2.5	120	- - - - -
MN of Pancreas	157	8	7.8	103	(44 - 202)
MN of Peritoneum and Unspecified Digestive Organs	158,159	2	0.7	286	- - - - -
MN of Larynx	161	2	2.2	91	- - - - -
MN of Trachea, Bronchus, and Lung	162-163	43	42.9	100	(73 - 135)
MN of Other Parts of Respiratory System	160,164	3	0.6	500	- - - - -
MN of Prostate	177	11	7.7	143	(72 - 259)
MN of Other Male Genital Organs	178-179	2	1.1	182	- - - - -
MN of Urinary Organs	180-181	2	7.7	26	- - - - -
MN of Other and Unspecified Major Sites	156B,165,190-199	14	17.7	79	(43 - 133)
Lymphosarcoma and Reticulosarcoma	200	6	4.0	150	(55 - 326)
Hodgkin's Disease	201	2	2.1	95	- - - - -
Leukemia and Aleukemia	204	10	5.9	169	(81 - 312)
Other Neoplasms of Lymphatic and Hematopoietic Tissues	202,203,205	1	2.0	50	- - - - -
Neoplasms of Brain	223,237	3	1.6	188	- - - - -
Diabetes Mellitus	260	8	10.9	73	(32 - 146)

Table 12. Cause-specific mortality of the Homestake Cohort.

Cause	ICD Codes (7th Revision)	Observed Deaths	Expected Deaths [1]	SMR	95% Confidence Intervals for SMR [2]
Diseases of Blood and Blood-Forming Organs	290-299	4	2.0	200	- - - - -
Mental, Psychoneurotic and Personality Disorders	300-326	4	4.4	91	- - - - -
Diseases of Nervous System	330-334,345	41	50.5	81	(58 - 110)
Diseases of Circulatory System	400-468	285	337.3	84	(75 - 95)
Influenza	480-483	2	1.6	125	- - - - -
Pneumonia (Except Newborn)	490-493	16	16.7	96	(55 - 156)
Bronchitis	500-502	2	2.5	80	- - - - -
Other Respiratory Diseases	510-527	53	19.0	279	(219 ----)
Diseases of Stomach and Duodenum	540,541,543	7	6.9	101	(41 - 209)
Hernia and Intestinal Obstruction	560,561,570	6	3.0	200	(73 - 435)
Cirrhosis of Liver	581	15	21.0	71	(40 - 118)
Chronic and Unspecified Nephritis; Other Renal Sclerosis	592-594	8	5.0	160	(69 - 315)
Diseases of Skin	690-716	2	0.6	333	- - - - -
Diseases of Bones and Organs of Movement	720-730	2	0.7	286	- - - - -
Unknown Causes (Blank)	780-793,795,blank	63	--	--	- - - - -
Transportation Accidents	800-866	35	33.7	104	(72 - 144)
Accidental Poisonings	870-895	2	2.4	83	- - - - -
Accidental Falls	900-904	11	6.7	164	(82 - 294)
Other Accidents	910-936,960-962	58	17.5	331	(263 ----)
Violence	963-964,970-985	18	25.9	69	(41 - 110)
All Others		40	35.8	--	- - - - -
All Deaths		861	765.8	112	(105- 120)

[1] Expected deaths based on person-years calculated in the length of employment analysis refer to Table 9.

[2] Confidence intervals were not calculated if observed deaths were less than five.

Table 13. Lung cancer mortality by latency and length of employment underground for Homestake workers.

