MAR

SM-224/509

SM-224/509

IAEA-SM-224

CANCER MORTALITY IN HANFORD WORKERS

SIDNEY MARKS, ETHEL S. GILBERT AND BRYCE D. BREITENSTEIN+

Battelle
Pacific Northwest Laboratory
Richland, Washington 99352 USA
+Hanford Environmental Health Foundation
Richland, Washington 99352 USA

ABSTRACT

Personnel and radiation exposure data for past and present employees of the Hanford plant have been collected and analyzed for a possible relationship of exposure to mortality. The occurrence of death in workers was established by the Social Security Administration and the cause of death obtained from death certificates. Mortality from all causes, all cancer cases and specific cancer types was related to the population at risk. Standardized mortality ratios were calculated for white males, using age- and calendar year-specific mortality rates for the U.S. population in the calculation of expected deaths. This analysis showed a substantial "healthy worker effect" and no significantly high standardized mortality ratios for specific disease categories. A test for association of mortality with levels of radiation exposure revealed no correlation for all

---

This paper is based on work performed by Battelle, Pacific Northwest Laboratories, for the U.S. Department of Energy under contract E(45-1)-1830.

causes and all cancer. In carrying out this test, adjustment was made for age and calendar year of death, length of employment and occupational category. A statistically significant test for trend was obtained for multiple myeloma and carcinoma of the pancreas. However, in view of the absence of such a correlation for diseases more commonly associated with radiation exposure such as myeloid leukemia, as well as the small number of deaths in higher exposure groups, the results cannot be considered definitive. Any conclusions based on these associations should be viewed in relation to the results of other studies. We have compared our results with those of other investigators who have analyzed the Hanford data.

INTRODUCTION

A study of the mortality of workers at the Hanford plant, located at Richland in the southeastern part of the State of Washington in the United States, has been in progress since 1964. The initial purpose of the plant was the manufacture, chemical separation and purification of plutonium. In addition, supporting research of a diverse character and, more recently, power generation have been conducted at the facility. The study was initiated by the U.S. Atomic Energy Commission and then sponsored successively by the Energy Research and Development Administration and now the Department of Energy. In addition to the study of Hanford workers, data have been collected at the Oak Ridge, Mound Laboratories and several uranium feed plants for the investigation of the health of workers. As in many occupational epidemiologic studies, mortality was selected as the most reliable and feasible measure of health. The principal investigator for the study was Dr. Thomas F. Mancuso of the University of Pittsburgh Graduate School of Public Health. Data for the Hanford and Oak Ridge plants were collected and processed on site. The Hanford Environmental Health Foundation, the contractor for occupational health services at the Hanford plant, collected data for the Hanford facility.

Hanford mortality data have been analyzed by Dr. Barkev Sanders, who was associated with the study as Dr. Mancuso's statistician from 1964 to 1976 [1-4]; by Dr. Samuel Milham, Jr., of the Department of Social and Health Services of the State of Washington in 1974 [5]; by one of the authors of this paper (E.S.G.) since 1975 at the Pacific Northwest Laboratories (PNL) in Richland (operated for the Department of Energy by Battelle Memorial Institute)[6]; by Drs. Alice Stewart and George Kneale, who have conducted analyses for Dr. Mancuso since 1976 [7]; and by Dr. Charles E. Land since 1976[8]. This report will present the PNL results based on currently available data and will provide comments on the other studies.

EMPLOYEE DATA

Employment and radiation exposure records have been kept on all employees since the plant was built. The employment records include data on age, sex, race, dates of employment and termination, and job classification. In defining a study population, we excluded persons hired after 1965,[1] leaving 20,842 white males, 7,721 white females, 185 nonwhite males and 63 nonwhite females. In the analysis we focused our attention on the largest group, the white males.

Life status was established by a Social Security Administration (SSA) search of their files. The SSA search fails to discover the deaths of a limited number of individuals for whom no death claims are filed. One available estimate of the percentage of deaths missed in this manner is 6% [9]. Death certificates were obtained from the states where deaths occurred, and the cause of death was coded according to the eighth revision of the International Classification of Diseases (ICD). Among deaths reported by the SSA, death certificates were obtained for 96.8% of the white males who died before April 1, 1974, the cutoff date for this study.[2] For men employed two or more years at Hanford, the group of greater interest in the analysis, certificates were obtained for 97.7%

Our analysis is limited to the consideration of exposure to whole body, penetrating radiation. The data, consisting of cumulative annual doses expressed in rem, are obtained primarily from measurements of personnel dosimeters worn by employees. Although internal exposure data have been collected on Hanford employees, the number of cases of internal deposition is too small to influence the analysis. Only about 450 verified cases of internal deposition have occurred in the entire employee population, including all survivors.

The distribution of cumulative doses through 1973 is presented in Figure 1. The distribution is highly skewed because the predominant exposure of most Hanford employees is to natural background radiation (typically about .1 rem per year), while only a smaller number (perhaps 5 to 10% of the work force) receive annual whole body doses in excess of 1 rem with a very small number of those exceeding 4 rem per year. Because of the skewed nature of the distribution and the lesser likelihood of demonstrating effects at low levels, we have chosen to present many of

---

[1]This eliminated only 20 white male deaths for which we had death certificates; of those only one had a cumulative radiation exposure greater than 1 rem.

