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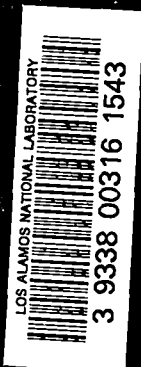
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Supplementary Documentation for an Environmental Impact Statement Regarding the Pantex Plant

Occupational Work Force Mortality Study

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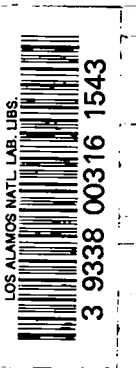
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SUPPLEMENTARY DOCUMENTATION FOR AN ENVIRONMENTAL IMPACT STATEMENT
REGARDING THE PANTEX PLANT:

OCCUPATIONAL WORK FORCE MORTALITY STUDY

by

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ABSTRACT

   This report documents work performed in support of an Environmental
Impact Statement (EIS) regarding the Department of Energy's Pantex Plant near
Amarillo, Texas. We compared total and cause-specific mortality for Pantex
Plant workers employed between 1951 and December 31, 1978, with expected
mortalities based on US death rates. We observed significantly fewer deaths
than expected from all causes of death: all cancers (digestive cancers and
lung cancer), arteriosclerotic heart disease, and digestive diseases. No
causes of death occurred significantly more frequently than expected.
Analyses of worker mortality by duration of employment, time since first
employment, and radiation exposure greater than 1.00 rem produced similar
results. We found no evidence that mortality from any cause of death was
increased as a result of employment at the Pantex Plant.

I. INTRODUCTION

   This report documents work performed in support of preparation of an
Environmental Impact Statement (EIS) regarding the Department of Energy's
(DOE) Pantex Plant, near Amarillo, Texas. The EIS addresses continuing
nuclear weapons operations at Pantex and the construction of additional
facilities to house those operations. The EIS was prepared in accordance
with current regulations under the National Environmental Policy Act.
Regulations of the Council on Environmental Quality (40 CFR 1500) require
agencies to prepare a concise EIS with fewer than 300 pages for complex
projects. This report was prepared by Los Alamos National Laboratory to
document details of work performed and supplementary information considered
during preparation of the Draft EIS.

II. BACKGROUND ON THE PANTEX PLANT

The Pantex Plant, located in the Panhandle of Texas in Carson County near Amarillo, was first used in 1942 by the Army Ordnance Corps for loading conventional ammunition shells and bombs. In 1950 the Atomic Energy Commission started rehabilitating portions of the original plant and building new facilities for nuclear weapons operations.

The Pantex Plant is primarily an assembly facility that receives conventional high-explosive materials (conventional means nonnuclear) and prefabricated weapons components from external suppliers. There are three major operations: (1) production of new nuclear weapons; (2) maintenance, modification, and quality assurance testing of nuclear weapons already in the military stockpile; and (3) retirement by disassembly of nuclear weapons no longer required in the military stockpile.

The high explosives shipped to the Pantex Plant are fabricated into the required shapes for use in nuclear weapons. All other nuclear weapon components are supplied by other manufacturers. At the Pantex Plant these components are assembled to produce nuclear weapons for delivery to the Department of Defense.

Maintenance and modification of weapons in the military stockpile involve partial disassembly to permit replacement, modification, or inspection of components. A statistically selected number of nuclear weapons from the military stockpile or from initial production of a new weapons system receive a series of inspections and component evaluations.

When a weapon is completely disassembled for retirement, the conventional high-explosive components are separated from the nuclear components and disposed of at the Pantex Plant by burning. The nuclear materials components are returned to the original manufacturer. All other components are returned to the manufacturers or sent elsewhere for reuse, salvage, or ultimate disposal.

The Pantex Plant also conducts research and development work on conventional high explosives to support weapons design and development programs for the DOE.