Latency (Years)	Length of Employment (Years)							
	1 - 4	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	≥ 30	Total
< 15								
O/E	0/ 1.62	1/ 0.78	1/0.51					2/ 2.91
SMR	--	128	196					68
15 - 19								
O/E	1/ 1.92	1/ 0.79	0/0.61	1/0.67				3/ 3.99
SMR	52	127	--	149				75
20 - 24								
O/E	5/ 3.11	0/ 1.18	0/0.69	2/0.62	0/0.70			7/ 6.30
SMR	161	--	--	323	--			111
25 - 29								
O/E	1/ 3.33	1/ 1.47	0/0.85	0/0.44	0/0.65	0/0.40		2/ 7.14
SMR	30	68	--	--	--	--		28
30 - 34								
O/E	3/ 2.28	1/ 1.78	3/1.20	1/0.50	1/0.46	0/0.50	0/0.15	9/ 6.87
SMR	132	56	250	200	217	--	--	131
35 - 39								
O/E	1/ 1.43	4/ 2.00	3/1.52	2/0.66	0/0.41	1/0.37	0/0.42	11/ 6.81
SMR	70	200	197	303	--	270	--	162
> 40								
O/E	1/ 0.26	1/ 2.07	4/2.66	2/1.57	0/0.85	0/0.57	1/0.88	9/ 8.86
SMR	385	48	150	127	--	--	115	102
Total								
O/E	12/13.95	9/10.07	11/8.04	8/4.46	1/3.07	1/1.84	1/1.45	43/42.88
SMR	86	89	137	179	33	54	69	100

Table 14. Lung cancer mortality by latency and dose for Homestake workers.

Latency (Years)	Dose (Dust-Days)							
	20000	20000-40000	40000-80000	80000-160000	160000-320000	320000-640000	≥ 640000	Total
< 15								
O/E	0/0.47	0/0.53	1/0.80	1/0.67	0/0.32	0/0.11	0/0	2/2.91
SMR	--	--	125	149	--	--		69
15-19								
O/E	0/0.46	0/0.58	2/0.93	0/0.96	1/0.68	0/0.40	0/0	3/4.01
SMR	--	--	215	--	147	--	--	75
20-24								
O/E	1/0.51	1/0.87	3/1.38	0/1.24	1/1.35	1/0.91	0/0.07	7/6.33
SMR	196	108	217	--	74	110	--	111
25-29								
O/E	0/0.11	0/0.81	1/1.39	1/1.39	0/1.68	0/1.59	0/0.25	2/7.22
SMR	--	--	72	72	--	--	--	28
30-34								
O/E	0/0.01	0/0.34	0/0.79	2/1.14	2/1.82	4/2.30	1/0.51	9/6.91
SMR	--	--	--	175	110	174	--	131
35-39								
O/E	0/0.01	0/0.08	1/0.45	0/0.85	2/1.85	8/2.87	0/0.72	11/6.83
SMR	--	--	222	--	108	279	--	161
≥ 40								
O/E		0/0.01	1/0.06	0/0.29	1/1.68	6/5.07	1/1.77	9/8.88
SMR	--	--	1667	--	60	118	56	101
Total								
O/E	1/1.57	2/3.22	9/5.80	4/6.54	7/9.38	19/13.25	2/3.32	43/43.10
SMR	63	62	155	61	75	143	60	100

Table 15. Nonmalignant respiratory disease mortality by latency and length of employment underground for Homestake workers.

Latency (Years)	Length of Employment (Years)							
	1 - 4	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	≥ 30	Total
< 15								
O/E	1/ 0.63	1/ 0.30	0/0.18					2/ 1.11
SMR	159	333	--					180
15 - 19								
O/E	0/ 0.54	0/ 0.23	0/0.19	0/0.20				0/ 1.16
SMR	--	--	--	--				--
20 - 24								
O/E	1/ 0.94	1/ 0.36	1/0.21	0/0.19	0/0.22			3/ 1.92
SMR	106	278	476	--	--			156
25 - 29								
O/E	2/ 1.20	1/ 0.55	0/0.28	4/0.14	1/0.22	1/0.14		9/ 2.53
SMR	167	182	--	2857	455	714		356
30 - 34								
O/E	1/ 1.02	0/ 0.83	2/0.50	1/0.19	2/0.19	3/0.20	0/0.05	9/ 2.98
SMR	98	--	400	526	1053	1500	--	302
35 - 39								
O/E	1/ 0.75	2/ 1.08	2/0.79	2/0.32	1/0.22	1/0.18	1/0.18	10/ 3.52
SMR	133	185	253	625	455	556	556	284
> 40								
O/E	0/ 0.16	3/ 1.28	4/1.71	4/1.05	1/0.63	5/0.38	3/0.57	20/ 5.78
SMR	--	234	234	381	159	1316	526	346
Total								
O/E	6/ 5.24	8/ 4.63	9/3.86	11/2.09	5/1.48	10/0.90	4/0.80	53/19.00
SMR	115	173	233	526	338	1111	500	279

