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Methods of Analyzing Mortality of Workers Exposed to Low Levels of Ionizing Radiation

By

Ethel S. Gilbert

Statistics Section
Energy Systems Department
Pacific Northwest Laboratories
Battelle Memorial Institute
Richland, WA 99352

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Introduction

Assessing the health effects of chronic low-level exposures to environmental contaminants poses a number of challenging problems in analysis. Many of these problems are well illustrated by a current study of the effects on mortality of occupational exposure to ionizing radiation at Hanford.

Hanford, which is located in southeastern Washington, was established in the early forties as an installation for plutonium production. Since this time, several thousand workers have been employed by the various contractors, many in jobs involving some exposure to radiation. Yearly records of this exposure, obtained from dosimeter readings, as well as occupational data, are maintained for these employees.

Mortality data are obtained by having the Social Security Administration periodically search their records for deaths of previous Hanford employees. Death certificates are then requested from the states where the deaths occurred, and the cause of death is coded by the Eighth Revision of the International Classification of Diseases [7]. We do not have an accurate estimate of the number of deaths missed by this method, but one study [19] suggests the error is about 6%. Death certificates have been obtained for 96.8% of the white male deaths occurring before April 1, 1974, the termination date of the study. For men employed at least two years at Hanford, the percentage of deaths with certificates is 97.7.

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Comparison With U.S. Mortality

As a first step in analyzing this data, mortality experience of Hanford workers is compared with that of the U.S. population*. Although such an analysis is subject to many limitations [8, 9], it provides a perspective on the magnitude of the death rates involved, and aids in examining a variety of variables that may need to be considered when mortality is later related to exposure status.

The application of this method requires allocating the years each individual is followed to various age-calendar year categories which are defined in five year intervals. The total person-years in each category are then multiplied by the appropriate age-calendar year specific death rates for U.S. white males, and the results summed over categories to obtain the number of deaths that would be expected in the Hanford population if U.S. rates prevailed. The number of deaths actually observed can then be expressed as a percentage of the expected deaths. The percentage (100 x "observed deaths" ÷ "expected deaths") is known as the standardized mortality ratio or SMR.

Dr. Richard Monson, at Harvard, has developed a computer program for carrying out such calculations for 23 categories of cancer and 34 categories of other causes. The method is described more completely in references [16] and [11].

*We considered using statistics for the State of Washington, but they do not appear much more comparable than those for the U.S. since the majority of the Washington population lives in the Puget Sound area, a region quite different from eastern Washington. More localized statistics are unavailable.

Cause specific SMR's are corrected for those deaths with no certificates on the assumption that the distribution of causes is similar for those with and without certificates.

In Table 1, SMR's are presented for a few major cause of death categories. Only white males employed prior to 1965 are included in this analysis with workers employed less than two years considered separately from longer term workers. To avoid bias in these calculations, the first two years experience for both groups are included in the person-years for the short term workers.

The number of observed deaths occurring to Hanford white males employed at least two years is 75% of what would be expected based on age-calendar year specific rates for U.S. white males. This low SMR is fairly typical of an employed population [15] since persons too ill to be employed are of course not included. This selective bias is sometimes refered to as the "healthy worker effect". The health of Hanford employees may be favorably affected by a program of medical surveillance including periodical physical examinations. And, of course, deaths missed by the Social Security Administration will result in underestimation of the SMR's.

A comparison of SMR's for short and long term workers reveals that SMR's for most major non-cancer causes are higher for the short term workers. The SMR's for cancer are nearly identical for the two groups. Many of the workers in the short term group were essentially transient in that they were employed only a few weeks. It seems reasonable that such workers would have less "healthy worker effect" than longer term employees.

McMichael [15] and Enterline [8] have noted that the selective bias involved in the healthy worker effect is generally less for deaths from malignant neoplasms than from other causes such as circulatory and respiratory disease. This is exhibited in the longer term Hanford workers by the relatively high SMR of 85% for malignant neoplasms.

McMichael [15] also demonstrates that the "healthy worker effect" wears off as time passes from the establishment of the employed status and as men more into retirement. This trend is found in the Hanford data as demonstrated in Tables 2 and 3.