III. STUDIES OF OTHER NUCLEAR WORKER POPULATIONS

This analysis of worker mortality at the Pantex Plant is one of several recent studies of nuclear facility workers, some of whom are exposed to low-level radiation. In all these studies, workers with radiation exposure and/or limited contact with specific chemicals are of particular interest. Numerous studies of populations with relatively high radiation exposure (i.e., >100 rad) have demonstrated higher rates of leukemia and cancers of

the lung, thyroid, female breast, and other sites (BEIR III 1980). Although these studies provide some theoretical background for the present study, other investigations of nuclear worker cohorts exposed to low levels of ionizing radiation provide a more reasonable basis for a priori expectations about mortality within the Pantex Plant cohort (Gilbert 1979, Rinsky 1981, Hadjimichael 1983, and Voelz).

Gilbert and Marks (1979) investigated cause-specific mortality among more than 17 000 workers with at least 2 years' employment at the Hanford nuclear facility. In general, workers had significantly fewer cancer and noncancer deaths than were expected based on US death rates. A significant increase in pancreatic cancer and multiple myeloma mortality was found with increasing radiation exposure levels from 0 to 2 rem, 2 to 5 rem, 5 to 15 rem, and 15+ rem. However, other cancers previously associated with radiation at high doses were less frequent than expected and did not increase with increasing radiation dose.

Rinsky (1981) reported on mortality among workers at the Portsmouth nuclear naval shipyard. This cohort included approximately 24 000 workers, 7 616 with measured radiation exposures ranging from 0.001 to 91 rem. For the total cohort, mortality from all causes was significantly less than was expected based on US rates, and no difference existed between observed and expected mortality for all cancers or lymphatic and hematopoietic neoplasms. There were no trends or radiation dose response relationships for any cause of death. Mortality rates for radiation workers and nonradiation workers were similar. However, further analyses did provide suggestive evidence of a lung cancer excess for workers with more than 1 rem cumulative exposure (Rinsky 1983). However, the implications of this finding are unclear because information on cigarette smoking, the major risk factor for lung cancer, was not available for this analysis.

Cancer mortality and morbidity among workers at a nuclear fuels fabrication plant have been evaluated by Hadjimichael (1983). In this study, the standardized mortality ratio (SMR) for all causes of death was significantly lower than expected, and there were no significant mortality findings for any cancer site. There was a significant excess of mortality from central nervous system and peripheral diseases (ICD 340-359)* and obstructive pulmonary disease among industrial workers. Analyses of cancer incidence showed fewer total cancers than expected, but there was a significant excess of brain cancer. This brain cancer excess was not found to be associated with radiation or other occupational exposures.

Recently, mortality for approximately 7 000 white males at the Rocky Flats nuclear weapons facility was analyzed (Voelz). This cohort was particularly interesting because workers had the potential for internal

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*International Classification of Diseases, 8th Revision.

radiation exposure from plutonium (an alpha-emitting radionuclide) as well as external radiation exposure. Further, studies of these workers are particularly relevant to the present study because Rocky Flats provides nuclear components for the assembly process at the Pantex Plant. Overall, mortality among Rocky Flats workers was significantly less than expected based on US death rates. Mortality was significantly more frequent than expected only for benign and unspecified neoplasms (ICD 210-239),* all of which were found to be intracranial. In further analyses, cancer mortality was not elevated among workers with exposure to either plutonium or external radiation. The excess of benign intracranial tumors was not associated with plutonium exposure but was centered among workers with more than 100 mrem cumulative external radiation exposure.

Taken together, these studies of nuclear workers exposed to low levels of ionizing radiation demonstrate less total mortality and usually less total cancer mortality than expected. Moreover, there has been no consistent pattern of excess mortality for any cancer site. There have been instances of significant findings for multiple myeloma, benign and unspecified brain tumors, and cancers of the brain and pancreas. Therefore, these diseases were of particular interest in our analysis of mortality among Pantex Plant workers.