Table 16. Nonmalignant respiratory disease mortality by latency and dose for Homestake workers.

Latency (Years)	Dose (Dust-Days)							
	20000	20000-40000	40000-80000	80000-160000	160000-320000	320000-640000	≥ 640000	Total
< 15								
O/E	0/0.17	0/0.20	2/0.30	0/0.24	0/0.14	0/0.06	0/0	2/1.11
SMR	--	--	667	--	--	--	--	180
15-19								
O/E	0/0.12	0/0.16	0/0.26	0/0.27	0/0.20	0/0.15	0/0	0/1.16
SMR	--	--	--	--	--	--	--	--
20-24								
O/E	0/0.14	0/0.25	1/0.41	2/0.39	0/0.42	0/0.29	0/0.02	3/1.92
SMR	--	--	--	513	--	--	--	156
25-29								
O/E	0/0.03	0/0.26	1/0.46	2/0.54	1/0.62	3/0.55	2/0.08	9/2.52
SMR	--	--	--	370	161	545	2500	357
30-34								
O/E	0/0	0/0.13	0/0.32	1/0.54	0/0.84	5/0.96	3/0.18	9/2.97
SMR	--	--	--	185	--	521	1667	303
35-39								
O/E	0/0	0/0.03	0/0.23	2/0.45	2/1.00	4/1.47	2/0.32	10/3.50
SMR	--	--	--	444	200	272	625	286
≥ 40								
O/E	0/0	0/0	0/0.04	0/0.18	2/1.04	9/3.28	9/1.21	20/5.76
SMR	--	--	--	--	192	274	744	347
Total								
O/E	0/0.46	0/1.03	4/2.01	7/2.61	5/4.26	21/6.76	16/1.82	53/18.95
SMR	--	--	199	268	117	311	879	280

Table 17. Respiratory tuberculosis mortality by latency and length of employment underground for Homestake workers.

Latency (Years)	Length of Employment (Years)							
	1 - 4	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	≥ 30	Total
< 15								
O/E	0/ 1.98	0/ 1.10	0/0.49					0/ 3.57
SMR	--	--	--					--
15 - 19								
O/E	0/ 0.34	1/ 0.43	2/0.63	0/0.32				3/ 1.72
SMR	--	233	317	--				174
20 - 24								
O/E	0/ 0.26	0/ 0.31	0/0.39	3/0.39	1/0.20			4/ 1.55
SMR	--	--	--	769	500			258
25 - 29								
O/E	1/ 0.17	0/ 0.22	0/0.25	1/0.20	4/0.22	0/0.12		6/ 1.18
SMR	588	--	--	500	1818	--		508
30 - 34								
O/E	0/ 0.08	0/ 0.15	1/0.18	2/0.13	1/0.13	2/0.14	0/0.07	6/ 0.88
SMR	--	--	556	1538	769	1429	--	682
35 - 39								
O/E	0/ 0.04	1/ 0.09	2/0.11	1/0.08	4/0.07	0/0.08	3/0.08	11/ 0.55
SMR	--	1111	1818	1250	5714	--	3750	2000
≥ 40								
O/E	0/ 0.01	0/ 0.06	0/0.09	3/0.08	0/0.07	3/0.06	0/0.09	6/ 0.46
SMR	--	--	--	3750	--	5000	--	1304
Total								
O/E	1/ 2.88	2/ 2.36	5/2.14	10/1.20	10/0.69	5/0.40	3/0.24	36/ 9.91
SMR	35	85	234	833	1449	1250	1250	363

Table 18. Respiratory Tuberculosis mortality by latency and dose for Homestake workers.