[2]Data on deaths occurring after April 1, 1974 are not yet available.

our results in terms of the number (or percent) of workers with doses exceeding 5 rem. In general, cumulative radiation exposure is correlated with length of employment. For example, a breakdown by duration of employment discloses that radiation doses of 5 rem or more were accumulated by 2 of 7767 (.03%) workers employed <2 years, by 339 of 5470 (6.2%) employed 2-9 years, by 998 of 3353 (25.9%) employed 9-19 years and by 1439 of 3752 (38.4%) employed 20 or more years.

METHODS OF ANALYSIS

In view of the availability of a defined population base to which the deaths due to any specific cause can be related, the analysis included the calculation of population-based mortality ratios and testing for a possible correlation between mortality and the level of radiation exposure. In epidemiologic terms, this study is of a cohort or prospective type despite the fact that much of the data were collected from past records. When data for the population at risk are available, this method is usually preferred to a retrospective or case-control approach, especially if quantitative estimates of risk are desired [10].

We calculated standardized mortality ratios (SMRs), which provide in percentage form the ratio of the number of deaths observed to that expected in the same population or subgroup (100 x observed deaths ÷ expected deaths). Expected deaths, corrected for age and calendar year of death, were based on U.S. vital statistics for deaths occurring in the same five-year age and calendar year group. In the application of this method, the years of observation are allocated to the appropriate categories as in the following example. A person who initiates employment on his 26th birthday at the beginning of 1953 and survives until April 1974 (the cut-off date for the study) will have 2 years allocated to the age 25-29, year 1950-54 category; 2 years to the age 25-29, year 1955-59 category; 3 years to the age 30-34, year 1955-59 category; and so forth. For any disease, the total person-years in each category in the study population are then multiplied by the appropriate age-calendar year-specific mortality rates for U.S. white males, and the results are summed over age-calendar year categories to obtain the expected number of deaths due to that cause. The SMRs are calculated in this manner by means of a computer program developed by Monson [11], which provides SMRs for 23 categories of cancer and 34 other disease categories. The cause-specific SMRs are corrected for the 2-4% of deaths with no certificates on the reasonable assumption that the distribution of causes is similar for identified deaths with and without death certificates. Statistical significance of the SMRs was tested by a continuity corrected chi-square test.

The interpretation of SMRs is conditioned by the "healthy worker effect," which is the reduction in the values of SMRs that is observed frequently in the case of workers in industries free of serious life threatening hazards [12]. The health of employees may be favorably influenced by pre-employment screening, health insurance and medical surveillance programs and by the socioeconomic benefits of steady employment. For these reasons, SMRs below 100 are not interpreted to signify protective effects of exposure factors. On the other hand, low SMRs are not compatible with important adverse effects but, instead, may reflect a favorable health experience under the conditions of employment. More importantly, in our results as in other studies, the magnitude of the healthy worker effect varies with the disease category. The SMRs are likely to be higher for cancer than for most other diseases because the factors responsible for the healthy worker effect are less likely to be effective for most cancer types than for other causes of death, such as cardiovascular disease [13]. This differential effect tends to bias proportional mortality analyses toward falsely indicating an excess of cancer [14].

In our analysis for a possible relationship of radiation exposure to mortality rates, we categorized the workers with respect to their cumulative exposure at a given time and then compared the subsequent mortality experience of the various exposure groups. Only past exposure to radiation is included for a stated time since future exposure would be correlated with survival. Four groups were selected with arbitrary cutpoints at <2, 2-5, 5-15 and >15 rem. Since these analyses are concerned with comparisons between workers grouped according to exposure, expected deaths are calculated from the combined experience of the groups under consideration, using the Mantel-Haenzel method [15]. Such expected values should not be confused with those calculated previously on the basis of U.S. vital statistics mortality rates. To illustrate this method of calculating expected rates, assume that there are P person-years and d deaths from all causes for a particular age group. If, in exposure category i, there are P_i person-years for that age group, the value for expected deaths in group i will be $\frac{P_i}{P} \times d$. Similar calculations can be made for all age groups and the results summed to obtain the total number of deaths that would be expected in group i if mortality rates are not affected by exposure. The total number of deaths expected for a given exposure category can then be compared with the number actually observed.

The possibility of a relationship between mortality and radiation exposure was analyzed by means of a statistical test for trend. The population defined for this analysis included only white males employed at least two years and excluded those who had terminated employment before January 1, 1960. Of the 2278 men with total doses exceeding 5 rem, only 77 (2.8%) fail to meet these criteria while only 3 (0.2%) of the 1211 men with total doses exceeding 15 rem are excluded from this group. The population was grouped according to exposure categories of <2, 2-5, 5-15 and >15 rem. In order to avoid biases in the comparison, the groups were adjusted for age (in 5 year intervals), occupational cateogry (craftsmen and operators vs. others) and calendar year combined with employment status in three strata.[3] Mantel's single degree of freedom chi-square test for trend, which accounts for the influence of the above factors, was used [16]. The test requires the assignment of scores to each group; we selected the median of each of the four groups as its score, i.e., 0.80, 3.21, 7.85 and 21.32. For a few causes of death, an exact permutation test was used in place of the chi-square because of the small number of deaths and the severely skewed nature of the distribution of radiation exposures in the worker population.