A. Occupational Environment and Radiation Personnel Dosimetry

The Pantex Plant is a chemical high-explosive fabrication and nuclear weapons assembly plant. Operations are contained in large zones, which are separated to isolate accidental explosions and prevent sympathetic detonations. Industrial hygiene, health, and safety measures center on preventing explosions during fabrication; pressing or machining of high-explosive materials; and minimizing solvent, chemical, and radiation exposures.

Many solvents and other chemicals are used in high-explosive fabrication and related processes (Macdonell 1982). Worker exposures are controlled through minimizing the amount of chemicals allowed in work areas, specialized local and remote containment vessels, and use of local exhaust ventilation. Use of a suspected carcinogen requires special authorization procedures, and these procedures can only be handled by trained personnel. Workplace area air sampling for hazard evaluation was done infrequently before 1973, but it has been done more regularly since then. These earlier records were unavailable for review. However, review of a sample of the more recent workplace air sampling records did not find any instances of noncompliance with applicable occupational standards.

Workers may receive low-level external radiation exposure from nuclear components (gamma or neutron) or industrial radiographic equipment (x ray)

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*International Classification of Diseases, 8th Revision.

(Elder 1982). Lifetime film badge measurements were available for workers employed during and after 1963, but they were unavailable for workers who terminated before 1963. A review of available radiation exposure records from 1963-1980 showed that Pantex Plant workers had lower average exposures than did workers at other nuclear facilities (Elder 1982). Table I presents a breakdown of cumulative radiation exposures through 1980 at the Pantex Plant.

IV. METHODS

The study cohort included all white males employed between the start of plant operations in 1951 and the end of the study on December 31, 1978. There were 2 686 white male employees and 154 nonwhite male employees listed by race in the personnel records. Another 878 males did not have race specified in their records, and for this study, we classified all these workers as white. Thus, the roster of eligible employees included 3 564 white males. Females (892) and nonwhites were excluded from the analysis because of their small numbers and few deaths (19 deaths and 4 deaths, respectively).

Personnel records for all eligible employees were double coded for name, sex, date of birth, social security number, date of hire, and date of termination. Cause of death, coded to the ICD8 revision by an independent nosologist, and date of death were also coded for deceased study subjects. Seven employees had missing information for birth date, date of hire, and/or date of termination in their personnel records. They were deleted from our study roster. We edited a random 5% sample of records in our analytic file

TABLE I

CUMULATIVE WHOLE BODY RADIATION DOSES FOR PANTEX PLANT WORKERS

rem	Number	Percentage 1963-1980	Percentage Total
0.00	1173	55.3	32.9
0.00 - 1.00	696	32.8	19.5
1.00 - 4.99	212	10.0	5.9
5.00+	40	1.9	1.1
Unknown*	1443	--	40.5
Total	3564	100.0	99.9

*Workers terminated before 1963.

and found no errors for the date of birth, social security number, sex, race, ICD8, and death date (upper 95% confidence interval (CI) 1.3% error). An error rate of 0.7% (95% CI 0.1-2.6%) was found for employment termination dates, whereas the error rate for the date of hire was 1.1% (95% CI 0.2-3.1%).

To verify the completeness of our study cohort, we checked our roster against 941A social security reports, health physics records, pension records, security clearance listings, current and former payroll records, and a listing of security terminations. Only two additional employees were added to the cohort through this process. Accordingly, we consider the study cohort to be completely enumerated.

We submitted a computerized cohort listing to the Social Security Administration (SSA) as the first step in determining employee vital status. SSA provided this information for approximately 90% of study subjects. Plant and pension records and the Texas Department of Motor Vehicles were searched for individuals not identified as alive or deceased by SSA. Telephone tracing was then attempted for individuals whose vital status remained unknown. The combination of these activities yielded vital status for approximately 97% of our study cohort. We were able to obtain death certificates for 257 of the 269 known employee deaths (96%). Deceased employees whose death certificates were not located were counted as deaths in our analysis, but death was not assigned to a specific cause. This procedure will result in minor underestimation of worker mortality from a few causes of death.

