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**Mortality Among a Cohort of White Male Workers at a
Uranium Processing Plant: Fernald Feed Materials Production Center,
1951-1989**

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Fernald Cohort Mortality Study, 1951-1989

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This study followed a cohort of 4,014 white males hired at the Feed Materials Production Center (FMPC) in Fernald, Ohio, between 1951 and 1981. Vital status through the end of 1989 ascertained 1,064 deaths. SMRs stratified by paycode showed a healthy worker effect in salaried, but not hourly employees when compared with mortality rates of U.S. white males. Significant increases were noted for salaried workers for deaths from stomach cancer [SMR=2.61, 90% confidence interval (C.I.) (1.22,5.14)] and for hourly workers for all cancers [SMR=1.21, (1.07,1.37)], lung cancer [SMR=1.26, (1.02,1.54)], and motor vehicle accidents [SMR=1.59, (1.14,2.15)]. Dosimetry data was available for 99% of the study members. Cumulative population doses for internal and external radiation were 163.6 Gray (Gy) and 58.9 Sieverts (Sv). Trend test statistics for 16 selected cancer and three noncancer causes of death revealed a borderline significant trend for lung cancer ($p=.08$) with external dose. Trend tests for nonmalignant respiratory diseases were significant for chronic respiratory diseases ($p=.01$) with internal dose. Dose-response analyses for lung cancer with external dose revealed an excess relative risk per Sv of 8.0 [90% C.I.: (0.1, 18.5)] with a 10 year lag and 10.7 (1.8, 23.7) with a 15 year lag. Models of lung cancer with internal dose revealed no significant relationships. Dose-response analyses for nonmalignant respiratory diseases and internal dose resulted in an excess relative risk per Gy of 13.8 (2.8,45.8) with a 10 year lag and 14.2 (2.2, 44.2) with a 15 year lag.

INTRODUCTION

From 1951 to 1989 the Fernald Feed Materials Production Center (FMPC) processed uranium ore concentrates, uranium of low-grade enrichment, and thorium into fabricated metal products and reactor core target elements for use by the nation's defense programs. Because of the work processes involved, the potential for internal radiation dose from inhalation of uranium dust was higher than the potential for penetrating dose received from external emitting sources.

The objectives of this study were to determine whether there was (1) excess mortality in the cohort compared to the United States general population, and (2) evidence that workers with higher doses of either internal or external penetrating radiation had elevated rates of certain diseases. Only white males were included in this study because they were the majority of the worker population and worked in areas with the greatest potential for radiation exposure.

METHODS

The population was enumerated from original work history records obtained from the facility for the years 1951 through 1989. The data elements available from these records included: birth date, race, gender, pay status, date of first employment, leave of absence dates, termination date, job title, department and plant.

Vital status was determined through searches conducted by the Social Security Administration, Pension Benefits, Inc., and the National Death Index. Death certificates were sought for all individuals where there was an indication of death from any of these sources. All certificates were coded according to the eighth revision of the International Classification of Diseases, Adapted for Use in the United States (ICDA). The underlying cause of death was used for all external analyses comparing the population to national statistics. Non-underlying cancer causes were used for all internal cancer-specific, dose-response analyses comparing exposed

cohort members to unexposed.

Assignment of External Doses

The deep dose equivalents provided by the facility were used for external radiation dose estimates. Weekly external radiation monitoring began at FMPC in 1952 for employees having the highest exposure potential. Employees having a lesser exposure potential were monitored biweekly. By the mid-1950s all workers were monitored monthly until the 1989 shutdown of the facility's operations. No neutron monitoring was performed as the potential for neutron exposure was not present. Film badges were used until 1983 when the site switched to thermoluminescent dosimeters (TLDs). Available exposure records were maintained as an annual cumulative dose equivalent which was rounded to the nearest mSv during the period of film badge use. The cumulative TLD measurements were recorded to the nearest 0.01 mSv with the exception of measurements that were less than 0.05 mSv, which were set to zero.

Monitoring results were not available for 1% of the working years for the cohort. For these missing years, the dose was prorated and assigned based on the dose equivalent for the year immediately preceding or following the missing dose provided similar activities continued. If there was no available information to assign a dose from nearby years, an appropriate median dose was assigned from a comparable population matched to the employee work history (0.5%). The median dose was used rather than the mean to avoid the biasing effect of any unusually high results.

Once the median and nearby dose equivalents were assigned, there were an additional 41 (0.1%) person-years without exposure data. These person-years were individually assigned dose equivalents by a health physicist, considering year of employment, plant, and job title information.

Assignment of Internal Doses

Internal exposure monitoring for radiation first began with a urinalysis program in 1952. Workers were monitored on a nonroutine basis as a control measure and by 1958 urinalysis became a primary means of internal exposure monitoring. This program required quarterly urine sample collection for all process area workers and semi-annual collection for the remaining workers. Beginning in 1968, the Y-12 (Oak Ridge, TN) mobile *In Vivo* Radiation Monitoring Laboratory visited the FMPC site twice a year to perform whole body counts (WBC). Worker WBC monitoring frequency was determined by job potential for exposure and previous results.

Much of the lung exposure was due to relatively insoluble uranium compounds. However, to limit the effect on doses from possible exposures to more soluble forms of uranium a technique was used which was earlier developed by Strom (1) to eliminate aberrantly-high results from both *in vivo* and urinalyses files.

The amounts of uranium (in units of mg) in the lung was calculated from *in vivo* lung count measurements. Urinalyses results were also used to estimate amount of uranium in the lung by a method developed by Strom (1) and later reported by Crawford-Brown (2). Since all internal data were converted to the same unit, the lung burdens estimated from the *in vivo* and urinalysis data were combined to determine the best integrated burden-day estimate for each employee-year of exposure (3). For years in which a worker had no internal monitoring results (6%), doses were estimated using nearby measurements. These reference measurements were taken from similar worker activities within a two year period (4%) surrounding the worker's year of missing results. If there was no available information to estimate a nearby dose (2%), an appropriate median dose was assigned from the population available that best matched the employee work history. The lung dose to uranium was calculated in units of internal radiation dose (mGy) per mg-day of internal exposure by converting these weight measurements into

activity units and assuming a straight line relationship.

Statistical Analyses

Standardized Mortality Ratio (SMR) analyses compared the overall and cause-specific mortality of the hourly and the salaried employees to U.S. white male population rates. Dose-response analyses investigated the relationship between radiation doses and mortality using an internal comparison group. Time-dependent cumulative internal (mGy) and external (mSv) radiation doses were stratified into groups using these intervals: 0, -5, -10, -20, -40, -80, -160, -320, 320+. For external doses the final interval was 160+ mSv since no person-years were assigned to the 320+ interval. For each cell, the mean cumulative dose of all its person-years was used as the dose value in analyses.

The first phase of the dose-response analysis consisted of tests for trend as a screening procedure for a dose-response relationship between selected causes of death and cumulative internal or external doses lagged ten years and also lagged two years for leukemia. Although internal doses were computed based on dose to the lung, these doses would be proportionately scaled for other organ sites. For the purpose of screening in trend tests, they are sufficient for ordering levels of exposure in the population. As detailed in Breslow and Day (4), Poisson trend statistics were calculated stratifying on 15 five-year age groups, eight five-year calendar periods, and two paycode groupings (hourly and salaried) for distributions of person-years at risk. The final phase of the analysis determined dose-response estimates for lung cancer and nonmalignant respiratory diseases. An excess relative risk model was employed utilizing Poisson regression techniques for modeling death rates as a function of dose and covariate values to obtain maximum likelihood estimates of the parameters. Precision of the estimates was indicated by likelihood based 90% confidence intervals. Cumulative doses were accumulated with 10-year and 15-year lags and intervals corresponded to those listed above in the description of the trend

tests. Dose-response estimates were adjusted by including these covariates in the models: age (natural log of age/52.5), birth cohort (1:pre-1920, 2:1920-30, 3:1930+), and paycode (1:hourly, 2:monthly). Dose-response relationships were investigated separately for internal dose and external dose by modeling dose as continuous with the mean cumulative dose in each cell being used as the dose value in the analysis. The resulting parameter estimates were scaled to represent the change in the excess relative risk for each Sv (for external dose) or Gy (for internal dose) increase in dose or, equivalently, the percent change for each 10 mSv or 10 mGy increase in dose.

