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A CONTINUING STUDY OF MORTALITY IN HANFORD WORKERS

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A CONTINUING STUDY OF MORTALITY IN HANFORD WORKERS

My presentation will consist of an update for those who have heard accounts of our study previously and a recap of the design and essential results for those who have not. The update consists of the addition to the study population of a number of deaths, most of which occurred between April 1, 1974 and May 1, 1977.

For those who are not familiar with the study, I will review briefly the design and methods of analysis. The Hanford plant, which is located in Washington state, started operation in 1944. At first, its mission was the production, chemical separation and purification of plutonium. Later, a large supporting research effort was developed. More recently, power has been generated in the one remaining active reactor, but additional power reactors are under construction at the site now.

The study of workers at Hanford and certain other sites such as Oak Ridge was started in 1964 by Dr. Thomas F. Mancuso of the University of Pittsburgh. From that time until now, the Hanford data have been collected by the Hanford Environmental Health Foundation, now headed by Dr. Breitenstein. Until 1976, the data were analyzed by Dr. Barkev S. Sanders. Dr. Sanders' principal analyses were concerned with the measurement of life shortening in the exposed workers.⁽¹⁾ Since 1975, Dr. Ethel Gilbert of our laboratory has conducted independent analyses. The results of those analyses have been presented at various meetings during the past three years and were recently published in depth in the July issue of the journal, Radiation Research.⁽²⁾ Since 1976, Dr. Alice Stewart and Mr. George Kneale have collaborated with Dr. Mancuso in analyzing the data and have published or presented a series of papers containing results that have received widespread distribution.^(3,4) Those

results and analyses have also occasioned much comment. I will return to this matter briefly before I close.

About 45,000 persons have been employed at the Hanford plant since the start of its operation. The population of radiation workers has been predominantly white and male. The population currently under study by Dr. Gilbert consists of 20,842 white males. The study population does not include workers who died before 1955 or were hired after 1965. These exclusions eliminate virtually no deaths with exposures of interest. The study is limited to mortality, and no attempt has been made to study morbidity. The Social Security Administration (SSA) identifies deaths in the employee population. Oak Ridge Associated Universities (ORAU) has an office that functions in an intermediary role, communicating our personnel rosters in a suitable format to SSA and receiving the results of the search of their files. ORAU also obtains death certificates from the states to establish the cause of death. SSA misses an undetermined number of deaths, which is believed to be about 5-8% of the total.

In our study of the role of radiation in health effects, we use the cumulative dose of external, penetrating radiation for each worker. The radiation exposure information available to us is probably as reliable as any in the industry. However, although prior radiation exposure data are obtained routinely now, that information is not available for transfers from other plants that occurred during the early period of plant operation when exposures elsewhere may have been relatively substantial in some poorly monitored installations. It also fails to include any exposure incurred subsequent to the individual's last period of employment at Hanford. The skewed nature of the distribution of cumulative doses in the study population is illustrated in the first slide.

Dr. Gilbert has used two principal methods of analysis. One involves the calculation of standardized mortality ratios (SMRs). These provide a comparison of mortality in the study population with mortality rates for the U.S. population based on vital statistics. The SMR is defined in the next slide.

The next slide shows SMRs for broad categories of causes of deaths. In this analysis Dr. Gilbert broke down the population by length of employment into those workers who had periods of employment of less than 2 years and those who were employed for 2 or more years. We see that, among the twenty thousand white male employees who began work before 1965, about two-thirds worked for at least two years. Of the others, only two individuals received as much as 5 rem of exposure. This slide demonstrates that the "healthy worker effect," which describes the more favorable mortality of workers in clean industries when compared with the general population, is less marked in the short-term than the longer term worker population. In other words, the mortality experience of the long-term workers is the more favorable. We also observe, as others have done, that the healthy worker effect for cancer is less marked than for other diseases such as cardiovascular disease. This is not surprising because we would expect cancer to be less influenced than other diseases by favorable working conditions and health programs. The result is that the proportion of cancer cases may be greater among the long-term, more heavily exposed workers than among the short-term, less exposed workers. This may create the erroneous impression that the long-term, more exposed workers have more cancer when, in fact, the proportion of cancer in those workers may be increased only because they have fewer deaths from other diseases.

The second and more important analytical approach used by Dr. Gilbert

involves testing for a relationship between the level of radiation exposure and mortality. In this analysis, she controlled for age, calendar year of death, occupational category and employment status. Here, we can provide an updated analysis with about a 20% increase in the number of deaths relative to our previous data set. This includes 390 additional deaths of all types, 94 of which are cancer deaths. In this analysis, the worker population is divided into groups according to their cumulative radiation dose, the cutpoints being at 2, 5 and 15 rem. Then, the trend of mortality with increasing exposure is tested. The expected deaths for each category of age, calendar year of death, employment status and occupation are calculated as if radiation dose were not a factor. If radiation dose were to influence the mortality rates, the observed deaths would significantly exceed the expected in the higher dose groups. The statistical method used here is the Mantel-Haenszel procedure. The statistical test for trend of increasing mortality with dose is called the Mantel test.