We evaluated total and cause-specific mortality among workers by comparing the number of observed deaths with the number of expected deaths based on US death rates. This comparison is typically expressed as the SMR, which is age- and calendar-period-adjusted according to the proportion of employee person-years in each 5-year age and calendar period. Statistical significance of individual SMRs was evaluated according to the associated 95% CI, which was judged to be significant if it did not include the null value of 1.00. Fisher's exact CI was used when the number of observed or expected deaths was less than five. Otherwise, 95% approximate CIs were calculated (Rothman 1979).

Employee person-years were counted from date of first employment until date of death (if within the study period) or December 31, 1978. Workers who terminated and were lost to follow-up contributed person-years only until their employment termination date. For analyses by duration of employment or time since first employment, person-years were counted only while the worker remained in the specified time interval. For example, our analysis of Pantex workers with 5-10 years duration of employment is based on deaths and person-years that occurred while each worker had worked more than 5 years but less than 10 years. Accordingly, a person employed at the Pantex Plant for 7

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years, from January 1, 1955, until December 31, 1962, would contribute 5 person-years from his hire date until December 31, 1960, to the 0-5 year duration of employment category. He would then contribute up to 18 person-years to the 5-10 year category from January 1, 1961, until his death date or December 31, 1978. The important point is that the employee must live through his first 5 years of employment to be included in the 5-10 year category. Therefore, we do not count any of his first 5 "immortal" person-years in the 5-10 year duration analysis to avoid incorrectly increasing the number of expected deaths and thereby underestimating the 5-10 year SMR. Similar considerations govern the allocation of employee person-years and deaths in the time since first employment analyses.

SMRs were also calculated for workers whose cumulative radiation exposure exceeded 1.00 rem. Because radiation records were not available for persons who terminated before 1963, person-years at risk for this analysis were counted after attainment of a 1-rem exposure level or from 1963 for workers who received 1 rem before 1963.

V. RESULTS

Some general characteristics of the Pantex Plant work force by decade of hire are detailed in Table II. Our study cohort consists of 3 564 white males of whom 269 (7.5%) were deceased. The average age at hire was 32.1 years and was consistent over the three decades included in this study. Average age at employment termination through our end-of-study date (December 31, 1978) was 40.1 years for the entire cohort and tended to be older among workers hired near the beginning of plant operations. The average duration

TABLE II

SELECTED CHARACTERISTICS OF WHITE MALE PANTEX PLANT WORKERS
BY DECADE OF HIRE

	1951-1959	1960-1969	1970-1978	Total
Number of workers	1522	1057	985	3564
Deaths	229	34	6	269
Average age at hire	32.8	31.6	31.6	32.1
Average age at termination	43.9	39.1	35.1	40.1
Average duration of employment	11.0	7.6	3.5	7.9
Average years of follow-up	24.5	13.3	4.8	15.1

of employment was 7.9 years for all workers and 11.0 years for the earliest workers. Length of follow-up averages 15.1 years for study subjects. Among the three subgroups, the longest follow-up is for workers first employed between 1951-1959, or 24.5 years. These data indicate that the majority of cohort members are relatively young and therefore unlikely to have died of cancer or other chronic diseases. Accordingly, we should expect a larger healthy worker effect in our cohort mortality analysis than that seen in occupational studies with longer follow-up, because the selective survival advantage of working populations is greatest at young ages and tends to diminish as cohort members near retirement age (Goldsmith 1975, McMichael 1975).

An analysis of observed and expected deaths from specific causes, with at least one death among Pantex Plant workers, is contained in Table III. Overall, SMRs were lower than expected for nearly every cause of death. In particular, deaths in the aggregate categories, all causes, all cancers, and circulatory diseases, were significantly fewer than expected. Cause-specific mortality from digestive cancers, lung cancer, arteriosclerotic heart disease, and digestive diseases also occurred significantly less frequently than expected. There were no other significant findings for the remaining cause-of-death categories.