RESULTS

The cohort consisted of 4,014 white males who entered follow-up between 1951 and 1981 and contributed a total of 121,038 person-years of follow-up through 1989. Of the total, 1,339 (33.4%) were salaried or white collar workers and 2,675 (66.6%) were hourly or blue collar workers. By the end of 1989 a total of 1,064 deaths (26.5% of the cohort) were identified, and 1,053 death certificates (99.0% of the deaths) were obtained.

The average age at hire was 30.7 years with a median of 29.0 years. Over 85% of the cohort was first hired before 1960 allowing 30 or more years of follow-up. This is reflected in the average length of follow-up of 30.2 years and a median of 33.6 years. For length of employment the minimum was three months and the maximum 37.1 years with a mean and median of 8.5 years and 5.5 years.

The SMR analysis results are listed in Table I for both salaried and hourly employees. There were no deaths from bone sarcoma and only one death from chronic nephritis in the population. The salaried employees exhibited the healthy worker effect with significantly decreased SMRs for all causes, and more particularly, all diseases of the circulatory system.

Other significant deficits in mortality for salaried employees occurred for lung cancer, diseases of the respiratory system, diseases of the genitourinary system, and all external causes of death. Only the SMR for stomach cancer was significantly elevated.

Hourly employees had a significant deficit for all diseases of the circulatory system and diseases of the genitourinary system. The all causes SMR did not show a healthy worker effect and this group showed significantly elevated mortality for all cancer deaths. A significant excess of lung cancer deaths was observed in this hourly worker population.

Dose distributions for the cohort by different lag periods and by hourly or salaried work status are shown in Tables II and III. The total population dose for internal radiation and external radiation were 163.6 Gy and 58.9 Sv. Internal doses tended to be higher than external doses, as evidenced in the means (40.9 mGy versus 14.8 mSv) and medians (29.9 mGy versus 2.0 mSv).

Table IV lists trend test statistics for specific cancers by internal dose. Non-Hodgkins lymphoma is of borderline significance but is based on only three cases. Lymphosarcoma and reticulum-cell sarcoma is also of borderline significance; however, the nine cases are all distributed among the lower dose groups below 20 mGy for a ten year lag and below 80 mGy for a two year lag.

Table V displays the results of the trend tests for cancers with external dose. With the exception of a borderline significant trend for lung cancer, none of the cancer causes exhibited a significant trend with increasing external radiation dose. Comparison of the observed to expected ratios by dose group for lung cancer shows that the increased risk occurs beginning in the 40-80 mSv dose group and continues into the higher dose groups.

Trend test results for nonmalignant respiratory disease deaths, along with the acute and chronic subgroups with internal and external doses are shown in Table VI. The acute subgroup includes diseases such as influenza and pneumonia, whereas chronic bronchitis, emphysema,

asthma, chronic interstitial pneumonia, and chronic obstructive pulmonary disease comprises the second subgroup. The trend test for all respiratory diseases together shows a borderline significant trend with internal dose. When subdivided, the trend for acute conditions is not significant. However, the trend for chronic conditions with internal dose is significant with a p-value of 0.01. Analyses using external doses did not reveal any significant relationship with nonmalignant respiratory diseases either as a whole or subgrouped by acute and chronic.

Table VIII displays the results of the excess relative risk modeling analyses for lag periods of 10 and 15 years. Presented are estimates per Gy of internal radiation dose along with likelihood based 90% confidence intervals for lung cancer and nonmalignant respiratory diseases. Confirming the results of the trend tests, there was no statistically-significant relationship between cumulative internal dose and lung cancer. For nonmalignant respiratory diseases both the 10- and 15-year lag models are statistically significant and the estimates of excess relative risk are quite similar. Because the distribution of deaths for the chronic lung diseases subgroup did not include any deaths in the four lowest dose groups, estimates could not be calculated. Also presented are estimates per Sv of external radiation dose for lung cancer. Both the 10- and 15-year lag models showed a significant relationship with the 15-year lag revealing a higher risk.

DISCUSSION

Examination of the results of standardized mortality ratio (SMR) analyses for this population of workers occupationally exposed to uranium dust revealed, in salaried workers, a deficit of deaths from all causes combined. This healthy worker effect was not evident in the hourly workers and was an unusual finding for an occupational epidemiologic study focused on a cohort where hiring first occurred in the 1950s and later. The SMR for deaths from diseases of the circulatory system was significantly decreased for both the salaried and hourly employees reflecting evidence of the healthy worker effect for cardiovascular diseases. The significant

increase in deaths among hourly workers from cancers is the result of generalized increases in deaths in most cancer categories rather than a large increase in a single category. The increases in cancer deaths among hourly employees included 14 of the 17 categories examined with the exceptions of cancer of the stomach, bone sarcoma, and cancer of the skin.

It is important to note that this population of radiation exposed workers exhibited neither excess leukemia nor a dose-response relationship between leukemia (excluding two observed CLLs) and either internal or external radiation dose. The total population dose for external radiation was 58.9 Sv with the average cumulative dose to a worker of 14.7 mSv, thus limiting the ability to detect such a dose-response relationship with external radiation.

Examination of the excess of stomach cancer deaths in the salaried employees reveals the majority of these deaths occurred in individuals who held administrative positions, and had very low potential for exposure to either internal or external radiation, or chemicals. Increases in stomach cancer have been observed in other populations where the primary exposure was to internal radiation; however, in the FMPC population there was no evidence of a dose-response relationship with either internal or external radiation.

An excess of lung cancer was observed in the hourly worker population when compared with the United States standard population. Lung cancer is not generally recognized as a consequence of external radiation exposure, whereas it might be expected as a consequence of chronic exposure to uranium dust. Published results from combined worker studies (5) did not demonstrate a trend for external dose with lung cancer, but rather revealed a negative trend. However, for the Fernald cohort the trend test for lung cancer with external dose was of borderline significance, and the dose-response estimate for lung cancer was also revealed as statistically significant. The major difference between the populations in the combined worker population study and the present study is the additional possibility of exposure to internal

radiation in the Fernald cohort. A study by Checkoway (6) investigated a population of workers who had similar types of exposures as the present population. The Checkoway study found a relationship between external dose and lung cancer in workers who had more than 5 mGy cumulative internal dose using a zero and a 10-year lag (the Checkoway study used a quality factor of 10 for the internal dose which has been removed here for the purpose of comparison). Examination of the Fernald population dose distribution with a 10-year lag reveals that 84% of the hourly workers and 67.7% of the salaried workers have more than 5 mGy of internal dose.

In a recent analysis of mortality among white male workers at the Los Alamos National Laboratory (7) a subgroup of workers who were monitored for plutonium exposure was examined. Because only 303 of the workers in this monitored group actually had doses from plutonium, it was possible to compare death rates in the plutonium-exposed monitored workers with those in the plutonium-unexposed monitored workers. The rate ratio for lung cancer comparing exposed to unexposed workers was 1.78 (95% confidence interval 0.79 to 3.99, based on eight cases). The next analysis excluded workers who had a cumulative external radiation dose of 10 mSv or more in order to remove confounding from external dose. This analysis showed a rate ratio for lung cancer of 1.04 (95% confidence interval 0.20 to 3.57). Although the numbers are small, this study may provide more evidence in worker populations that internal radiation dose may facilitate the effectiveness of external radiation dose in carcinogenesis.