The next slide presents results of the analysis of trend with increasing dose. Here, in order to compress the material into a readable slide, we have pooled the 0-2 and 2-5 rem categories so that the combined groups are displayed as a 0-5 rem category. The slide shows observed and expected deaths for all causes, for "all cancer" cases and for several selected cancer types. The p value in the right column gives the level of significance of the one-tailed test. Values below .5 indicate an increasingly positive trend at the higher dose levels whereas values above .5 may indicate the opposite, namely, a decreasing trend in higher dose ranges. We see that for all causes the higher dose ranges show fewer deaths than expected. The same is true for all cancer. Corresponding to these, we have high p values. Multiple myeloma shows a positive test at the 1% level of significance; the pancreas is not

quite significant at the 5% level. At the opposite end of the scale, the prostate gland and the category of "other digestive organs," which includes esophagus, small intestine, liver and gallbladder, show a negative trend with p values greater than .95. Myeloid leukemia shows no trend.

The principal difference between our early and current sets of data occurs in cancer of the stomach where we had no cases in the high dose range previously but have three cases now. However, the test for trend is still not significant. In response to suggestions that we attempt to verify the diagnoses, especially for cancer of the pancreas, which is especially likely to be misdiagnosed, we checked back into the medical records of the multiple myeloma and pancreatic cancer cases. We did find that, in one high dose, pancreatic case, there was no autopsy, but the death certificate diagnosis of cancer of the pancreas did not agree with the attending physician's diagnosis, which was cancer of the stomach. We have not changed our formal analysis to reflect this observation because we have not had an opportunity to do a systematic search on all relevant cancer types. However, if that diagnosis were changed, the pancreas would not approach significance while the stomach would have a level of significance somewhere between 5 and 10%. This case illustrates the precarious nature of any conclusions that might be based on the small numbers of cases in each disease category.

We are continuing this study with periodic searches for additional deaths and updates of the analyses. Our most recent submission to SSA includes a group of several thousand workers employed by a construction firm at the plant; many of those workers incurred nontrivial radiation exposure while doing maintenance work. Smoking histories for the study population are inadequate, but we plan to investigate differences in smoking incidence between different occupational categories in the event that lung cancer

becomes a matter of interest in the study. We are also interested in developing an estimate of the percentage of deaths missed in our population by SSA by means of a suitable stratified sampling follow-up study. This would provide the basis for an adjustment of our Standardized Mortality Ratios. Finally, if we can develop a feasible unbiased system for verification of diagnosis, we will undertake a project directed to that end.

We have recognized for some time that other exposures, principally chemical, may serve as confounding factors to radiation where trends of increased mortality with radiation exposure are observed. However, obtaining historical information of value about such exposures has proven to be virtually impossible to achieve. At present we are seeking such information about the few high dose myeloma and cancer of the pancreas cases.