Table IV provides an analysis of cause-specific worker mortality by 5-year duration-of-employment categories. This type of analysis is useful for investigating nonspecific workplace disease risk factors, for which length of employment would be highly correlated with amount of exposure. Again, SMRs are less than 1.00 for the majority of disease/duration of employment categories. Of particular importance is the significant deficit of observed all-cause mortality (SMR = 0.65, 95% CI 0.48-0.87%) for workers with 15 or more years employment at the Pantex Plant. Further, for this group and throughout the entire analysis, there were neither instances of significantly elevated mortality nor trends of increasing mortality with increasing duration of employment. The low observed-to-expected mortality ratio seen in the general cohort SMR analysis seems to hold regardless of duration of employment.

Table V presents an analysis of worker mortality by the amount of time since their initial employment at the Pantex Plant. This type of analysis is usually employed to discern occupation-related health effects that require years to develop after workplace exposures (e.g., cancers, chronic respiratory disease, etc.). The three 10-year categories correspond roughly to the year-of-hire periods 1970-1978, 1960-1969, and 1951-1959. Again, the majority of SMRs are less than 1.00, and there are no major SMR increments with increasing time since first employment. More important, mortality for the subgroup with the longest period since first employment is consistent with the expected values. SMRs do tend to increase slightly for all causes of death and all cancers as time since first employment increases. This

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TABLE III

COMPARISON OF OBSERVED AND EXPECTED DEATHS AT THE
PANTEX PLANT--WHITE MALE EMPLOYEES 1951-1978

Cause (ICD8)	OBS*	EXP**	SMR***	95% CI+
All causes (1-999)	269	373.71	0.72	0.64-0.81
All cancers (140-209)	44	72.99	0.60	0.44-0.81
Oral cancer (140-149)	1	2.48	0.40	0.01-2.25
Digestive cancers (150-159)	8	18.82	0.43	0.18-0.84
Stomach (151)	1	3.38	0.30	0.01-1.65
Large intestine (153)	4	6.01	0.67	0.18-1.70
Rectum (154)	1	2.11	0.47	0.01-2.64
Liver (155)	1	1.22	0.82	0.02-4.57
Pancreas (157)	1	3.91	0.26	0.01-1.43
Lung cancer (162-163)	12	24.40	0.49	0.25-0.86
Prostate cancer (185)	2	2.91	0.69	0.08-2.48
Bladder cancer (188)	1	1.71	0.59	0.02-3.26
Kidney cancer (189)	1	1.96	0.51	0.01-2.84
Brain cancer (191,192)	4	2.95	1.36	0.37-3.47
Lymphosarcoma (200)	2	1.88	1.06	0.13-3.84
Hodgkins (201)	1	1.35	0.74	0.02-4.13
Leukemia (204-207)	4	3.13	1.28	0.35-3.27
All lymphopoietic cancer (200-209)	7	8.22	0.85	0.34-1.76
Benign neoplasms (210-239)	1	1.11	0.90	0.02-5.02
Diabetes mellitus (250)	3	5.14	0.58	0.12-1.71
Circulatory diseases (390-458)	133	174.74	0.76	0.64-0.90
Arteriosclerotic heart disease (410-413)	97	129.33	0.75	0.61-0.91
Cerebrovascular diseases (430-438)	18	19.37	0.75	0.55-1.47
Respiratory diseases (460-519)	11	18.79	0.59	0.29-1.05
Digestive disease (520-577)	10	21.63	0.46	0.22-0.85
External causes (800-998)	43	55.94	0.77	0.56-1.04

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*Number of observed deaths among Pantex Plant employees during 1951-1978.
**Number of expected deaths based on race, sex, and age-specific US death rates 1951-1978.
***SMR is the ratio of observed deaths to expected deaths.
+Exact Fischer 95% CI (Rothman 1979).