Latency (Years)	Dose (Dust-Days)							
	20000	20000-40000	40000-80000	80000-160000	160000-320000	320000-640000	≥ 640000	Total
< 15								
O/E	0/0.15	0/0.42	0/0.73	0/0.82	0/0.92	0/0.56	0/0	0/3.60
SMR	--	--	--	--	--	--	--	--
15-19								
O/E	0/0.03	0/0.06	0/0.12	0/0.19	1/0.39	2/0.94	0/0.01	3/1.72
SMR	--	--	--	--	256	213	--	174
20-24								
O/E	0/0.01	0/0.04	0/0.09	0/0.14	0/0.28	3/0.85	1/0.12	4/1.54
SMR	--	--	--	--	--	353	833	260
25-29								
O/E	0/0	0/0.02	1/0.06	0/0.09	0/0.20	4/0.57	1/0.23	6/1.18
SMR	--	--	1667	--	--	702	435	508
30-34								
O/E	0/0	0/0.01	0/0.02	0/0.05	0/0.13	4/0.38	2/0.28	6/0.88
SMR	--	--	--	--	--	1053	714	682
35-39								
O/E	0/0	0/0	0/0.01	0/0.02	1/0.08	4/0.25	6/0.18	11/0.55
SMR	--	--	--	--	1250	1600	3333	2000
≥ 40								
O/E	0/0	0/0	0/0	0/0.01	0/0.05	3/0.22	3/0.18	6/0.46
SMR	--	--	--	--	--	1364	1667	1304
Total								
O/E	0/0.19	0/0.55	1/1.03	0/1.32	2/2.05	20/3.77	13/1.00	36/9.93
SMR	--	--	96	--	98	531	1300	362

Table 19. Nonmalignant respiratory disease and respiratory tuberculosis mortality by latency and length of employment underground for Homestake workers.

Latency (Years)	Length of Employment (Years)							
	1 - 4	5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	≥ 30	Total
< 15								
O/E	1/ 2.61	1/ 1.40	0/0.67					2/ 4.68
SMR	38	71	--					43
15 - 19								
O/E	0/ 0.88	1/ 0.66	2/0.82	0/0.52				3/ 2.88
SMR	--	152	244	--				104
20 - 24								
O/E	1/ 1.20	1/ 0.67	1/0.60	3/0.58	1/0.42			7/ 3.47
SMR	83	149	167	517	238			202
25 - 29								
O/E	3/ 1.37	1/ 0.77	0/0.53	5/0.34	5/0.44	1/0.26		15/ 3.71
SMR	219	130	--	1471	1136	385		404
30 - 34								
O/E	1/ 1.10	0/ 0.98	3/0.68	3/0.32	3/0.32	5/0.34	0/0.12	15/ 3.86
SMR	91	--	441	938	938	1471	--	389
35 - 39								
O/E	1/ 0.79	3/ 1.17	4/0.90	3/0.40	5/0.29	1/0.26	4/0.26	21/ 4.07
SMR	127	256	444	750	1724	385	1538	516
≥ 40								
O/E	0/ 0.17	3/ 1.34	4/1.80	7/1.13	1/0.70	8/0.44	3/0.66	26/ 6.24
SMR	--	224	222	619	143	1818	455	417
Total								
O/E	7/ 8.12	10/ 6.99	14/6.00	21/3.29	15/2.17	15/1.30	7/1.04	89/28.91
SMR	86	143	233	638	691	1154	673	308

Table 20. Nonmalignant respiratory disease and respiratory tuberculosis mortality by latency and dose for Homestake workers.