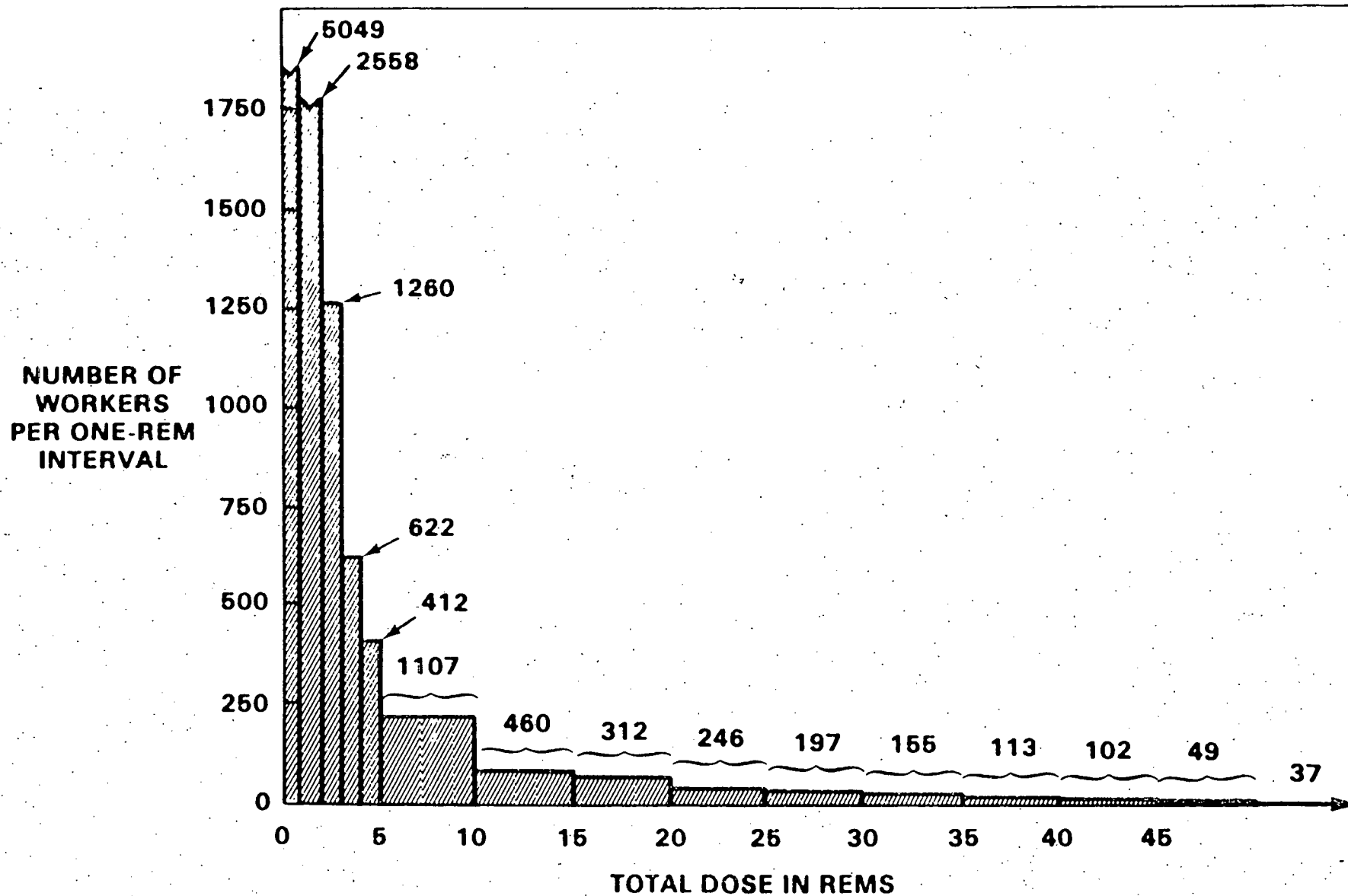
I have refrained from commenting on the analyses, results and conclusions reported by Mancuso, Stewart and Kneale. If any of you are interested in reading critiques of their studies, I recommend to you reviews by Anderson,⁽⁵⁾ Reissland,⁽⁶⁾ and the team of Hutchison, MacMahon, Jablon and Land,⁽⁷⁾ and our own publications.

To summarize, I have presented an updated analysis of data on the mortality experience of about 12,500 white, male workers who were employed at Hanford for at least two years. The addition of 390 deaths, including 94 cancer deaths to our file has not changed our previous results in any important respect. Using a Mantel-Haenszel analysis of trend of mortality with radiation dose, a positive trend of multiple myeloma with increasing dose at the 1% level of significance remains. Cancer of the pancreas shows a doubtful trend with dose. Certain other organs show fewer deaths than expected in the higher dose ranges. All deaths and all cancer deaths display a similar negative trend with dose.

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



POPULATION ANALYSIS

O_i = NUMBER OF DEATHS FROM CAUSE i OBSERVED
IN THE HANFORD POPULATION.

E_i = NUMBER OF DEATHS FROM CAUSE i EXPECTED
IN THE HANFORD POPULATION BASED ON
AGE - CALENDAR YEAR SPECIFIC RATES
FOR U.S. WHITE MALES

$SMR_i = 100 \times O_i / E_i$ = STANDARDIZED MORTALITY
RATIO FOR CAUSE i

OBSERVED AND EXPECTED DEATHS
AND SMRs FOR WHITE MALES

	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.	SMR	Obs.	Exp.	SMR
All Causes	1905	2216.6	86	2089	2796.8	75
All Malignant Neoplasms	319	363.0	88	414	487.7	85
Diseases of the Circulatory System	839	965.4	87	955	1254.2	76
Accidents, Poisonings and Violence	243	222.9	109	216	288.8	75
All Other Causes	423	568.1	74	455	700.8	65

Slide 3

OBSERVED AND EXPECTED DEATHS BY
EXPOSURE CATEGORY FOR WHITE MALES

Cause of Death	Exposure Category						p value
	0-5 rem		5-16 rem		15+ rem		
	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	
All Causes	1982	1969.0	144	147.8	90	99.2	0.88
All Cancer	408	400.7	29	32.8	20	23.5	0.81
Stomach	25	24.8	0	1.9	3	1.3	0.13
Large Intestine	41	39.6	1	2.1	1	1.3	0.66
Pancreas	27	27.4	1	2.9	4	1.8	>0.06
Other Digestive Organs	33	30.5	0	1.5	0	1.0	>0.95
Lung	115	117.0	16	11.9	7	9.1	0.54
Prostate Gland	32	28.6	2	3.0	0	2.5	>0.97
Myeloid Leukemia	7	7.2	1	0.6	0	0.3	0.71
Multiple Myeloma	4	5.9	0	0.6	3	0.5	0.006

Slide 4