In Table 4, SMR's for several specific cancers are presented for the longer term workers. The wide range of SMR's is at least partially due to the small numbers involved. Cancer of the lung and leukemia are the only cancers which are significantly different from 100 at the .05 level. It is particularly interesting that the SMR for leukemia is so low since this is the cancer that has been most strongly associated with radiation exposure in other studies [6].

The above analysis provides no evidence of excess mortality from cancer or any other cause in the Hanford population. However, to adequately investigate the role of radiation exposure, the relationship between mortality and exposure data must be examined at greater lengths.

Exposure Data

Information on radiation exposure is obtained from badge readings, which are recorded by the Personnel Dosimetry Section of Battelle Pacific Northwest Laboratories. This data consists of yearly readings of exposure to several types of ionizing radiation, but only whole body penetrating radiation is considered in this paper. This exposure is measured in rems and includes only ionizing radiation that is able to reach the internal organs.

Of the 13,075 white males employed at least two years at Hanford, 12,678 (97.0%) were monitored and thus have dosimetry data. Figure 1 shows

the distribution of doses accumulated through 1973. The distribution is highly skewed, reflecting the fact that most Hanford employees are subject mainly to natural background radiation (typically about .1 rem per year) while a relatively small number receive occupational exposure on a regular basis (in the range of 1 to 4 rems per year).

Current estimates of health effects from low level exposure to radiation are limited to linear extrapolation from data on single very high level exposures (generally exceeding 100 rems) such as those experienced by atomic bomb survivors at Hiroshima and Nagasaki [2] and by British patients treated with irradiation for ankylosing spondylitis [4]. Our interest is in assessing directly the effects of chronic low level exposures. It is important to note that if the estimates obtained from linear extrapolation are correct, or if they are conservative in the sense of being too high (as they are intended to be), then statistically detectible effects are not likely to be found in the Hanford population. The purpose of the analysis, then, is to investigate the possibility that some phenomenon, not now understood, might cause effects very much larger than those predicted by other studies.

Radiation exposure is correlated with a number of variables that may also affect mortality. Calendar year, in particular, is very highly correlated with exposure--partly because few doses of much magnitude (over 1 rem) were received in the early days at Hanford, and partly because it takes a number of years to accumulate a substantial dose. This is especially important with respect to lung cancer since U.S. rates from this cause have nearly doubled between 1950 and 1970 [17]. Also changes in the character of Hanford as a place to live and work could affect mortality.

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Most of the higher exposures were received by those in a few very specific occupations. There may be other hazards associated with these occupations -- for example, all involve some potential for asbestos exposure. The occupations with a record of high exposure are all classified as craftsmen and operators [1]. It has been demonstrated that occupation and its associated socio-economic status can affect mortality patterns [19]. In the Hanford study, craftsmen and operators have significantly higher SMR's ($p < .05$) for cancer of the lung and prostate than do other workers. As will be seen later, these causes are not correlated with radiation exposure, but these results illustrate the importance of accounting for occupation in the exposure analysis.

Exposure Analysis

The exposure analysis is limited to 7729 white males who were employed at Hanford at least two years and whose employment period extends beyond January 1, 1960. Of the 2778 men with total doses exceeding 5 rems, only 77 (2.8%) fail to meet these criteria while just 3 (.2%) of the 1211 men with total doses exceeding 15 rems are excluded from this group.

The basic method of analysis is to consider all workers employed at a given point in time, categorize them with respect to their accumulated exposure <u>at this point</u>, and then compare the subsequent mortality experience of the various exposure groups. It is necessary to categorize workers only by their past exposure at a given time since exposure yet to come will be positively correlated with survival -- that is the longer one survives the more opportunity one has to accumulate exposure. The method is somewhat analogous to that used by Newhouse [18] in her analysis of asbestos exposure.

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Follow-up interval and calendar year are controlled by the use of three strata. Stratum 1 consists of experience (person-years and deaths) between January 1, 1960 and January 1, 1965 for those men employed at least two years as of January 1, 1960. These workers are categorized with respect to their exposure as of January 1, 1960. If a man terminates employment before January 1, 1965 (but is still alive on this date) his remaining person-years (from January 1, 1965 until death or April 1, 1974) and his death, if it occurs, are counted in Stratum 2.