RESULTS

Standardized mortality ratios are presented for a variety of causes of death for white males grouped according to length of employment (<2, 2+ years) in Tables I and II. The reader may wish to adjust the SMRs for missing deaths by adding an increment of 6% in accordance with the estimate cited above. The SMR for deaths from all causes among workers employed at least two years is 75, which may be interpreted to mean that the number of observed deaths is 75% of that expected on the basis of age-calendar year-specific rates for U.S. white males. In the case of workers employed less than two years, the SMR is 86. These low values are compatible with the healthy worker effect discussed above [13]. The SMRs for all cancer cases are 85 for 2+ years and 88 for <2 years, which are greater than the SMRs for all causes. The less marked healthy worker effect for cancer than for other diseases in those employed at least two years is attributed to a lesser impact of the benefits of employee selection and prolonged employment on cancer than on most other diseases as

---

[3]Stratum 1 - employed 2 years and working on January 1, 1960; stratum 2 - those of stratum 1 who terminate employment before January 1, 1965 but are alive on that date; stratum 3 - employed 2 years and working on January 1, 1965 (mostly derived from stratum 1). The three strata, which are not mutually exclusive, account for employment period (termination before or after 1965) and time of death (before or after 1965). Cumulative exposure is calculated to 1960 or 1965, depending upon the stratum.

discussed previously. For specific cancer types the distribution of SMRs in Table II is more or less random relative to the reference value of 100; there are more SMRs below than above 100. The only statistically significant SMRs are those for all malignant neoplasms, carcinoma of the lung and leukemia, all of which are low. SMRs below 100 should not be interpreted as evidence that environmental factors such as radiation are protective. It is equally misleading to conclude that SMRs above 100 are representative of radiation effects while those below 100 are due to random variation. Random variation will account for SMRs both above and below 100. In addition, biases must be considered along with causative factors to account for SMRs that are statistically significantly low or high.

Since myeloid leukemia is a cause of death for which an association with radiation exposure has been demonstrated consistently [17, 18], additional detail for this disease and for other neoplasms of lymphatic and hematopoietic tissue is presented in Tables III and IV. Table III shows the relevant cause of death categories for which published U.S. vital statistics are available prior to 1968 while Table IV shows a more detailed breakdown based on vital statistics available since 1968 when the eighth revision of the ICD was implemented. Table IV includes deaths occurring from 1965 to 1974. Only about half the expected number of leukemia deaths are observed in Table III. This deficiency is still present to a lesser extent in Table IV where only experience and deaths from 1965 to 1974 are considered. For the latter period the deficiency in leukemia deaths is confined to the lymphatic type; observed and expected deaths are approximately equal for other types, including myeloid leukemia, which is most likely to be influenced by radiation exposure.

In Table V, the observed and expected deaths for the four exposure categories and results of the test for trend are presented for all causes of death, all cancer, and several specific cancer types. A significant test for trend is determined by a generally increasing ratio of observed over expected deaths with increasing exposure. The categories of all causes and all malignant neoplasms do not show a significant trend with increasing radiation exposure. For all malignant neoplasms 26 deaths are observed in comparison with 29.8 expected. Among the individual cancer types, only multiple myeloma ($p = .01$) and carcinoma of the pancreas ($p = .03$) are statistically significant. The numbers of deaths in the higher exposure categories are small for the individual cancer types. For multiple myeloma, there is 1 observed vs. 0.8 expected in the 5-15 rem group and 2 observed vs. 0.4 expected in the 15+ rem group. For carcinoma of the pancreas, 1 death is observed vs. 2 expected in the 5-15 rem group and 3 observed vs. 1 expected in the 15+ rem group. The highest two exposure groups showed no excess of observed over expected deaths for lung cancer; leukemia and brain are not noteworthy; prostate and the category of other digestive organs are examples of types for which fewer deaths are observed than expected.

As an additional means of summarizing our data, mortality rates for white males, aged 25-70, included in the exposure analysis discussed above, were calculated. The rates, expressed in deaths per 1000 and adjusted for age, occupational category and calendar year, are presented in Table VI. U.S. mortality rates as applied to this population are included for comparison.

DISCUSSION

Neither the SMRs nor the analysis of trend relative to exposure levels indicate an association of radiation with overall mortality or with malignant neoplasms as a group. The only statistically significant tests for trend were obtained with multiple myeloma and carcinoma of the pancreas. However, these diseases are not typically associated with radiation exposure. Myeloid leukemia and carcinoma of the lung, which have been identified as associated with radiation exposure in several studies [17,18], were not present in excess in this population and failed to show a correlation of mortality with level of exposure.

Prior consideration of a relationship of multiple myeloma to radiation exposure rests upon evidence from studies of radiologists and of the Japanese survivors of the atomic bombings. Matanoski et al. have reported higher mortality from multiple myeloma in a particular cohort of radiologists when compared with the control groups of internists, ophthalmologists and otolaryngologists [19] but, on the basis of more recent data, she finds no important difference between radiologists and the ophthalmologists and otolaryngologists [20]. These medical specialists, and internists to a lesser extent, have high relative risks for multiple myeloma, but Matanoski suspects that an unidentified common factor rather than radiation is responsible. In the Japanese atomic bomb survivors, one death due to multiple myeloma was recorded for the group exposed to 100 rad or more and 5 for exposures between 1 and 99 rad [22]. The Japanese results have been variously interpreted as supporting a relationship between radiation and multiple myeloma [22] and, on the other hand, providing no evidence for such a relationship [23].