Latency (Years)	Dose (Dust-Days)							
	20000	20000-40000	40000-80000	80000-160000	160000-320000	320000-640000	≥ 640000	Total
< 15								
O/E	0/0.32	0/0.62	2/1.03	0/1.06	0/1.06	0/0.62	0/0	2/4.71
SMR	--	--	194	--	--	--	--	42
15-19								
O/E	0/0.15	0/0.22	0/0.38	0/0.46	1/0.59	2/1.09	0/0	3/2.88
SMR	--	--	--	--	169	183	--	104
20-24								
O/E	0/0.15	0/0.29	1/0.50	2/0.53	0/0.70	3/1.14	1/0.14	7/3.46
SMR	--	--	200	377	--	263	714	202
25-29								
O/E	0/0.03	0/0.28	2/0.52	2/0.63	1/0.82	7/1.12	3/0.31	15/3.70
SMR	--	--	385	317	122	625	968	405
30-34								
O/E	0/0	0/0.14	0/0.34	1/0.59	0/0.97	9/1.34	5/6.46	15/3.85
SMR	--	--	--	169	--	672	1087	390
35-39								
O/E	0/0	0/0.03	0/0.24	2/0.47	3/1.08	8/1.72	8/0.50	21/4.05
SMR	--	--	--	426	278	465	1600	519
≥ 40								
O/E	0/0	0/0.02	0/0.04	0/0.19	2/1.09	12/3.50	12/1.39	26/6.22
SMR	--	--	--	--	184	343	863	418
Total								
O/E	0/0.65	0/1.60	5/3.05	7/3.93	7/6.31	41/10.53	29/2.80	89/28.87
SMR	--	--	164	178	111	389	1036	308

Table 21. Mortality according to subcohorts by year first employed in underground jobs at Homestake.

Cause of Death	Year First Employed Underground								
	Before 1930	1930-34	1935-39	1940-44	1945-49	1950-54	1955-59	1960-64	After 1951
Respiratory Tuberculosis (RTB)									
O/E	32/ 4.04	2/ 1.99	0/ 0.10	0/ 0.05	2/ 0.92	0/ 0.36	0/ 0.11	0/ 0.03	0/ 0.40
SMR	792	101	--	--	217	--	--	--	--
"Other" Non-Malignant Respiratory Diseases (NMRD)									
O/E	33/ 6.47	5/ 3.89	6/ 3.40	3/ 1.46	2/ 2.11	3/ 1.12	1/ 0.40	0/ 0.17	4/ 1.40
SMR	510	129	176	205	95	268	250	--	286
Lung Cancer									
O/E	15/ 12.02	8/ 7.72	6/ 7.15	4/ 3.64	6/ 6.66	3/ 3.84	1/ 1.45	0/ 0.41	3/ 4.76
SMR	125	104	84	110	90	78	69	--	63
Diseases of the Circulatory System									
O/E	117/118.90	50/ 60.85	30/ 53.21	20/24.37	38/ 42.16	21/24.16	6/ 9.24	3/ 4.16	26/31.66
SMR	98	82	56	82	90	87	65	72	63
"Other" Accidents									
O/E	9/ 3.21	5/ 2.12	2/ 2.26	3/ 1.43	15/ 3.72	12/ 2.53	9/ 1.44	3/ 0.83	21/ 4.23
SMR	280	236	88	210	403	474	625	361	496
All Malignant Neoplasms									
O/E	51/ 44.90	25/ 24.92	18/ 22.37	11/10.83	17/ 19.77	12/11.61	1/ 4.73	2/ 1.85	14/15.39
SMR	114	100	80	102	86	103	21	108	91
All Deaths									
O/E	335/247.89	134/130.27	96/116.65	55/56.10	113/106.46	78/64.50	29/28.86	21/14.67	109/92.66
SMR	135	103	82	98	106	121	100	143	118

Table 22. Mortality by year of death among Homestake workers.