If a man is still employed as of January 1, 1965, his experience from this date on is counted in Stratum 3. The experience of a worker initiating employment after January 1, 1958 is also counted in Stratum 3 if the worker has been employed at least two years as of January 1, 1965. Workers in Stratum 3 are categorized by their exposure as of January 1, 1965. Note that a man who starts in Stratum 1 and remains employed until 1965 may start in one exposure group and progress to a higher category when he enters Stratum 3.

The four exposure groups considered are < 2 rems, 2-5 rems, 5-15 rems, and 15 or more rems. The Mantel-Haenszel method [12] was used to calculate expected deaths for each exposure category with adjustment for age (in 5-year intervals), for occupation (by separating craftsmen and operators from the remaining occupations), and for follow-up stratum (as described above). The method is adapted to use person-years rather than frequencies as described in [10]. It is important to note that rates for the U.S. population are <u>not</u> used. Instead, the combined experience of all four exposure groups is used to calculate the number of deaths expected in each dose group if mortality rates are not affected by exposure.

A single degree of freedom chi-square was calculated to assess the significance of the trend in the ratio between observed and expected deaths. The test is described by Mantel [13] and takes account of the fact that the expected numbers have been derived from separate calculations for each age group, occupational group, and follow-up stratum.

The test requires assigning scores to each of the four groups. The scores were taken to be the median of each stratum 3 exposure group: .80, 3.21, 7.85, and 21.32 respectively. Observed and expected mean scores are calculated and compared. For example, the observed deaths from all causes for the four exposure categories are 587, 122, 84, and 44 respectively yielding a score of (587 x .80 + 122 x 3.21 + 84 x 7.85 + 44 x 21.32)/837 = 2.94 rems. The corresponding expected deaths are 578.5, 113.1, 96.3 and 49.1 yielding an expected score of (578.5 x .8 + 113.1 x 3.21 + 96.3 x 7.85 + 49.1 x 21.32)/837 = 3.14 rems. Because the data are so skewed (reflected in the high value for the top exposure group), it was sometimes necessary to carry out an exact permutation test rather than to rely on the chi-square approximation proposed by Mantel.

Results and Discussion of Exposure Analysis

Table 5 presents the observed and expected deaths for the four exposure categories, as well as the observed and expected scores used for the test for trend.

Examining first the two highest exposure groups, it is seen that there are less deaths than expected both from all causes and from all malignant neoplasms. There are just two causes for which the observed deaths in the two highest exposure groups exceed the expected by more than a fraction of a death. These are multiple myeloma and cancer of the pancreas. The one-tailed exact permutation test for trend yields a p-value of .01 for

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multiple myeloma and .03 for cancer of the pancreas. To keep this exact test manageable, the test was restricted to craftsmen and operators, a group which includes all deaths from these causes with 5 or more rems of exposure.

The inference for multiple myeloma is based on the deaths of three workers with relatively high exposures. The association between increased mortality from multiple myeloma and the relatively high exposures of the three workers certainly warrants attention. However, other possible occupational factors must be considered as well as the absence of a demonstrable association between radiation exposure and multiple myeloma in studies of Japanese survivors of the atomic bombings [2] and British ankylosing spondylitis patients [4].

The evidence of correlation of radiation exposure and death from cancer of the pancreas is not as strong as for multiple myeloma. When a large number of statistical tests are performed, false positive findings are not unusual. This cancer has not been clearly implicated in other studies of the effects of radiation exposure.

Although the evidence at this time seems insufficient to draw conclusions about a causal association of radiation exposure and multiple myeloma or cancer of the pancreas, certainly it is important to watch these causes especially closely as additional mortality data become available.

In interpreting the above results, it is important to note that since most of the higher exposures have been accumulated relatively late in Hanford's history, it is far too soon to draw firm conclusions about cancers that may take ten or twenty years to develop. It is however encouraging to note that the one cancer known to have a relatively short latent period, namely leukemia, is not showing up in excess among those with higher exposures.

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Future Analysis

The analysis of the Hanford experience needs to be continually updated as new mortality data become available. There are a number of refinements which we hope to incorporate into such future analysis. The analysis presented above groups doses into four categories and considers accumulated exposures at just two points in time. Although any cancer which is positively correlated with exposure should show up as excessive in the higher dose groups of this analysis, the two refinements described below should provide a more powerful test of significance.