An excess of cancer of the pancreas as well as excess cancer of the pharynx, esophagus, stomach and large intestine have been described in patients heavily irradiated for the treatment of ankylosing spondylitis [17,23]. The dosimetry to the abdominal organs has not been published yet, but the radiation exposures are known to have been well into the therapeutic range (probably hundreds of rad). In the study of medical specialists by Matanoski, radiologists, internists, ophthalmologists and otolaryngologists had roughly comparable SMRs for carcinoma of the pancreas, which are all less than 100 [20]. Among the Japanese survivors, no excess of pancreatic cancer was detected in the life span study, using

customary follow-up procedures. However, tumor registry data indicated excess mortality in Nagasaki but not in Hiroshima. The report of these findings warned against possibilities of bias in the use of tumor registry data [17]. In general, the epidemiologic study of mortality due to pancreatic cancer may be complicated by variation in the reliability of diagnosis of this disease.

We have not yet had the opportunity to consider the exposure of Hanford workers to agents other than radiation. Some manufacturing, such as chemical processing, and research activities at the plant do involve important exposure to chemicals. Furthermore, the first prime contractor at the plant was a major chemical company, and many employees in the early cohorts had previously worked in chemical plants including munitions factories. The role of chemical exposure warrants further consideration in view of the tentative report of an excess of deaths due to carcinoma of the pancreas and malignant lymphoma in chemists [24].

In considering analyses of the Hanford data by other investigators, the studies by Sanders [1-4] were directed principally to a comparison of longevity among study and control groups and to a possible relationship between all cancer deaths or all deaths from other causes and mean cumulative radiation dose. Sanders found that life span was greater for exposed than unexposed employees and for exposed employees than their siblings. He also concluded that, to date, his analysis of the relationship of radiation exposure to cancer or other mortality did not indicate any adverse effect of radiation on the exposed workers. The analysis by Sanders did not treat specific causes of death in depth.

The study by Milham in 1974 was a proportional mortality analysis of the deaths that occurred between 1950 and 1971 among workers in numerous occupations in the State of Washington and was supplemented by 1972 and 1973 data. His results in summary form for the individual occupations were published in a monograph [5], which included a category of atomic energy workers associated with the Hanford project. Milham found that cancer of the pancreas showed a significant elevation of the proportional mortality ratio (PMR)[4] in men 20 years of age or older while cancer of the large intestine had an elevated PMR in men aged 20-64. Leukemias had low PMRs and multiple myeloma only a small PMR increase based on four deaths. The study by Milham was considered preliminary because information on less than a fourth of the deaths was available to

---

[4]PMR refers to the ratio of the proportion of deaths due to a specified cause or set of causes in the study population to the proportion of deaths due to that cause or set of causes in the reference population (the population of all deaths in the State of Washington for 1950-1971 in this case).

him, and the bias introduced by the marked healthy worker effect for causes other than cancer in this population could seriously affect his proportional analysis.

The study by Stewart and Kneale, conducted for Mancuso in 1976, approached the analysis in several different ways as a result of which they reached striking conclusions [7]. They stated hat at certain ages "there is probably a cancer hazard associated with low level radiation which affects bone marrow cancers more than other neoplasms and cancers of the pancreas and lung more than other solid tumors." In addition, they concluded that 12.2 rad would double the normal risk of dying from any cancer and that the doubling dose for pancreas is 7.4, for lung 6.1 and for reticuloendothelial system or bone marrow cancers 0.8 rad. The authors estimated that 25.8 deaths in the study population were induced by radiation and provided a breakdown of the deaths by cancer type.

In their analyses, Mancuso et al. found a greater percentage of exposed workers among all cancer deaths than the percentage of exposed workers among all noncancer deaths in the total population and concluded that this constitutes evidence of a radiation causation of cancer. As we indicated above, the SMR for all causes other than cancer is lower than the SMR for all cancer in the long-term, predominantly exposed workers but not in the short-term, predominantly unexposed workers. Therefore, diseases other than cancer are associated with a higher percentage of unexposed workers than one would expect if long- and short-term workers had the same distribution of noncancer causes of death. Conversely, cancer as the complement of noncancer is associated with a higher percentage of exposed workers despite similar SMRs for cancer for long- and short-term workers. Thus, a bias in this population due to the differential healthy worker effect in the long-term, exposed workers has led to Mancuso's inference regarding a radiation causation of cancer.