TABLE IV

COMPARISON OF OBSERVED AND EXPECTED DEATHS BY DURATION
OF EMPLOYMENT AT THE PANTEX PLANT--WHITE MALE EMPLOYEES 1951-1978

| | Observed Deaths and SMRs | | | | | | | | | | | |
| Cause of Death | Employed for <5 Years | | | Employed for 5-10 Years | | | Employed for 10-15 Years | | | Employed for 15+ Years | | |
	OBS*	SMR**	(95% CI)***	OBS*	SMR**	(95% CI)***	OBS*	SMR**	(95% CI)***	OBS*	SMR**	(95% CI)***
All causes	154	0.80	(0.68-0.94)	30	0.50	(0.34-0.72)	38	0.77	(0.55-1.06)	47	0.65	(0.48-0.87)
All cancers	20	0.56	(0.34-0.86)	5	0.44	(0.14-1.03)	4	0.42	(0.11-1.06)	15	0.93	(0.52-1.54)
Digestive cancers	5	0.54	(0.17-1.26)	1	0.34	(0.01-1.92)	0	0.00	(0.00-1.45)	2	0.49	(0.01-1.76)
Lung cancer	2	0.17	(0.02-0.63)	2	0.55	(0.07-1.97)	2	0.62	(0.08-2.25)	6	1.00	(0.37-2.18)
Brain cancer	4	2.54	(0.69-6.52)	0	0.00	(0.00-7.38)	0	0.00	(0.00-10.25)	0	0.00	(0.00-7.23)
Leukemia	1	0.60	(0.02-3.34)	1	1.96	(0.05-10.93)	1	2.50	(0.06-13.93)	1	1.79	(0.05-9.95)
All lymphopoietic cancer	3	0.69	(0.14-2.01)	1	0.74	(0.02-4.10)	1	0.98	(0.03-5.46)	2	1.35	(0.16-4.88)
Arteriosclerotic heart disease	51	0.81	(0.60-1.06)	13	0.65	(0.35-1.11)	14	0.77	(0.42-1.30)	19	0.68	(0.41-1.06)
Respiratory disease	4	0.44	(0.12-1.12)	2	0.71	(0.09-2.55)	5	1.88	(0.61-4.39)	0	0.68	(0.00-0.89)
Digestive disease	7	0.63	(0.25-1.30)	1	0.27	(0.01-1.54)	1	0.35	(0.01-1.95)	1	0.25	(0.01-1.37)

*Number of observed deaths among Pantex Plant employees during 1951-1978.
**SMR is the ratio of observed deaths to expected deaths.
***Exact Fischer 95% CI (Rothman 1979).

TABLE V

COMPARISON OF OBSERVED AND EXPECTED DEATHS BY TIME SINCE
FIRST EMPLOYMENT AT THE PANTEX PLANT--WHITE MALE EMPLOYEES 1951-1978

| | Observed Deaths and SMRs | | | | | | | | |
| | <5 Years Since First Employment | | | 5-10 Years Since First Employment | | | 10-15 Years Since First Employment | | |
Cause of Death	OBS*	SMR**	(95% CI)***	OBS*	SMR**	(95% CI)***	OBS*	SMR**	(95% CI)***
All causes	62	0.50	(0.38-0.64)	121	0.78	(0.65-0.93)	86	0.91	(0.73-1.13)
All cancers	8	0.38	(0.17-0.76)	21	0.68	(0.42-1.04)	15	0.71	(0.40-1.18)
Digestive cancers	0	0.00	(0.00-0.69)	6	0.76	(0.27-1.62)	2	0.37	(0.04-1.32)
Large intestine	0	0.00	(0.00-2.32)	3	1.17	(0.24-3.42)	1	0.53	(0.01-2.98)
Lung cancer	0	0.00	(0.00-0.63)	6	0.56	(0.21-1.23)	6	0.76	(0.28-1.65)
Brain cancer	2	1.75	(0.21-6.34)	2	1.67	(0.20-6.02)	0	0.00	(0.00-5.95)
All lymphopoietic cancer	2	0.65	(0.08-2.35)	2	0.62	(0.07-2.23)	3	1.59	(0.33-4.64)
Leukemia	1	0.85	(0.02-4.72)	2	1.63	(0.20-5.85)	1	1.39	(0.04-7.74)
Respiratory disease	1	0.20	(0.01-1.12)	4	0.50	(0.14-1.28)	6	1.03	(0.38-2.25)