A case-control study of 787 lung cancer among workers employed in four uranium processing or fabrication operations (8) did not find any dose-response effect for internal dose to the lung. External radiation exposure was available for 105 of the case-control pairs so that an analysis of the joint effect of internal and external dose could be completed. The odds ratios increased with level of external exposure in those who had more than 5 mGy internal dose, although the odds ratios were not significant. For categories of external radiation of <10 mSv,

10-49 mSv, and ≥ 50 mSv, the odds ratios were 0.64, 1.03, and 1.18.

Results of studies on uranium miners are not directly comparable to the study of the Fernald population because the uranium miners were exposed to radon progeny from the decay of radium while the Fernald workers were primarily exposed to uranium compounds.

Figures 1 and 2 display the mortality rates with 95% confidence intervals for separate dose categories in mGy (Figure 1) and mSv (Figure 2) as follows: below 5, 5 to less than 20, 20 to less than 80, 80 to less than 160, and 160 or greater. Some dose categories collapsed because of small numbers in order to create the figures. Figure 1 shows the relationship between cumulative internal dose and nonmalignant respiratory diseases while Figure 2 shows cumulative external dose and lung cancer. Although there is a suggestion of dose-response relationships in these two graphs, which was also demonstrated in trend analyses and the ERR modeling, the wide confidence intervals illustrate the extent of the uncertainty involved.

The Fernald population was also exposed to multiple chemical exposures, most notably nitric acid, sodium hydroxide, tributyl phosphate, trichloroethylene, and kerosene. It is known that the level of these chemical exposures would also increase as the level of external or internal radiation dose increased. Therefore, it is possible that some of the excess risk observed for lung cancer and NMRD may actually be caused by these chemical exposures or caused by a potentiating role of the chemical exposures making the radiation exposures more effective.

The significant dose-response relationship for non-malignant respiratory disease (NMRD) in this population was previously described (9) although the magnitude of the relationship was not calculated per unit dose. The earlier study used a semiquantitative ranking scheme to place workers in different levels of potential for exposure to uranium dust and found that the relative risk of NMRD increased with increasing cumulative uranium dust exposure.

The absolute values of the dose-response estimates for this population must be viewed cautiously because of the inextricable nature of the doses for internal and external radiation. Table IX displays the population average internal dose for each external dose group and the population average external dose for each internal dose group. As external dose increases, so does internal dose in a somewhat steady manner indicating a dose-response observed for external radiation may be due to internal dose. The measurement of internal dose is less straightforward than the measurement of external dose. For this population, it is possible, a level of inaccuracy is associated with the placement of workers into internal dose categories.

The average external dose for each internal dose category fluctuates and is not nearly as straightforward as the internal dose for external-dose category comparison. This might indicate whatever dose response is observed for internal radiation-dose categories is not likely to be due to the level of the external dose whereas a dose response observed for external dose may be highly related to internal dose.

We conclude that there is evidence of a radiation dose-response relationship in this population for both non-malignant respiratory disease and lung cancer. These findings were produced without controlling for other lung carcinogens that were likely present in the work environment at this facility. Inclusion of other confounding exposures may have altered the findings presented here.

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TABLE IV

Trend Test Statistics for Specific Cancers with Internal Doses

	Trend Test Statistic ^a	p-value	Observed and Expected Deaths by Dose Category in mGy (obs/exp) ^b									
			0	>0-5	5-10	10-20	20-40	40-80	80-160	160-320	>320	
Doses Lagged 10 Years												
All cancers (140-209)	0.18	0.67	34/30.86	30/28.29	26/22.43	27/35.33	72/65.32	115/111.93	42/52.61	4/4.53	2/0.70	
Esophagus (150)	0.50	0.48	0/0.94	0/0.72	2/0.52	2/0.87	2/1.59	2/2.81	1/1.41	0/0.12	0/0.02	
Stomach (151)	0.02	0.88	2/1.94	1/1.48	2/1.05	1/1.84	3/3.11	5/4.10	1/1.35	0/0.10	0/0.02	
Colon (153)	0.22	0.64	5/2.01	2/2.41	1/1.98	2/3.11	7/5.84	7/9.35	5/3.99	0/0.29	0/0.03	
Rectum (154)	1.53	0.22	1/0.88	0/0.55	1/0.36	1/0.59	2/1.18	3/2.59	0/1.71	0/0.12	0/0.02	
Pancreas (157)	0.73	0.39	1/2.00	0/1.83	2/1.27	1/1.90	7/3.34	2/5.13	5/2.29	0/0.21	0/0.03	
Lung (162, 163)	1.75	0.19	9/10.06	11/8.61	8/7.36	12/11.73	19/22.43	[43/40.87	17/20.72	3/1.97	2/0.30	65/63
Prostate (185)	1.17	0.28	3/1.00	1/1.70	0/1.59	1/2.70	4/5.18	10/9.83	8/4.56	0/0.39	0/0.05	
Bladder (188)	0.06	0.81	2/0.30	0/0.38	0/0.40	0/0.64	1/1.40	3/3.11	2/1.61	0/0.12	0/0.03	
Kidney (189)	0.06	0.80	0/0.17	0/0.57	0/0.43	0/0.59	1/0.91	4/1.44	0/0.77	0/0.09	0/0.02	
Lymphosarcoma and reticulum-cell sarcoma (200)	3.36	0.07	2/1.87	1/1.67	2/0.68	4/0.70	0/1.03	0/2.08	0/0.89	0/0.06	0.0.00	

TABLE IV
(continued)

	Trend Test Statistic ^a	p-value	Observed and Expected Deaths by Dose Category in mGy (obs/exp) ^b								
			0	>0-5	5-10	10-20	20-40	40-80	80-160	160-320	>320
Hodgkins disease (201)	1.15	0.28	1/1.21	1/0.35	1/0.39	1/0.61	1/1.19	1/1.49	0/0.67	0/0.65	0/0.21
Non-Hodgkins lymphoma (202.2)	2.90	0.09	0/0.12	0/0.18	0/0.19	0/0.26	0/0.48	2/1.07	0/0.64	1/0.06	0/0.01
Multiple myeloma (203)	0.44	0.51	0/0.52	1/0.68	0/0.43	0/0.57	3/1.19	4/2.94	0/1.51	0/0.14	0/0.02
Chronic lymphocytic leukemia (CLL) (204.1)	0.02	0.88	0/0.07	0/0.11	0/0.10	0/0.16	1/0.42	1/0.83	0/0.28	0/0.02	0/0.00
Leukemia excluding CLL (204-207 except 204.1)	0.04	0.84	1/1.49	1/0.90	1/0.89	0/1.39	3/2.44	6/3.58	0/1.19	0/0.10	0/0.02
Doses lagged 2 years											
Lymphosarcoma and reticulum-cell sarcoma (200)	3.43	0.06	0/0.29	0/1.35	2/0.71	5/0.88	1/1.63	1/2.74	0/1.27	0/0.13	0/0.00
Leukemia excluding CLL (204-207, except 204.1)	0.00	0.98	0/0.60	6/1.12	1/1.01	1/1.57	3/2.49	5/3.63	1/1.44	0/0.12	0/0.03

^aTrend test statistic was calculated using average dose per cell, where cells were stratified by 15 levels of age, 8 levels of calendar year, and 2 levels of paycode.

^bExpected deaths were calculated based on person-years distribution of age, calendar year, and paycode for all workers in the study.