Much of the analysis by Mancuso was concerned with a comparison of mean cumulative radiation exposures for various disease categories. Use of this approach ignores the severely skewed distribution of exposures (Figure 1), which results in the undue influence of single or a few high values when the sample size is small. Mancuso used the t test improperly to test for statistical significance between the means of cumulative exposures for disease categories. The skewed distribution of exposures makes the t test inappropriate when samples are small. Their Monte Carlo simulation of their test of significance for bone marrow neoplasms when compared with their use of a t test increased the p value from $p < .0001$ to $p < .06$ and for carcinoma of the pancreas from $p < .001$ to $p < .01$. The excessive influence of isolated large values also undermines the credibility of their calculation of doubling doses. An additional factor that may influence the length of work and, consequently, level of radiation exposure at various intervals before death is the

self-selection practiced by victims of chronic diseases. They are likely to transfer into less strenuous work assignments or terminate employment earlier than do patients suffering from diseases having a rapid clinical course such as cancer of the lung and pancreas. This factor can bias the mean cumulative exposures used by Mancuso to estimate the magnitude of radiation effect.

Mancuso calculated proportional mortality ratios, which they improperly named standardized mortality ratios. However, these ratios are not adjusted for age and calendar year of death. Furthermore, the authors used as a basis for comparison, proportions based on U.S. vital statistics for 1960, which precedes the period when most Hanford deaths occurred. A substantial increase in mortality from cancer of the lung has occurred as well as nontrivial increases in mortality rates for pancreas and multiple myeloma between 1960 and 1974. The problem of bias due to low noncancer SMRs for this population further weakens the validity of proportional mortality ratios as discussed above.

Mancuso et al. calculated doubling doses that are hardly credible. The unorthodox grouping of myeloid leukemia and multiple myeloma into cancer of bone marrow was assigned a doubling dose of .8 rad; the doubling doses for lung and pancreas were 6.1 and 7.4 rad, respectively. The variation in natural background radiation among the states in the U.S., due to such factors as altitude and terrestrial composition, results in as much as a threefold difference in natural background exposure between populations at sea level and in mountain states [25]. With an estimated difference of 120 mrem per year in background exposure between the State of Colorado and the United States as a whole, several doubling doses for myeloid leukemia and multiple myeloma would be accumulated in Colorado during an average lifetime and at least one doubling dose for pancreas and lung. The average annual age-adjusted white male mortality rates for the period 1950-1969 for carcinoma of the pancreas were 9.23 in Colorado and 9.63 in the U.S.; for lung 28.29 in Colorado and 37.98 in the U.S.; for multiple myeloma 1.75 in Colorado and 1.76 in the U.S.; and for leukemia and aleukemia 8.59 in Colorado and 8.81 in the U.S. These comparisons indicate that at higher altitudes we do not encounter the excess mortality from these diseases that we might expect on the basis of the doubling doses reported by Mancuso.

Land has carried out a refined contingency table analysis of the relationship of radiation exposure to mortality for various causes of death [8]. His method of analysis included adjustment for age and calendar year of death. He demonstrated a statistically significant correlation of carcinoma of the pancreas and multiple myeloma with exposure.

In reviewing the various studies, the results of our population-based analysis and the lack of evidence for a correlation between all cancer deaths and radiation exposure contradict the conclusion by Mancuso et al. that radiation has increased overall cancer mortality in the employee population. Land's study and ours indicate a positive correlation between radiation exposure and mortality from cancer of the pancreas and multiple myeloma. These findings are in agreement with those of Mancuso concerning these particular diseases if one separates myeloid leukemia, for which we established no effect, from multiple myeloma in the broader cateogry "cancer of the bone marrow" used by Mancuso. However, the absence of increased mortality for more typically radiation related cancer types such as leukemia and carcinoma of the lung, as well as the small numbers of cases that determined statistical significance for cancer of the pancreas and multiple myeloma, led us to consider these findings promising leads rather than definitive relationships. Observation and analysis will be continued in the future to check further our current findings and monitor any new developments that might occur in this employee population.

ACKNOWLEDGMENTS

We acknowledge the superb performance of Mrs. Clever Kirklin in collecting and organizing the data file that was used in the study and that made this and other studies of this data possible; K. R. Heid for his excellent cooperation in providing the radiation dosimetry data; Kent B. Stewart and E. L. Kelley for their valuable support in the computer programming; and W. W. Weyzen of the Department of Energy for his encouragement.

REFERENCES

[1] MANCUSO, T.F., SANDERS, B.S., BRODSKY, A., "Study of the Lifetime Health and Mortality Experience of Employees of AEC Contractors," Progress Report Nos. 1-8 (1965-1972).

[2] MANCUSO, T.F., SANDERS, B.S., "Study of the Lifetime Health and Mortality Experience of Employees of AEC Contractors," Progress Report Nos. 9-11 (1973-1975).

[3] MANCUSO, T.F., SANDERS, B.S., BRODSKY, A., "Study of the Lifetime Health and Mortality Experience of Employees of AEC Contractors, Part I: Methodology and Some Preliminary Findings Limited to Mortality for Hanford Employees," (Proc. 6th Ann. Health Phys. Soc. Topical Symposium, 1971) AEC Publication No. COO-3428-1 (1971).

[4] SANDERS, B.S., Low level radiation and cancer deaths, Health Phys. (In press).

[5] MILHAM, S., JR., Occupational Mortality in Washington State, 1950-1971, Vol. I, HEW Publication No. (NIOSH) 76-175-A (1976) 29-30.

[6] GILBERT, E.S., Methods of analyzing mortality of workers exposed to low levels of ionizing radiation, presented at Annual Meeting of the Biometric Society, Western North American Region, in Palo Alto, CA, June 1977.