*Number of observed deaths among Pantex Plant employees during 1951-1978.
**SMR is the ratio of observed deaths to expected deaths.
***Exact Fischer 95% CI (Rothman 1979).

occurrence is expected because SMR increments often result from sampling increasingly older segments of the cohort with the attendant lessening of the healthy worker effect with increasing age (Goldsmith 1975). SMRs for the remaining specific causes of death do fluctuate slightly over the three time periods, most likely as a consequence of the small numbers in these categories.

Table VI shows observed versus expected mortality for all workers known to have received 1 rem cumulative exposure before December 31, 1978. While 252 workers were exposed to 1 rem through 1980 (see Table I), only 209 workers received 1 rem during the study period (1951 through 1978). The small number of workers in this category limits the scope of any ensuing analysis. However, enough employee data exist to show that overall mortality was significantly less frequent than expected (SMR = 0.39, 95% CI 0.13-0.79%) among these workers. This finding was based on 6 observed deaths, whereas 16.5 were expected. Deaths due to specific causes were divided among lung cancer (2), arteriosclerotic heart disease (2), cerebrovascular disease (1), and external causes (1). Mortality for all of these categories was consistent with the expected values.

TABLE VI

COMPARISON OF OBSERVED AND EXPECTED DEATHS FOR ALL PANTEX PLANT
WORKERS KNOWN TO HAVE 1 REM CUMULATIVE RADIATION EXPOSURE THROUGH 1978

Cause	OBS*	EXP**	SMR***	95% CI[+]
All causes	6	16.50	0.36	0.13-0.79
All cancers	2	3.11	0.64	0.08-2.32
Lung cancer	2	1.05	1.90	0.23-6.88
Arteriosclerotic heart disease	2	5.17	0.39	0.05-1.40
Cerebrovascular disease	1	0.65	1.55	0.04-8.57
External causes	1	3.41	0.29	0.01-1.63

*Number of observed deaths among Pantex Plant employees during 1951-1978.
**Number of expected deaths based on race, sex, and age-specific US death rates 1951-1978.
***SMR is the ratio of observed deaths to expected deaths.
+Exact Fischer 95% CI (Rothman 1979).

VI. DISCUSSION

Our analysis of mortality among Pantex Plant workers provides no evidence that employment has affected the frequency or cause-specific distribution of deaths. In general, observed mortality was less than that expected for most causes of death and was significantly less than that expected for all causes, all cancers, and circulatory diseases. We did not find a significant excess for any cause of death. Analyses by duration of employment, by time since first employment, and among those with 1 rem or more cumulative radiation exposure did not identify any significant associations with cause-specific mortality. Cancers previously found to occur in excess among other nuclear worker cohorts were less frequent than expected at the Pantex Plant.

These results should be considered in light of the limitations of this study. Perhaps the major limitation is that the majority of cohort members are still relatively young to have died from putatively occupation-related chronic diseases. This circumstance would especially affect our results for cancers, which are strongly associated with advancing age. Also, the brief average worker follow-up time (15.1 years) and low percentage of deceased workers (7.5%) necessitate results and conclusions based on only a small fraction of the eventual follow-up and mortality experience of this cohort. Our analyses of workers with longest duration of employment (15+ years) and time since first employment (20+ years) partially address these problems. Mortality for both of these subgroups was either significantly less than or consistent with the expected values. However, only continued follow-up will fully resolve these issues.