TABLE V

Trend Test Statistics for Specific Cancers with External Doses

Cause of Death	Trend Test Statistic ^a	p-value	Observed and Expected Deaths by Dose Category in mSv (obs/exp) ^b							
			0	>0-5	5-10	10-20	20-40	40-80	80-160	>160
Doses Lagged 10 Years										
All cancer (140-209)	0.68	0.41	121/114.07	88/98.94	30/27.09	23/30.92	39/37.74	38/31.13	12/11.22	1/0.88
Esophagus (150)	0.93	0.34	3/2.86	5/2.39	0/0.68	0/0.78	0/1.01	1/1.01	0/0.35	0/0.03
Stomach (151)	0.81	0.37	8/6.61	2/4.56	1/0.91	0/0.92	2/1.09	2/0.75	0/0.15	0/0.00
Colon (153)	0.06	0.80	9/9.38	9/8.93	3/2.34	3/2.71	1/2.71	3/2.25	1/0.63	0/0.04
Rectum (154)	0.81	0.37	3/2.20	2/1.95	2/0.67	0/0.83	0/1.06	1/0.86	0/0.39	0/0.05
Pancreas (157)	0.43	0.51	3/6.67	6/4.78	2/1.34	2/1.40	2/1.87	3/1.47	0/0.45	0/0.03
Lung (162, 163)	3.09	0.08	41/36.97	29/33.58	9/10.01	4/11.06	15/14.47	18/12.61	7/4.85	1/0.43 ^{41/32.36}
Prostate (185)	0.33	0.57	7/7.28	2/8.35	6/2.17	4/2.59	4/3.03	3/2.47	1/1.02	0/0.09
Bladder (188)	0.07	0.80	2/1.70	2/1.99	1/0.72	1/0.95	0/1.20	2/1.06	0/0.35	0/0.22
Kidney (189)	0.02	0.89	1/1.71	2/1.30	0/0.37	0/0.38	1/0.50	1/0.49	0/0.23	0/0.02
Lymphosarcoma and reticulum-cell sarcoma (200)	1.75	0.18	6/4.14	2/2.13	1/0.55	0/0.68	0/0.73	0/0.80	0/0.15	0/0.00
Hodgkins disease (201)	0.03	0.85	3/2.56	2/1.38	0/0.44	0/0.44	0/0.59	1/0.42	0/0.15	0/0.01

TABLE V
(continued)

Cause of Death	Trend Test Statistic ^a	p-value	Observed and Expected Deaths by Dose Category in mSv (obs/exp) ^b							
			0	>0-5	5-10	10-20	20-40	40-80	80-160	>160
Multiple myeloma (203)	0.33	0.57	2/1.89	2/2.26	1/0.55	0/0.91	3/1.18	0/0.84	0/0.36	0/0.01
Chronic lymphocytic leukemia (CLL) (204.1)	0.11	0.74	0/0.49	1/0.56	0/0.22	1/0.25	0/0.26	0/0.18	0/0.03	0/0.00
Leukemia excluding CLL (204-207 except 204.1)	0.88	0.35	7/4.65	3/3.37	0/0.91	0/0.92	2/1.06	0/0.84	0/0.24	0/0.01
Doses lagged 2 years										
Lymphosarcoma and reticulum-cell sarcoma (200)	2.22	0.14	4/2.92	4/2.57	1/0.69	0/0.84	0/0.93	0/0.70	0/0.32	0/0.03
Leukemia excluding CLL (204-207, except 204.1)	0.61	0.43	6/3.92	4/3.78	0/1.01	0/0.89	1/1.13	1/0.88	0/0.37	0/0.03

^aTrend test statistic was calculated using average dose per cell where cells were stratified by 15 levels of age, 8 levels of calendar year, and 2 levels of paycode.

^bExpected deaths were calculated based on person-years distribution of age, calendar year, and paycode for all workers in the study.

TABLE VI

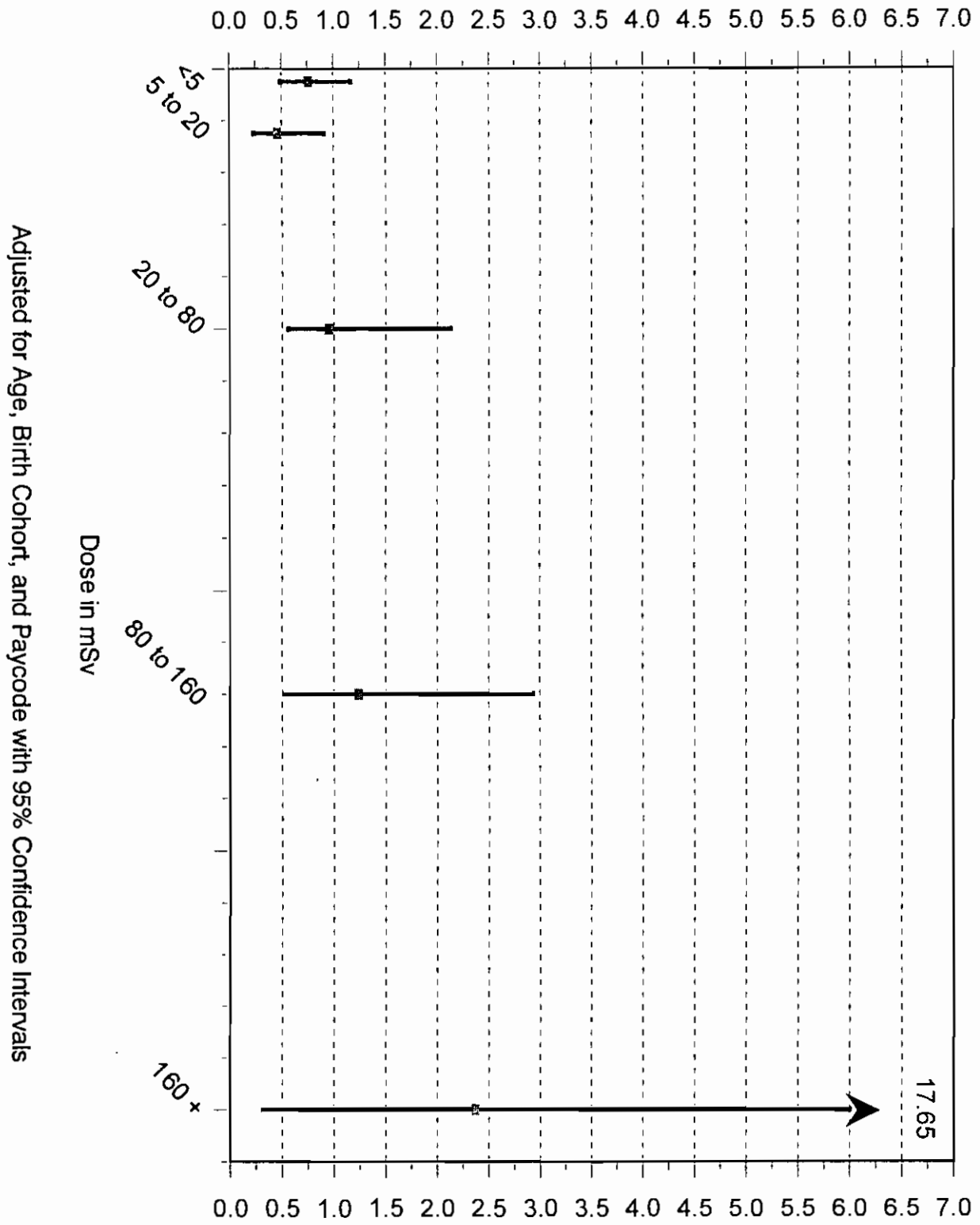
Trend Test Statistics for Nonmalignant Respiratory Diseases