[7] MANCUSO, T.F., STEWART, A., KNEALE, G., Radiation exposures of Hanford workers dying from cancer and other causes, Health Phys. 33 5 (1977). 369-385.

[8] LAND, C. E., Report to be published (1978).

[9] OTT, M. G., HOLDER, B. B., LANGNER, R. R., Determinants of mortality in an industrial population, J. Occ. Med. 18 3 (1976) 171-177.

[10] FOX, J. P., HALL, C. E., ELVEBACK, L. R., Epidemiology, Collier-Macmillan Ltd., London (1970) 292-309.

[11] MONSON, R. R., Analysis of relative survival and proportional mortality, Comput. Biomed. Res. 7, (1974) 325-332.

[12] McMICHAEL, A. J., HAYNES, S. G., TYROLER, H. A., Observations on the evaluation of occupational mortality data, J. Occup. Med. 17 2 (1975) 128-131.

[13] McMICHAEL, A. J., Standardized mortality ratios and the "healthy worker effect": Scratching beneath the surface, J. Occup. Med. 18 3 (1976) 165-168.

[14] GAFFEY, W. R., Cause-specific mortality, J. Occup. Med. 17 2 (1975) 128.

[15] MANTEL, N., HAENZEL, W., Statistical aspects of the analysis of data from retrospective studies of disease, J. Nat. Cancer Inst. 22 (1959) 719-748.

[16] MANTEL, N., Chi-square tests with one degree of freedom. Extensions of the Mantel-Haenzel procedure, J. Amer. Statistical Ass. 58 (1963) 690-700.

[17] Sources and Effects of Ionizing Radiation: UN Scientific Committee on the Effects of Ionizing Radiation report to the General Assembly, with Annexes, "Radiation carcinogenesis in man," UN, New York (1977) 361-423.

[18] BEEBE, G. W., KATO, H., LAND, C. E., Studies of the mortality of A-bomb survivors. 8. Mortality experience of A-bomb survivors, 1950-74. Radiation Effects Research Foundation, RERF Technical Report 1-77 (1977).

[19] MATANOSKI, G. N., SELTSER, R., SARTWELL, P. E. et al., The current mortality rates of radiologists and other physician specialists: specific causes of death, Amer. J. Epidemiol. 101 3 (1975) 199-210.

[20] MATANOSKI, G. N. Unpublished observations (1978).

[21] NISHIYAMA, H., ANDERSON, R. E., ISHIMARU, T. et al., The incidence of malignant lymphoma and multiple myeloma in Hiroshima and Nagasaki A-bomb Survivors, 1945-1965, Cancer 32 6 (1973) 1301-1309.

[22] JABLON, S., Environmental factors in cancer induction: Appraisal of epidemiologic evidence - leukemia, lymphoma and radiation, Excerpta Medica International Congress Series No. 351, Vol. 3 Cancer epidemiology, environmental factors (Proc. XI Int. Cancer Congress, Florence, 1974) 239-243.

[23] COURT BROWN, W. M., DOLL, R., Mortality from cancer and other causes after radiotherapy for ankylosing spondylitis, Brit. Med. J. 2 (1965) 1327-1332.

[24] LI, F. P., FRAUMENI, J. F., Jr., MANTEL, N., MILLER, R. W., Cancer mortality among chemists, J. Nat. Cancer Inst. 43 5 (1969) 1159-1164.

[25] KLEMENT, A. W., JR., MILLER, C. R., MINX, R. P., SHLEIEN, B., Estimates of Ionizing Radiation in the United States, 1960-2000, U.S. EPA Document ORP/CSD72-1(1972) 3-10.

TABLE I. Observed deaths, expected deaths and Standardized Mortality Ratios (SMRs) for major causes of death in white males grouped by length of employment.

	Length of Employment					
	<2 Years			2+ Years		
Population at Risk	7,767			13,075		
Number with 5+ Rem Cumulative Dose	2			2,778		
Cause of Death	Obs.	Exp.[a]	SMR	Obs.	Exp.	SMR
All Causes	1905	2216.6	86	2089	2796.8	75
All Malignant Neoplasms (140-209)[b]	319	363.0	88	414	487.7	85
Diseases of the Circulatory System (390-429, 440-458)	839	965.4	87	955	1254.2	76
Accidents, Poisonings and Violence (800-999)	243	222.9	109	216	288.8	75
All Other Causes	423	568.1	74	455	700.8	65
No Death Certificate[c]	81			49		

[a]Expected deaths are calculated from age-calendar year specific U.S. mortality rates for white males, 1945-1967.

[b]International Classification of Diseases (ICD) codes.

[c]Expected deaths and SMRs are corrected for those deaths with no certificates on the assumption that the distribution of causes is similar for those with and without certificates.

TABLE II. Observed deaths, expected deaths and Standardized Mortality Ratios (SMRs) for specific cancer types in white males grouped by length of employment.