A second limitation concerns our use of US death rates as a standard for evaluating mortality among Pantex Plant workers. The biases inherent in using national death rates as a standard for worker mortality have been discussed in detail by Enterline (1975A). Briefly, for causes of death characterized by regional variations, comparisons based on national rates can produce SMRs that markedly over- or underestimate worker mortality. Regional, state, or local death rates would be a preferable basis for comparison, but only US rates were available to us for this study. However, the bias produced by using national rates for this study should be minimal because of the close correspondence between US and Texas state and county death rates over the study period (Riggan).

A related problem in studies of worker mortality is evaluating results in light of the healthy worker effect--the general tendency of industrial workers to have favorable life expectancy compared with the general population. The healthy worker effect in any cohort probably results from selection of healthy persons into the work force and individual fitness required to remain employed in the industry (Fox 1976). SMRs indicative of the healthy worker effect tend to increase toward the null value (i.e.,

1.00); three highly correlated variables increase: age, time since first employment, and length of follow-up. The healthy worker effect is strongest for heart and respiratory diseases because persons with early forms of these diseases are often symptomatic and will be less likely to be hired (selection factor) or remain employed (fitness factor) in an industrial position (McMichael 1975, Enterline 1975A). Selection factors should not operate as strongly for diseases like cancer, which have long asymptomatic latent periods. However, this point has been disputed and conflicting evidence has been presented (Enterline 1975A, Enterline 1975B, Fox 1976).

Our results are consistent with the existence of a large healthy worker effect, as overall worker mortality was only 72% of the expected value. Deficits in observed deaths from arteriosclerotic heart disease (75%), external causes (77%), cerebrovascular disease (75%), and particularly from cancer (60%) account for this finding. The cancer finding may be due to the youth of the cohort, lifestyle factors, or the absence of carcinogenic workplace exposures. In instances of a large healthy worker effect, Waxweiler (1981) has proposed evaluating cancer mortality relative to an all-cause SMR of 1.00, called a relative standardized mortality ratio (RSMR) analysis. This analysis for the study cohort, detailed in Table VII, shows no significant RSMRs for specific or total cancers. Accordingly, our cancer mortality findings (Table III) appear valid even if we underestimated the effect on worker mortality caused by the selection of healthy workers into the Pantex Plant work force.

TABLE VII

RELATIVE STANDARDIZED MORTALITY ANALYSIS FOR
PANTEX PLANT WHITE MALE EMPLOYEES 1951-1978

Cause	OBS*	AEXP**	RSMR***	95% CI+
All causes	269	269.00	1.00	0.88-1.13
All cancers	44	52.55	0.82	0.61-1.12
Large intestine	4	4.33	0.92	0.25-2.37
Lung cancer	12	17.57	0.68	0.35-1.19
Brain cancer	4	2.12	1.89	0.51-4.83
Leukemia	4	2.25	1.78	0.48-4.55
All lymphopoietic cancer	7	5.92	1.18	0.47-2.44

*Number of observed deaths among Pantex Plant employees during 1951-1978.
**Adjusted expected deaths (AEXP) is the number of expected deaths
   multiplied by all causes SMR (0.72) from Table III.
***RSMR is the ratio of observed deaths to adjusted expected deaths.
+Exact Fischer 95% CI (Rothman 1979).

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Finally, this study of Pantex Plant workers adds to the health effects literature on workers with low-level radiation exposure. The Pantex Plant cohort is noteworthy because radiation exposures are relatively low compared with other nuclear facilities (Elder 1982). The results of this investigation provide further evidence that mortality from major causes of death is not measurably increased by low-level occupational radiation exposure. Further, we could not corroborate the findings from other nuclear facilities of excess mortality resulting from pancreatic cancer, multiple myeloma, brain cancer, or benign/unspecified intracranial neoplasms. The results of this study provide no evidence that workers experience any increased risk of dying as a result of employment at the Pantex Plant.

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