Cause of Death (ICD8)	Trend Test Statistic ^a	p-value	Observed and Expected Deaths by Dose Category in mSv (for external) and mGy (for internal) (obs/exp) ^b								
			0	>0-5	5-10	10-20	20-40	40-80	80-160	160-320	>320
<u>Internal Doses Lagged 10 Years</u>											
All nonmalignant respiratory diseases (460-519)	2.73	0.10	3/3.12	1/3.21	1/3.09	5/4.94	11/9.58	14/18.30	17/9.75	1/0.85	0/0.16
Acute (460-489)	0.72	0.40	3/1.52	1/1.22	1/1.14	3/1.91	2/3.31	5/5.16	2/2.49	0/0.21	0/0.03
Chronic (490-519)	6.33	0.01	0/1.60	0/1.99	0/1.95	2/3.03	9/6.26	9/13.14	15/7.27	1/0.64	0/0.12
<u>External Doses Lagged in 10 Years</u>											
All nonmalignant respiratory diseases (460-519)	1.21	0.27	10/13.93	16/14.44	2/4.33	5/5.16	10/6.62	6/5.96	4/2.37	0/0.20	
Acute (460-489)	0.11	0.74	8/5.71	4/4.81	1/1.21	2/1.36	0/1.72	1/1.53	1/0.61	0/0.05	
Chronic (490-519)	2.22	0.14	2/8.22	12/9.64	1/3.11	3/3.80	10/4.90	5/4.43	3/1.75	0/0.15	

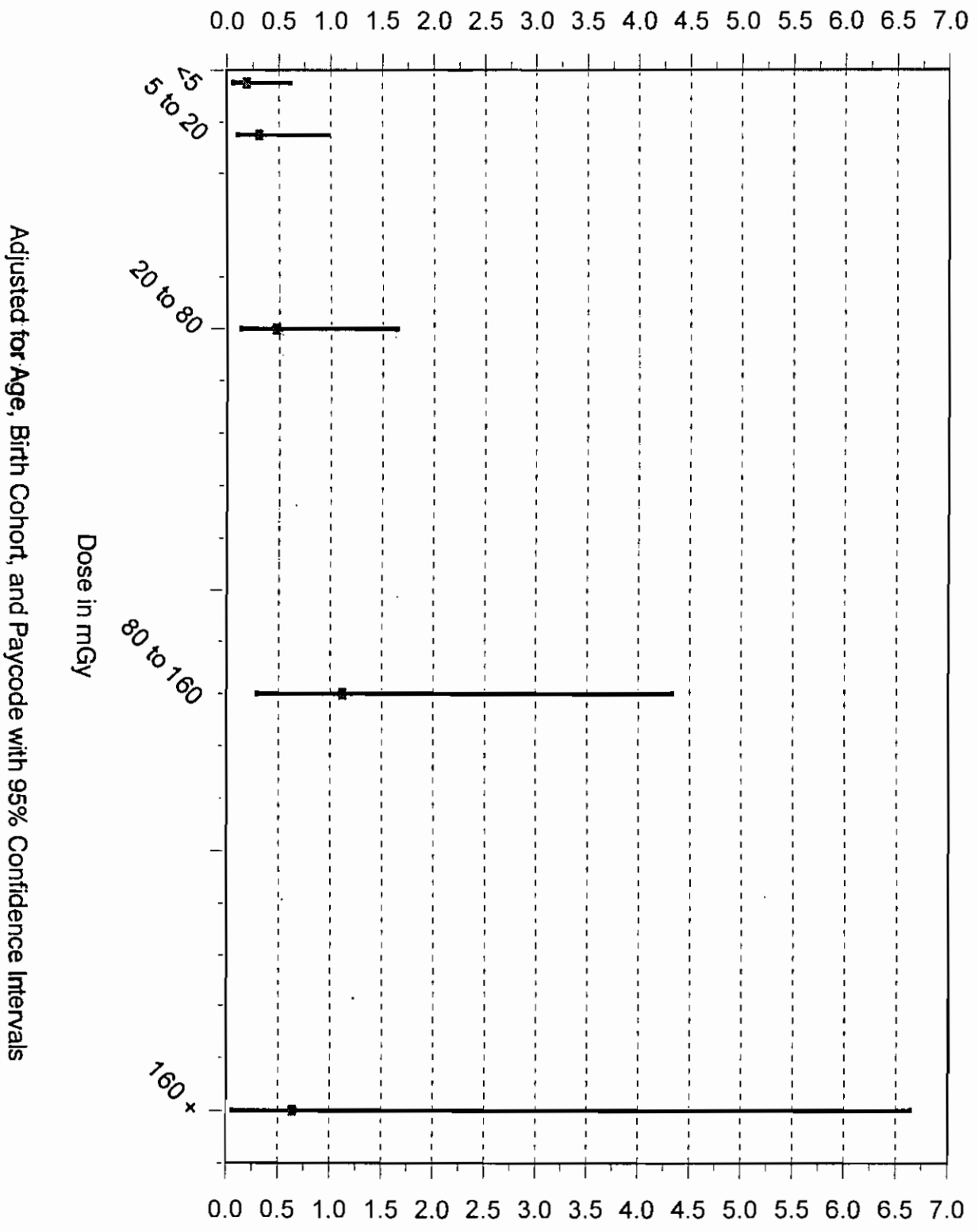
^aTrend test statistic was calculated using average dose per cell where cells were stratified by 15 levels of age, 8 levels of calendar year, and 2 levels of paycode.

^bExpected deaths were calculated based on person-years distribution of age, calendar year, and paycodes for all workers in the study.

Rate per 1000 Person-years



Rate per 1000 Person-years



Fernald Cohort Mortality Study, 1951-1989

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CRAGLE, D.L., WATKINS, J.P., INGLE, J.N., ROBERTSON-DEMERS, K., TANKERSLEY, W.G., and WEST, C.M. Mortality Among a Cohort of White Male Workers at a Uranium Processing Plant: Fernald Feed Materials Production Center, 1951-1989. *Radiat. Res.*

This study followed a cohort of 4,014 white males hired at the Feed Materials Production Center (FMPC) in Fernald, Ohio, between 1951 and 1981. Vital status through the end of 1989 ascertained 1,064 deaths. SMRs stratified by paycode showed a healthy worker effect in salaried, but not hourly employees when compared with mortality rates of U.S. white males. Significant increases were noted for salaried workers for deaths from stomach cancer [SMR=2.61, 90% confidence interval (C.I.) (1.22,5.14)] and for hourly workers for all cancers [SMR=1.21, (1.07,1.37)], lung cancer [SMR=1.26, (1.02,1.54)], and motor vehicle accidents [SMR=1.59, (1.14,2.15)]. Dosimetry data was available for 99% of the study members. Cumulative population doses for internal and external radiation were 163.6 Gray (Gy) and 58.9 Sieverts (Sv). Trend test statistics for 16 selected cancer and three noncancer causes of death revealed a borderline significant trend for lung cancer ($p=.08$) with external dose. Trend tests for nonmalignant respiratory diseases were significant for chronic respiratory diseases ($p=.01$) with internal dose. Dose-response analyses for lung cancer with external dose revealed an excess relative risk per Sv of 8.0 [90% C.I.: (0.1, 18.5)] with a 10 year lag and 10.7 (1.8, 23.7) with a 15 year lag. Models of lung cancer with internal dose revealed no significant relationships. Dose-response analyses for nonmalignant respiratory diseases and internal dose resulted in an excess relative risk per Gy of 13.8 (2.8,45.8) with a 10 year lag and 14.2 (2.2, 44.2) with a 15 year lag.

INTRODUCTION

From 1951 to 1989 the Fernald Feed Materials Production Center (FMPC) processed uranium ore concentrates, uranium of low-grade enrichment, and thorium into fabricated metal products and reactor core target elements for use by the nation's defense programs. Because of the work processes involved, the potential for internal radiation dose from inhalation of uranium dust was higher than the potential for penetrating dose received from external emitting sources.

The objectives of this study were to determine whether there was (1) excess mortality in the cohort compared to the United States general population, and (2) evidence that workers with higher doses of either internal or external penetrating radiation had elevated rates of certain diseases. Only white males were included in this study because they were the majority of the worker population and worked in areas with the greatest potential for radiation exposure.

METHODS

The population was enumerated from original work history records obtained from the facility for the years 1951 through 1989. The data elements available from these records included: birth date, race, gender, pay status, date of first employment, leave of absence dates, termination date, job title, department and plant.