	Length of Employment					
	<2 Years			2+ Years		
	Obs.	Exp.[a]	SMR	Obs.	Exp.	SMR
All Malignant Neoplasms (M.N.) (140-209)[b]	319	363.0	88	414	487.7	85
M.N. of Buccal Cavity and Pharynx (140-149)	6	12.6	48	18	17.0	106
M.N. of Esophagus (150)	8	8.7	92	11	11.9	93
M.N. of Stomach (151)	17	27.8	61	23	33.3	69
M.N. of Large Intestine (153)	23	33.5	69	44	43.6	101
M.N. of Rectum (154)	6	15.0	40	13	19.0	68
M.N. of Liver (155, 156)	12	10.7	112	7	13.6	51
M.N. of Pancreas (157)	27	20.7	130	28	28.1	100
M.N. of Larynx (161)	5	8.8	57	5	7.9	63
M.N. of Lung (162)	93	101.5	92	115	147.7	78
M.N. of Prostate (185)	24	24.0	100	25	27.8	90
M.N. of Kidney (189.0)	9	9.0	100	13	12.5	104
M.N. of Bladder (188, 189.1-189.9)	5	11.9	42	10	14.9	67
M.N. of Skin (172, 173)	5	6.7	75	8	9.5	84
M.N. of Brain (191, 192)	11	11.5	96	17	16.7	102
M.N. of Thyroid (193)	(2)[c]	(0.8)	250	(0)	(1.3)	0
M.N. of Bone (170)	(0)	(2.5)	0	(2)	(3.1)	65
Leukemia (204-207)	9	16.1	56	10	21.6	46
M.N. of Other Lymphatic and Hematopoietic Tissue (200-203, 208, 209)	18	22.2	81	33	31.5	105
Other M.N. (Residual)	51	44.3	115	47	60.0	78

[a]Expected deaths are calculated from age-calendar year-specific mortality rates for U.S. white males, 1945-1967. For cancer of the lung, leukemia and M.N. of other lymphatic and hematopoietic tissue, U.S. data for 1945-1973 are used.

[b]ICD codes.

[c]Observed or expected death values less than 5 are enclosed in brackets.

TABLE III. Observed deaths, expected deaths and Standardized Mortality Ratios for malignant neoplasms of lymphatic and hematopoietic tissue for white males grouped by length of employment.

	Length of Employment					
	<2 years			2+ years		
	Obs.	Exp.[a]	SMR	Obs.	Exp.	SMR
Lymphosarcoma and reticulosarcoma (200)[b]	6	8.4	71	13	12.4	105
Hodgkin's Disease (201)	6	6.1	98	7	8.1	86
All Leukemia (204-207)	9	16.1	56	10	21.6	46
Other (202, 203, 208, 209)	6	7.7	78	13	11.0	118

[a]Expected deaths are calculated from age-calendar year-specific U.S. mortality rates for white males, 1945-1973.

[b]ICD codes.

TABLE IV. Observed and expected deaths due to specific leukemia types, multiple myeloma and certain other neoplasms of lymphatic and hematopoietic tissue for deaths occurring in white males during and after 1965.

	Length of Employment			
	<2 Years		2+ Years	
	Obs.	Exp.[a]	Obs.	Exp.
All Leukemia	5	7.6	9	11.4
Lymphatic (204)[b]	1	2.4	1	3.4
Myeloid (205)	3	3.3	5	5.1
Monocytic (206)	0	0.4	1	0.6
Other and Unspecified (207)	1	1.5	2	2.3
Certain Other Neoplasms of Lymphatic and Hematopoietic Tissue	4	5.1	10	7.8
Multiple myeloma (203)	4	2.5	5	3.8
Other (202, 208, 209)	0	2.6	5	4.0

[a]Expected deaths are calculated from age-calendar year-specific U.S. mortality rates for white males, 1968-1973.

[b]ICD codes.

TABLE V. Observed and expected deaths due to selected causes by exposure category for white males included in exposure study (see text).

Cause of Death	Exposure Category								Probability of Trend Arising Due to Chance[b]
	0-2 rem		2-5 rem		5-15 rem		15+ rem		
	Obs.	Exp.[a]	Obs.	Exp.[a]	Obs.	Exp.[a]	Obs.	Exp.[a]	
All Causes	587	578.5	122	113.1	84	96.3	44	49.1	>.5
All Malignant Neoplasms (M.N.)	112	117.9	33	23.3	15	19.5	11	10.3	>.5
M.N. of Buccal Cavity	4	3.3	0	.7	1	.6	0	.4	>.5
M.N. of Colon	11	12.5	4	1.8	0	1.2	1	.5	>.5
M.N. of Pancreas	7	8.8	3	2.2	1	2.0	3	1.0	.03
M.N. of Other Digestive Organs	11	10.9	3	1.9	1	1.6	0	.7	>.5
M.N. of Lung	34	39.1	14	8.2	7	7.0	3	3.8	>.5
M.N. of Prostate	10	7.4	1	1.5	0	1.3	0	.9	>.5
M.N. of Brain	4	6.3	3	1.2	1	.9	1	.7	.18
Lymphosarcoma and Reticulum Cell Sarcoma	2	1.5	0	.5	1	.6	0	.4	>.5
Hodgkin's Disease	2	2.1	1	.5	0	.4	0	.0	>.5
Leukemia	3	2.9	0	.6	1	.3	0	.2	>.5
Multiple Myeloma	1	2.3	0	.6	1	.8	2	.4	.01
Other Neoplasms of Lymphatic and Hematopoietic Tissue	2	1.6	0	.3	0	.1	0	.0	>.5
All Other Cancers	21	19.3	4	3.4	1	2.8	1	1.4	>.5
All Noncancer Causes	461	449.2	89	87.3	66	74.9	33	37.6	
No Death Certificate	14	11.4	0	2.5	3	1.9	0	1.2	

[a] Expected deaths are calculated from the experience of all workers in the exposure study, allowing for age, occupation, and follow-up stratum.