First, accumulated exposures could be considered at yearly intervals. This would require the introduction of at least 14 calendar year strata to cover the time period January 1, 1960 to April 1, 1974. Even more strata would be required if terminated employees are separated from those who are still employed (as illustrated by strata 2 and 3 in the analysis above). It may be desirable to introduce a lag of a year or two in determining exposure in order to allow for the interval between termination caused by a fatal cancer and subsequent death.

It may be helpful to illustrate the above by considering the control group of exposures for a worker who dies, for example, in 1971. If this worker was still employed as of January 1, 1970, an appropriate control might be the exposures accumulated up to this date by other workers still employed as of this date. On the other hand, if this worker had terminated in 1964, a more appropriate control would be the doses accumulated as of January 1, 1964 by workers still employed on this date. The age and occupation of the worker would also need to be considered in defining his stratum or control.

A second refinement involves consideration of exact doses instead of

exposure categories. The exact distribution of doses of those dying of a particular cause would then be compared with the distribution of doses for an appropriate control (see above). An appropriate summary measure and test of significance pose some problems because of the highly skewed nature of the data. Any transformation "strong" enough to make the data behave reasonably probably loses real information. There are considerations which make a square root transformation seem desirable, but this will not convert the data to a form for which means and t-tests will be generally appropriate.

To evaluate the overall death rates, or death rates from all malignant neoplasms, (and possibly from lung cancer), means and t-tests may be adequate. For very rare causes of death, such as multiple myeloma, an exact permutation test may be reasonable. For intermediate causes, the solution is not so clear. Possibly computer simulation or some sort of approximation to the exact test can be used. For example, the lower exposures might be grouped while higher exposures are considered individually.

The analysis described above can be put in the context of Cox's regression model for survival with concomitant variables [5]. Cox's time variable is taken to be calendar year (grouped in one year intervals), the single independent variable of interest is total accumulated doses (time dependent) with stratification to handle age, occupation, and terminated versus employed status. There are of course many additional possibilities offered by Cox's model including handling several measures of radiation exposure simultaneously, accounting for additional concomitant variables (for example, length of employment at Hanford), and estimating the magnitude of any effects observed. Breslow [3] considers a similar model which allows the use of a large external control population (such as U.S. white males) as a means of refining control of variables such as age and calendar year.

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If effects of radiation exposure are identified, some measure of the size of the effect as well as the relation of the effect to the timing of the exposures will be of interest. The question of latency periods, for example, needs consideration. A possible method for investigating this question is to lag the accumulated exposure by various time intervals. Another possibility is to treat the interval from initial exposure* as yet another stratification variable, or to include this interval as a variable in Cox's model. These options are discussed by Pasternack and Shore [20]. Mantel and Byar [14] (in an analysis of heart transplant data) consider the question of earlier versus later exposure (transplant in their case). In addition to considering the total accumulated dose, such measures as the maximum yearly dose, or the number of doses exceeding a certain value may be of interest. Such variables could be analyzed separately and/or included in Cox's model.

The limited data at higher doses makes it very difficult to come up with meaningful and reliable estimates of the magnitude of any effects found, especially in the absence of knowledge as to what models might be most appropriate. This is particularly true for very rare causes of death.

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*There are several options for defining the time of initial exposure. Some reasonable choices might be 1) the first year a single exposure exceeds a certain value; or 2) the first year the accumulated exposure exceeds a certain value.

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TABLE 1

STANDARD MORTALITY RATIOS (SMR) FOR WHITE MALES BY LENGTH OF EMPLOYMENT

(NUMBER OF DEATHS IS GIVEN IN PARENTHESES)

	LENGTH OF EMPLOYMENT	
	<2 YEARS	2 + YEARS
NUMBER OF WORKERS	7767	13,075
CAUSE OF DEATH		
ALL CAUSES	86 (1905)	75 (2089)
ALL MALIGNANT NEOPLASMS	88 (319)	85 (414)
CIRCULATORY DISEASES	87 (839)	76 (955)
ACCIDENTS, POISONINGS & VIOLENCE	109 (243)	75 (216)
ALL OTHER CAUSES	74 (423)	65 (455)
NO DEATH CERTIFICATE	(81)	(49)

THIS IS THE ONLY SMR PRESENTED IN THIS TABLE WHICH IS NOT SIGNIFICANTLY DIFFERENT FROM 100. ALL OTHER SMR'S ARE SIGNIFICANTLY LOWER THAN 100 AT THE .01 LEVEL.