Cause of Death	Year of Death							
	1941-44	1945-49	1950-54	1955-59	1960-64	1965-69	1970-74	1975-77
Respiratory Tuberculosis (RTB)								
O/E	2/ 2.44	11/ 2.70	4/ 1.78	9/ 1.11	7/ 0.88	2/ 0.68	0/ 0.41	1/ 0.16
SMR	89	407	225	811	795	294	--	625
"Other" Non-Malignant Respiratory Diseases (NMRD)								
O/E	1/ 0.27	4/ 0.34	3/ 0.63	6/ 1.22	10/ 2.31	13/ 4.21	9/ 6.22	7/ 3.83
SMR	370	1176	476	492	433	309	145	183
Lung Cancer								
O/E	0/ 0.40	0/ 0.99	0/ 1.94	3/ 3.52	10/ 5.67	10/ 9.13	15/ 13.30	5/ 7.97
SMR	--	--	--	85	176	110	113	63
Diseases of the Circulatory System								
O/E	3/ 8.57	9/15.89	32/25.04	31/36.29	41/ 50.68	65/ 69.70	68/ 85.72	36/45.58
SMR	35	57	128	85	81	93	79	79
"Other" Accidents								
O/E	10/ 1.38	8/ 1.75	7/ 2.09	8/ 2.48	11/ 2.72	6/ 3.08	8/ 2.82	0/ 1.29
SMR	725	457	335	323	404	195	284	--
All Malignant Neoplasms								
O/E	1/ 2.90	3/ 5.64	13/ 9.43	10/14.28	23/ 20.11	25/ 28.72	43/ 37.97	19/99.55
SMR	34	53	138	70	114	87	113	86
All Deaths								
O/E	30/27.27	60/42.45	81/61.08	109/83.92	132/112.56	164/153.10	189/186.73	96/99.55
SMR	110	141	133	130	117	107	101	96

Table 23. Mortality from chronic renal disease by length of underground employment among Homestake workers.

Length of Underground Employment (Years)	Observed Deaths	Expected Deaths	SMR
1 - 4	1	1.72	58
5 - 9	1	1.09	92
10 - 14	2	0.92	217
15 - 19	1	0.55	182
20 - 24	1	0.35	286
25 - 29	1	0.23	435
$\geq$ 30	1	0.18	556
Total	8	5.04	159

Table 24. Mortality from chornic renal disease by dose among Homestake workers.

Dose (Total Dust-Days)	Observed Deaths	Expected Deaths	SMR
< 20000	0	0.28	--
20000 - 40000	0	0.42	--
40000 - 80000	1	0.66	152
80000 - 160000	0	0.68	--
160000 - 320000	1	0.91	110
320000 - 640000	4	1.56	256
$\geq$ 640000	2	0.54	370
Total	8	5.04	159

Table 25. Description of deaths due to malignant neoplasms (MN) of peritoneum (and unspecified digestive organs) and of other parts of the respiratory system in the Homestake Study.

Case	Underlying Cause of Death Coded from Death Certificate	Date of Death	Age at Death	Approximate Length of Underground Employment	Latency at Time of Death	Hospital Report
1	MN of Peritoneum (158.9)	6/20/75	70	17 years	51 years	Not Obtained
2	MN of Unspecified Digestive Organs (159)	4/05/75	85	16 years	50 years	Pulmonary Edema Bilateral Pleural Effusions - Metastatic Adenocarcinoma
3	MN of Respiratory System Other Than Trachea, Bronchus & Lung - Maxiliary Sinus (160.2)	12/07/73	47	25 years	26 years	Adenoid Cystic Carcinoma of Left Antrum with Metastatis
4	MN of Respiratory System Other Than Trachea, Bronchus & Lung - Mediastinum (163.1)	1/10/72	57	14 years	20 years	Mediastinal Mass on Chest
5	MN of Respiratory System Other Than Trachea, Bronchus & Lung - Mediastinum (164)	7/25/62	58	9 years	29 years	Not Obtained