[b] The significance levels are for a one-tailed test.

TABLE VI. Age-, calendar year-, and occupation-adjusted mortality rates by exposure category for white males aged 25 to 70 included in exposure study (see text); rates per 1000 person-years (approximate 95% confidence limits are given in parentheses)

	Exposure Category			U.S. white males
	0-2 rem	2-5 rem	5+ rem	
All Causes	7.8 (±.9)	7.5 (±1.4)	6.7 (±1.3)	11.0
All Malignant Neoplasms	1.7 (±.4)	2.1 (±.8)	1.4 (±.6)	2.1

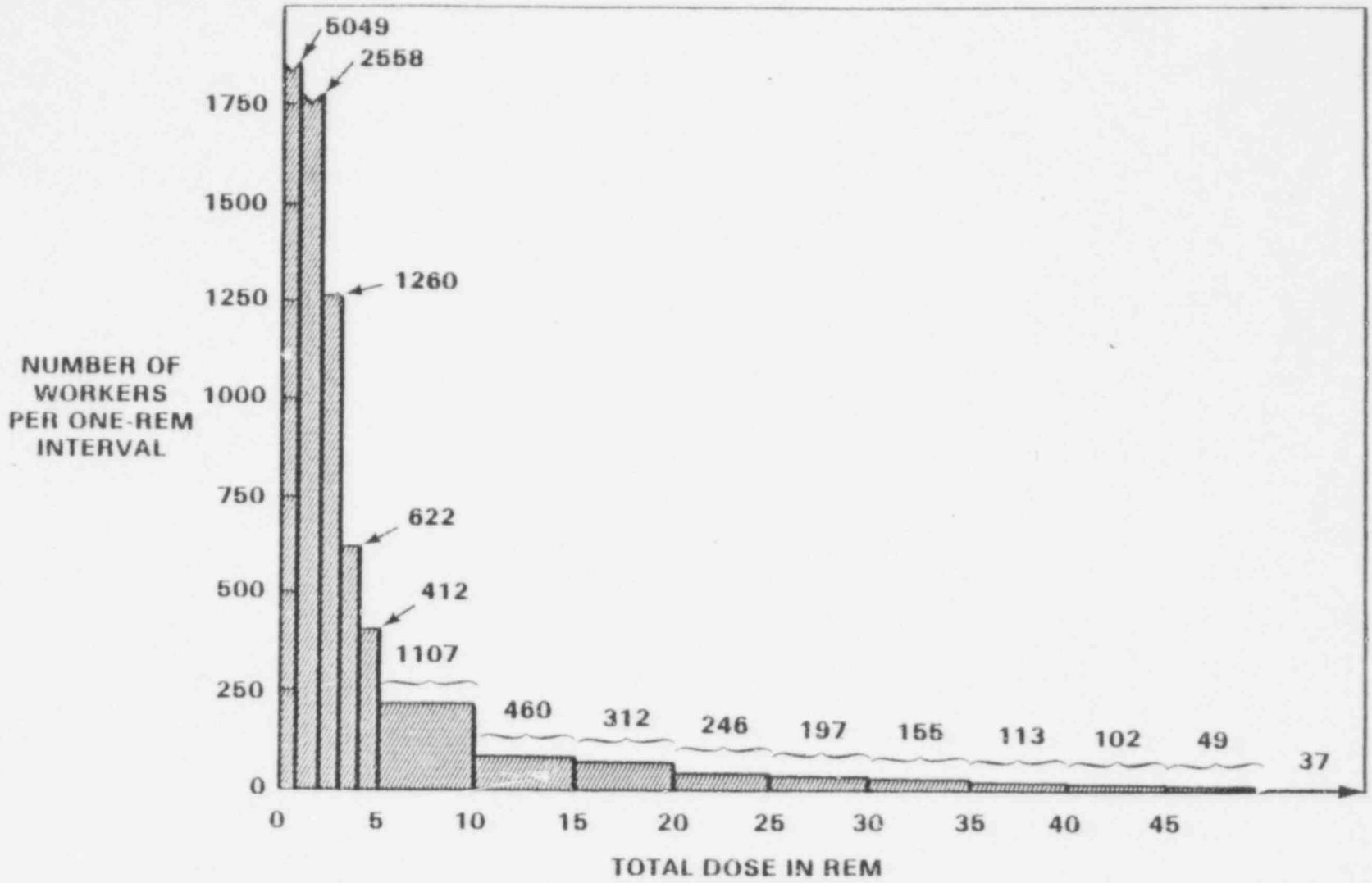

FIG. 1. DISTRIBUTION OF CUMULATIVE DOSES FOR WHITE MALE WORKERS EMPLOYED AT LEAST TWO YEARS

FIGURE

Fig. 1. Distribution of cumulative doses for white male workers employed at least two years