Table 2

SMR's By Follow-Up Interval for White Males Employed at Least Two Years
(The number of deaths is given in parentheses)

	Years After Cohort Identification		
SMR for	2-7	7-12	12-
All Causes	46 (142)	65 (274)	81 (1673)
Malignant Neoplasms	56 (25)	82 (57)	89 (332)

Table 3

SMR's by Age For White Males Employed at Least Two Years
(The number of deaths is given in parentheses)

SMR for	Under 45	45-54	55-64	65-74	Over 75
All Causes	61 (234)	64 (401)	72 (618)	87 (564)	96 (27)
Malignant Neoplasms	62 (30)	77 (84)	88 (151)	96 (119)	84 (30)

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TABLE 4

STANDARD MORTALITY RATIOS (SMR) FOR WHITE MALES EMPLOYED AT LEAST TWO YEARS

(NUMBER OF DEATHS IS GIVEN IN PARENTHESES)

CAUSE OF DEATH	SMR
MALIGNANT NEOPLASM OF THE	
STOMACH	69 (23)
LARGE INTESTINE	101 (44)
PANCREAS	100 (28)
OTHER DIGESTIVE ORGANS	70 (33)
LUNG	78 (115) **
PROSTATE	90 (25)
LEUKEMIA	47 (10) *
OTHER LYMPHATIC TISSUE	109 (33)
OTHER (RESIDUAL)	87 (103)

*Significantly different from 100 at the .05 level

**Significantly different from 100 at the .01 level.

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Table 5

Observed and Expected Deaths Due to Selected Causes by Exposure Category for White Males Included in Exposure Study (see text).