Vital status was determined through searches conducted by the Social Security Administration, Pension Benefits, Inc., and the National Death Index. Death certificates were sought for all individuals where there was an indication of death from any of these sources. All certificates were coded according to the eighth revision of the International Classification of Diseases, Adapted for Use in the United States (ICDA). The underlying cause of death was used for all external analyses comparing the population to national statistics. Non-underlying cancer causes were used for all internal cancer-specific, dose-response analyses comparing exposed

cohort members to unexposed.

Assignment of External Doses

The deep dose equivalents provided by the facility were used for external radiation dose estimates. Weekly external radiation monitoring began at FMPC in 1952 for employees having the highest exposure potential. Employees having a lesser exposure potential were monitored biweekly. By the mid-1950s all workers were monitored monthly until the 1989 shutdown of the facility's operations. No neutron monitoring was performed as the potential for neutron exposure was not present. Film badges were used until 1983 when the site switched to thermoluminescent dosimeters (TLDs). Available exposure records were maintained as an annual cumulative dose equivalent which was rounded to the nearest mSv during the period of film badge use. The cumulative TLD measurements were recorded to the nearest 0.01 mSv with the exception of measurements that were less than 0.05 mSv, which were set to zero.

Monitoring results were not available for 1% of the working years for the cohort. For these missing years, the dose was prorated and assigned based on the dose equivalent for the year immediately preceding or following the missing dose provided similar activities continued. If there was no available information to assign a dose from nearby years, an appropriate median dose was assigned from a comparable population matched to the employee work history (0.5%). The median dose was used rather than the mean to avoid the biasing effect of any unusually high results.

Once the median and nearby dose equivalents were assigned, there were an additional 41 (0.1%) person-years without exposure data. These person-years were individually assigned dose equivalents by a health physicist, considering year of employment, plant, and job title information.

Assignment of Internal Doses

Internal exposure monitoring for radiation first began with a urinalysis program in 1952. Workers were monitored on a nonroutine basis as a control measure and by 1958 urinalysis became a primary means of internal exposure monitoring. This program required quarterly urine sample collection for all process area workers and semi-annual collection for the remaining workers. Beginning in 1968, the Y-12 (Oak Ridge, TN) mobile *In Vivo* Radiation Monitoring Laboratory visited the FMPC site twice a year to perform whole body counts (WBC). Worker WBC monitoring frequency was determined by job potential for exposure and previous results.

Much of the lung exposure was due to relatively insoluble uranium compounds. However, to limit the effect on doses from possible exposures to more soluble forms of uranium a technique was used which was earlier developed by Strom (1) to eliminate aberrantly-high results from both *in vivo* and urinalyses files.

The amounts of uranium (in units of mg) in the lung was calculated from *in vivo* lung count measurements. Urinalyses results were also used to estimate amount of uranium in the lung by a method developed by Strom (1) and later reported by Crawford-Brown (2). Since all internal data were converted to the same unit, the lung burdens estimated from the *in vivo* and urinalysis data were combined to determine the best integrated burden-day estimate for each employee-year of exposure (3). For years in which a worker had no internal monitoring results (6%), doses were estimated using nearby measurements. These reference measurements were taken from similar worker activities within a two year period (4%) surrounding the worker's year of missing results. If there was no available information to estimate a nearby dose (2%), an appropriate median dose was assigned from the population available that best matched the employee work history. The lung dose to uranium was calculated in units of internal radiation dose (mGy) per mg-day of internal exposure by converting these weight measurements into

activity units and assuming a straight line relationship.

Statistical Analyses

Standardized Mortality Ratio (SMR) analyses compared the overall and cause-specific mortality of the hourly and the salaried employees to U.S. white male population rates. Dose-response analyses investigated the relationship between radiation doses and mortality using an internal comparison group. Time-dependent cumulative internal (mGy) and external (mSv) radiation doses were stratified into groups using these intervals: 0, -5, -10, -20, -40, -80, -160, -320, 320+. For external doses the final interval was 160+ mSv since no person-years were assigned to the 320+ interval. For each cell, the mean cumulative dose of all its person-years was used as the dose value in analyses.

The first phase of the dose-response analysis consisted of tests for trend as a screening procedure for a dose-response relationship between selected causes of death and cumulative internal or external doses lagged ten years and also lagged two years for leukemia. Although internal doses were computed based on dose to the lung, these doses would be proportionately scaled for other organ sites. For the purpose of screening in trend tests, they are sufficient for ordering levels of exposure in the population. As detailed in Breslow and Day (4), Poisson trend statistics were calculated stratifying on 15 five-year age groups, eight five-year calendar periods, and two paycode groupings (hourly and salaried) for distributions of person-years at risk. The final phase of the analysis determined dose-response estimates for lung cancer and nonmalignant respiratory diseases. An excess relative risk model was employed utilizing Poisson regression techniques for modeling death rates as a function of dose and covariate values to obtain maximum likelihood estimates of the parameters. Precision of the estimates was indicated by likelihood based 90% confidence intervals. Cumulative doses were accumulated with 10-year and 15-year lags and intervals corresponded to those listed above in the description of the trend

tests. Dose-response estimates were adjusted by including these covariates in the models: age (natural log of age/52.5), birth cohort (1:pre-1920, 2:1920-30, 3:1930+), and paycode (1:hourly, 2:monthly). Dose-response relationships were investigated separately for internal dose and external dose by modeling dose as continuous with the mean cumulative dose in each cell being used as the dose value in the analysis. The resulting parameter estimates were scaled to represent the change in the excess relative risk for each Sv (for external dose) or Gy (for internal dose) increase in dose or, equivalently, the percent change for each 10 mSv or 10 mGy increase in dose.

RESULTS

The cohort consisted of 4,014 white males who entered follow-up between 1951 and 1981 and contributed a total of 121,038 person-years of follow-up through 1989. Of the total, 1,339 (33.4%) were salaried or white collar workers and 2,675 (66.6%) were hourly or blue collar workers. By the end of 1989 a total of 1,064 deaths (26.5% of the cohort) were identified, and 1,053 death certificates (99.0% of the deaths) were obtained.

The average age at hire was 30.7 years with a median of 29.0 years. Over 85% of the cohort was first hired before 1960 allowing 30 or more years of follow-up. This is reflected in the average length of follow-up of 30.2 years and a median of 33.6 years. For length of employment the minimum was three months and the maximum 37.1 years with a mean and median of 8.5 years and 5.5 years.

The SMR analysis results are listed in Table I for both salaried and hourly employees. There were no deaths from bone sarcoma and only one death from chronic nephritis in the population. The salaried employees exhibited the healthy worker effect with significantly decreased SMRs for all causes, and more particularly, all diseases of the circulatory system.

Other significant deficits in mortality for salaried employees occurred for lung cancer, diseases of the respiratory system, diseases of the genitourinary system, and all external causes of death. Only the SMR for stomach cancer was significantly elevated.

Hourly employees had a significant deficit for all diseases of the circulatory system and diseases of the genitourinary system. The all causes SMR did not show a healthy worker effect and this group showed significantly elevated mortality for all cancer deaths. A significant excess of lung cancer deaths was observed in this hourly worker population.

Dose distributions for the cohort by different lag periods and by hourly or salaried work status are shown in Tables II and III. The total population dose for internal radiation and external radiation were 163.6 Gy and 58.9 Sv. Internal doses tended to be higher than external doses, as evidenced in the means (40.9 mGy versus 14.8 mSv) and medians (29.9 mGy versus 2.0 mSv).

Table IV lists trend test statistics for specific cancers by internal dose. Non-Hodgkins lymphoma is of borderline significance but is based on only three cases. Lymphosarcoma and reticulum-cell sarcoma is also of borderline significance; however, the nine cases are all distributed among the lower dose groups below 20 mGy for a ten year lag and below 80 mGy for a two year lag.