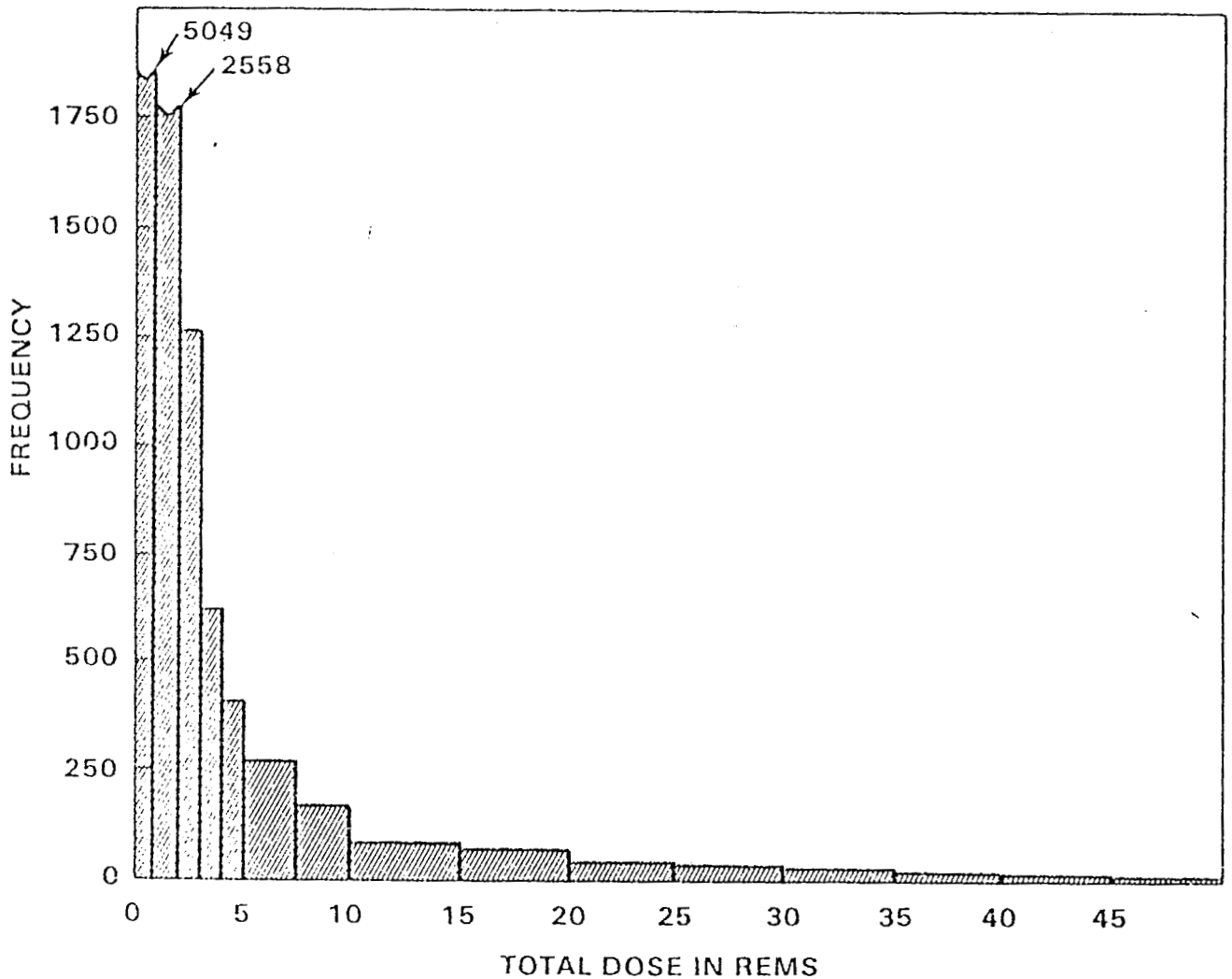
Cause of Death	Exposure Category: 0-2 rems Obs.	0-2 rems Exp.*	2-5 rems Obs.	2-5 rems Exp.*	5-15 rems Obs.	5-15 rems Exp.*	15 + rems Obs.	15 + rems Exp.*	Exposure Score for Trend Test Obs.	Exposure Score for Trend Test Exp.	Probability of Trend Arising Due to Chance**
All Causes	587	578.5	122	113.1	84	96.3	44	49.1	2.9	3.1	>.5
All Malignant Neoplasms (M.N.)	112	117.9	33	23.3	15	19.5	11	10.3	3.2	3.2	>.5
M.N. of Buccal Cavity	4	3.3	0	.7	1	.6	0	.4	2.2	3.6	>.5
M.N. of Colon	11	12.5	4	1.8	0	1.2	1	.5	2.7	2.2	>.5
M.N. of Pancreas	7	8.8	3	2.2	1	2.0	3	1.0	6.2	3.7	.03
M.N. of Other Digestive Organs	11	10.9	3	1.9	1	1.6	0	.7	1.8	2.8	>.5
M.N. of Lung	34	39.1	14	8.2	7	7.0	3	3.8	3.3	3.3	>.5
M.N. of Prostate	10	7.4	1	1.5	0	1.3	0	.9	1.0	3.6	>.5
M.N. of Brain	4	6.3	3	1.2	1	.9	1	.7	4.7	3.3	.18
Lymphosarcoma and Reticulum Cell Sarcoma	2	1.5	0	.5	1	.6	0	.4	3.2	5.6	>.5
Hodgkins Disease	2	2.1	1	.5	0	.4	0	.0	1.6	2.2	>.5
Leukemia	3	2.9	0	.6	1	.3	0	.2	2.6	2.7	>.5
Multiple Myeloma	1	2.3	0	.6	1	.8	2	.4	12.8	4.3	.01
Other Neoplasms of Lymphatic and Hematopoietic Tissue	2	1.6	0	.3	0	.1	0	.0	.8	1.8	>.5
All Other Cancers	21	19.3	4	3.4	1	2.8	1	1.4	2.2	2.9	>.5
All Non-Cancer Causes	461	449.2	89	87.3	66	74.9	33	37.6	2.9	3.1	
No Death Certificate	14	11.4	0	2.5	3	1.9	0	1.2	2.0	3.4	

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

**The significance levels are for a one-tailed test.

FIGURE 1

DISTRIBUTION OF TOTAL DOSES FOR WHITE MALE WORKERS EMPLOYED AT LEAST TWO YEARS



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