Table V displays the results of the trend tests for cancers with external dose. With the exception of a borderline significant trend for lung cancer, none of the cancer causes exhibited a significant trend with increasing external radiation dose. Comparison of the observed to expected ratios by dose group for lung cancer shows that the increased risk occurs beginning in the 40-80 mSv dose group and continues into the higher dose groups.

Trend test results for nonmalignant respiratory disease deaths, along with the acute and chronic subgroups with internal and external doses are shown in Table VI. The acute subgroup includes diseases such as influenza and pneumonia, whereas chronic bronchitis, emphysema,

asthma, chronic interstitial pneumonia, and chronic obstructive pulmonary disease comprises the second subgroup. The trend test for all respiratory diseases together shows a borderline significant trend with internal dose. When subdivided, the trend for acute conditions is not significant. However, the trend for chronic conditions with internal dose is significant with a p-value of 0.01. Analyses using external doses did not reveal any significant relationship with nonmalignant respiratory diseases either as a whole or subgrouped by acute and chronic.

Table VIII displays the results of the excess relative risk modeling analyses for lag periods of 10 and 15 years. Presented are estimates per Gy of internal radiation dose along with likelihood based 90% confidence intervals for lung cancer and nonmalignant respiratory diseases. Confirming the results of the trend tests, there was no statistically-significant relationship between cumulative internal dose and lung cancer. For nonmalignant respiratory diseases both the 10- and 15-year lag models are statistically significant and the estimates of excess relative risk are quite similar. Because the distribution of deaths for the chronic lung diseases subgroup did not include any deaths in the four lowest dose groups, estimates could not be calculated. Also presented are estimates per Sv of external radiation dose for lung cancer. Both the 10- and 15-year lag models showed a significant relationship with the 15-year lag revealing a higher risk.

DISCUSSION

Examination of the results of standardized mortality ratio (SMR) analyses for this population of workers occupationally exposed to uranium dust revealed, in salaried workers, a deficit of deaths from all causes combined. This healthy worker effect was not evident in the hourly workers and was an unusual finding for an occupational epidemiologic study focused on a cohort where hiring first occurred in the 1950s and later. The SMR for deaths from diseases of the circulatory system was significantly decreased for both the salaried and hourly employees reflecting evidence of the healthy worker effect for cardiovascular diseases. The significant

increase in deaths among hourly workers from cancers is the result of generalized increases in deaths in most cancer categories rather than a large increase in a single category. The increases in cancer deaths among hourly employees included 14 of the 17 categories examined with the exceptions of cancer of the stomach, bone sarcoma, and cancer of the skin.

It is important to note that this population of radiation exposed workers exhibited neither excess leukemia nor a dose-response relationship between leukemia (excluding two observed CLLs) and either internal or external radiation dose. The total population dose for external radiation was 58.9 Sv with the average cumulative dose to a worker of 14.7 mSv, thus limiting the ability to detect such a dose-response relationship with external radiation.

Examination of the excess of stomach cancer deaths in the salaried employees reveals the majority of these deaths occurred in individuals who held administrative positions, and had very low potential for exposure to either internal or external radiation, or chemicals. Increases in stomach cancer have been observed in other populations where the primary exposure was to internal radiation; however, in the FMPC population there was no evidence of a dose-response relationship with either internal or external radiation.

An excess of lung cancer was observed in the hourly worker population when compared with the United States standard population. Lung cancer is not generally recognized as a consequence of external radiation exposure, whereas it might be expected as a consequence of chronic exposure to uranium dust. Published results from combined worker studies (5) did not demonstrate a trend for external dose with lung cancer, but rather revealed a negative trend. However, for the Fernald cohort the trend test for lung cancer with external dose was of borderline significance, and the dose-response estimate for lung cancer was also revealed as statistically significant. The major difference between the populations in the combined worker population study and the present study is the additional possibility of exposure to internal

radiation in the Fernald cohort. A study by Checkoway (6) investigated a population of workers who had similar types of exposures as the present population. The Checkoway study found a relationship between external dose and lung cancer in workers who had more than 5 mGy cumulative internal dose using a zero and a 10-year lag (the Checkoway study used a quality factor of 10 for the internal dose which has been removed here for the purpose of comparison). Examination of the Fernald population dose distribution with a 10-year lag reveals that 84% of the hourly workers and 67.7% of the salaried workers have more than 5 mGy of internal dose.

In a recent analysis of mortality among white male workers at the Los Alamos National Laboratory (7) a subgroup of workers who were monitored for plutonium exposure was examined. Because only 303 of the workers in this monitored group actually had doses from plutonium, it was possible to compare death rates in the plutonium-exposed monitored workers with those in the plutonium-unexposed monitored workers. The rate ratio for lung cancer comparing exposed to unexposed workers was 1.78 (95% confidence interval 0.79 to 3.99, based on eight cases). The next analysis excluded workers who had a cumulative external radiation dose of 10 mSv or more in order to remove confounding from external dose. This analysis showed a rate ratio for lung cancer of 1.04 (95% confidence interval 0.20 to 3.57). Although the numbers are small, this study may provide more evidence in worker populations that internal radiation dose may facilitate the effectiveness of external radiation dose in carcinogenesis.

A case-control study of 787 lung cancer among workers employed in four uranium processing or fabrication operations (8) did not find any dose-response effect for internal dose to the lung. External radiation exposure was available for 105 of the case-control pairs so that an analysis of the joint effect of internal and external dose could be completed. The odds ratios increased with level of external exposure in those who had more than 5 mGy internal dose, although the odds ratios were not significant. For categories of external radiation of <10 mSv,

10-49 mSv, and ≥ 50 mSv, the odds ratios were 0.64, 1.03, and 1.18.

Results of studies on uranium miners are not directly comparable to the study of the Fernald population because the uranium miners were exposed to radon progeny from the decay of radium while the Fernald workers were primarily exposed to uranium compounds.

Figures 1 and 2 display the mortality rates with 95% confidence intervals for separate dose categories in mGy (Figure 1) and mSv (Figure 2) as follows: below 5, 5 to less than 20, 20 to less than 80, 80 to less than 160, and 160 or greater. Some dose categories collapsed because of small numbers in order to create the figures. Figure 1 shows the relationship between cumulative internal dose and nonmalignant respiratory diseases while Figure 2 shows cumulative external dose and lung cancer. Although there is a suggestion of dose-response relationships in these two graphs, which was also demonstrated in trend analyses and the ERR modeling, the wide confidence intervals illustrate the extent of the uncertainty involved.

The Fernald population was also exposed to multiple chemical exposures, most notably nitric acid, sodium hydroxide, tributyl phosphate, trichloroethylene, and kerosene. It is known that the level of these chemical exposures would also increase as the level of external or internal radiation dose increased. Therefore, it is possible that some of the excess risk observed for lung cancer and NMRD may actually be caused by these chemical exposures or caused by a potentiating role of the chemical exposures making the radiation exposures more effective.

The significant dose-response relationship for non-malignant respiratory disease (NMRD) in this population was previously described (9) although the magnitude of the relationship was not calculated per unit dose. The earlier study used a semiquantitative ranking scheme to place workers in different levels of potential for exposure to uranium dust and found that the relative risk of NMRD increased with increasing cumulative uranium dust exposure.

The absolute values of the dose-response estimates for this population must be viewed cautiously because of the inextricable nature of the doses for internal and external radiation. Table IX displays the population average internal dose for each external dose group and the population average external dose for each internal dose group. As external dose increases, so does internal dose in a somewhat steady manner indicating a dose-response observed for external radiation may be due to internal dose. The measurement of internal dose is less straightforward than the measurement of external dose. For this population, it is possible, a level of inaccuracy is associated with the placement of workers into internal dose categories.

The average external dose for each internal dose category fluctuates and is not nearly as straightforward as the internal dose for external-dose category comparison. This might indicate whatever dose response is observed for internal radiation-dose categories is not likely to be due to the level of the external dose whereas a dose response observed for external dose may be highly related to internal dose.

We conclude that there is evidence of a radiation dose-response relationship in this population for both non-malignant respiratory disease and lung cancer. These findings were produced without controlling for other lung carcinogens that were likely present in the work environment at this facility. Inclusion of other confounding exposures may have altered the findings presented here.

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The submitted manuscript has been authored by a contractor of the U.S. Government under Contract No. DE-AC05-76OR00033. Accordingly, the U.S. Government retains a nonexclusive, royalty-free license to publish or reproduce the published form of this contribution, or allow others to do so for U.S. Government purposes.

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TABLE I
SMRs through 1989 Stratified by Paycode

Cause of death	Salaried (N = 1,339)			Hourly (N = 2,675)		
	Observed	SMR	95% C.I.	Observed	SMR	95% C.I.
All causes (001-998)	250	0.71 ^{29%}	(0.62,0.80)	814	0.95	(0.88,1.01)
All cancers (140-209)	83	0.99	(0.79,1.23)	249	1.21 ^{21%}	(1.07,1.37)
All infective and parasitic diseases (001-139)	2	0.56	(0.06,2.02)	3	0.35	(0.07,1.02)
Cancer of buccal cavity and pharynx (140-149)	2	0.82	(0.09,2.95)	7	1.15	(0.46,2.38)
Cancer of digestive organs and peritoneum (150-159)	26	1.26	(0.83,1.85)	61	1.19	(0.91,1.53)
Cancer of stomach (151)	8	2.61 ^{161%}	(1.22,5.14)	7	0.91	(0.37,1.88)
Cancer of large intestine (153)	9	1.24	(0.57,2.36)	19	1.06	(0.64,1.66)
Cancer of rectum (154)	0	(1.85) ^a		7	1.50	(0.60,3.10)
Cancer of pancreas (157)	5	1.18	(0.38,2.76)	13	1.24	(0.66,2.12)
Cancer of respiratory system (160-163)	21	0.65 35%	(0.41,1.00)	99	1.25	(1.02,1.53)
Cancer of lung (162)	19	0.62 ^{35%}	(0.37,0.97)	95	1.26	(1.02,1.54)
Bone sarcoma (170)	0	(0.28) ^a		0	(0.66) ^a	
Cancer of skin (172-173)	3	1.61	(0.21,2.92)	1	0.24	(0.00,1.32)
Cancer of prostate (185)	9	2.02	(0.92,3.84)	16	1.40	(0.80,2.28)
Cancer of bladder (188)	0	(1.86) ^a		8	1.69	(0.73,3.32)
Cancer of brain and other central nervous system (191-192)	2	0.72	(0.08,2.60)	10	1.54	(0.74,2.83)
Leukemia and aleukemia (204-207)	5	1.59	(0.51,3.71)	8	1.07	(0.46,2.11)
Other lymphatic tissue (202,203,208)	2	0.71	(0.08,2.57)	8	1.18	(0.51,2.33)
All lymphopoietic cancer (200-209)	10	1.21	(0.58,2.22)	28	1.42	(0.94,2.05)
Benign neoplasms (210-239)	0	(1.04) ^a		1	0.40	(0.01,2.23)
All diseases of circulatory system (390-458)	100	0.62 ^{36%}	(0.50,0.75)	360	0.88 ^{2%}	(0.79,0.98)
Chronic rheumatic heart (393-398)	4	1.55	(0.42,3.96)	9	1.39	(0.63,2.63)

TABLE I
(continued)

Cause of death	Salaried (N = 1,339)			Hourly (N = 2,675)		
	Observed	SMR	95% C.I.	Observed	SMR	95% C.I.
All vascular lesions of CNS (430-438)	12	0.74	(0.38,1.30)	36	0.88	(0.62,1.22)
All respiratory diseases (460-519)	9	0.42	(0.19,0.80)	44	0.82	(0.60,1.10)
All diseases of digestive system (520-577)	17	0.97	(0.56,1.55)	32	0.76	(0.52,1.07)
Cirrhosis of liver (571)	14	1.30	(0.71,2.19)	20	0.79	(0.48,1.22)
Diseases of the genitourinary system (580)	0	(3.84) ^a		3	0.32	(0.06,0.92)
Chronic nephritis (582)	0	(0.98) ^a		1	0.42	(0.01,2.32)
Symptoms, senility, and ill-defined conditions (780-799)	1	0.23	(0.00,1.30)	4	0.39	(0.11,1.01)
All external causes of death (800-998)	23	0.57 ^{37%}	(0.36,0.86)	89	1.07	(0.86,1.32)
All accidents (801-929)	13	0.50	(0.26,0.85)	67	1.23	(0.95,1.56)
Motor vehicle accidents (810-827)	8	0.61	(0.26,1.21)	41	1.59 ^{59%}	(1.14,2.15)

^aExpected number of deaths.

TABLE II

Distribution of Internal Dose (mGy) Category by Lag and Paycode

DOSE	LAG 0 ^a				LAG 10				LAG 15			
	Hourly		Salaried		Hourly		Salaried		Hourly		Salaried	
	N	%	N	%	N	%	N	%	N	%	N	%
0	90	(3.4)	55	(4.1)	188	(7.0)	112	(8.4)	271	(10.1)	148	(11.0)
>0-5	234	(8.7)	339	(25.3)	237	(8.8)	320	(23.9)	225	(16.8)	304	(22.7)
>5-10	224	(8.4)	173	(12.9)	220	(8.2)	167	(12.5)	207	(15.4)	160	(11.9)
>10-20	319	(11.9)	209	(15.6)	305	(11.4)	201	(15.0)	303	(11.3)	204	(15.2)
>20-40	466	(17.4)	261	(19.5)	466	(17.4)	257	(19.2)	470	(17.6)	261	(19.5)
>40-80	807	(30.2)	254	(19.0)	778	(29.1)	240	(17.9)	761	(28.4)	224	(16.7)
>80-160	480	(17.9)	44	(3.3)	434	(16.2)	38	(2.8)	399	(14.9)	34	(2.5)
>160-320	49	(1.8)	3	(0.2)	41	(1.5)	3	(0.2)	33	(1.2)	3	(0.2)
>320	6	(0.2)	1	(0.1)	6	(0.2)	1	(0.1)	6	(0.2)	1	(0.1)

^amean 40.9 mGy

median 29.9 mGy

max 598.4 mGy

Total 163.6 Gy

TABLE III

Distribution of External Dose (mSv) Category by Lag and Paycode

DOSE	LAG 0 ^a				LAG 10				LAG 15			
	Hourly		Salaried		Hourly		Salaried		Hourly		Salaried	
	N	%	N	%	N	%	N	%	N	%	N	%
0	631	(23.6)	670	(50.0)	721	(26.9)	705	(52.6)	794	(29.7)	739	(55.2)
>0-5	678	(25.3)	497	(37.1)	663	(24.8)	470	(35.1)	661	(24.7)	447	(33.4)
>5-10	242	(9.0)	82	(6.1)	235	(8.8)	77	(5.8)	238	(8.9)	70	(5.2)
>10-20	284	(10.6)	44	(3.3)	267	(10.0)	46	(3.4)	250	(9.3)	50	(3.7)
>20-40	358	(13.4)	32	(2.4)	347	(13.0)	29	(2.2)	336	(12.6)	23	(1.7)
>40-80	317	(11.8)	13	(1.0)	310	(11.6)	11	(0.8)	288	(10.8)	10	(0.7)
>80-160	142	(5.3)	1	(0.1)	119	(4.4)	1	(0.1)	103	(3.8)	0	(0.0)
>160-320	23	(0.8)	0	(0.0)	13	(0.5)	0	(0.0)	5	(0.2)	0	(0.0)
>320	0	(0.0)	0	(0.0)	0	(0.0)	0	(0.0)	0	(0.0)	0	(0.0)

^amean 14.8 mSv

median 2.0 mSv

max 277.0 mSv

TOTAL 58.9 Sv