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Supplementary Documentation for an Environmental Impact Statement Regarding the Pantex Plant

Radiological Consequences of Immediate Inhalation of Plutonium Dispersed by Postulated Accidents

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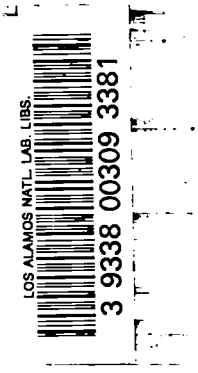

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SUPPLEMENTARY DOCUMENTATION FOR AN ENVIRONMENTAL IMPACT STATEMENT REGARDING THE PANTEX PLANT:

RADIOLOGICAL CONSEQUENCES OF IMMEDIATE INHALATION OF PLUTONIUM DISPERSED BY POSTULATED ACCIDENTS

by

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ABSTRACT

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. It describes methods used to estimate potential health consequences offsite resulting from inhalation of plutonium dispersed by each of several postulated accidents. The primary topic of this report is the delayed health effects of the plutonium in a nonnuclear detonation debris cloud inhaled directly by members of the population in the cloud path. The expected form and size of the plutonium particles are derived from experimental data obtained in the Roller Coaster test series of 1963. Retention characteristics based on the International Commission on Radiation Protection (ICRP) Task Group Lung Model and organ dose calculations based on a modified computer model are described. Health risk estimates based on organ dose are made using appropriate risk factors recommended by international radiation protection organizations. The relative seriousness of each accident at each alternative site is assessed on the basis of the health risk estimates.

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. That 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 concise EISs with less 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.

The EIS addressed both normal operations and accident situations that might have a lasting adverse effect on the environment and the nearby population. This report addresses the offsite consequences of each of several postulated accidents at three alternative sites (Pantex Plant, Iowa Army Ammunition Plant, and Hanford Reservation) in terms of radiation-induced health effects in a population exposed to a debris cloud moving downwind from the accident site. The primary hazard to be described is the inhalation of plutonium particles and subsequent irradiation of the lungs and other organs to which plutonium is biologically transferred. Other aspects of the accident analysis are included in the EIS and in support documents similar to this report. The reader will be directed to the appropriate support document in these accident-related areas: probability and amount of radioactive material release, dispersion of the released material, decontamination methods and costs, and long-term radiological risk of radioactive material not removed during decontamination activities.

Estimates of accident probabilities, locations, causes, and methods for estimating the amounts of radioactive material released (primarily weapons-grade plutonium) are described in detail by Chamberlin (1982). The letter designations, explosive amounts, cloud heights, and plutonium amounts released in the 20 postulated accidents are listed in Table I. In each case, a non-nuclear detonation of a nuclear weapon caused by one of three credible initiating events is assumed to detonate a specified number of other weapons in the vicinity. The initiating events shown to be credible (at some locations) were an aircraft crash, a tornado, and an operational accident (Chamberlin 1982).

Each postulated accident was assumed to produce a debris cloud of a height related to an equivalent high-explosive amount involved. The stabilized cloud, no longer growing by thermal buoyancy, was then dispersed in the direction of the wind by Gaussian puff calculations in the DIFOUT model, as described in detail by Dewart (1982). This model accounts for cloud depletion both from lateral diffusion and from fallout of particles. Its usefulness comes from its ability to calculate the integrated air concentration or dosage ($\mu g \cdot s/m^3$) and the ground deposition or dosage ($\mu g/m^2$) at any desired location along the cloud path.

The health consequences of each postulated accident were analyzed under two dispersion conditions: median and unfavorable. For the median case, meteorological conditions with the highest frequency (most likely to exist at the time of an accident) were derived from meteorological records at each of the alternative sites (Dewart 1982). For the unfavorable case, a poor dispersion condition and cloud movement toward the nearest population center were assumed.

TABLE I

POSTULATED ACCIDENTS: HIGH-EXPLOSIVE DETONATED AND PLUTONIUM RELEASED

Accident Designation*	High Explosive Equivalent+ (lb)	Plutonium Released (kg)	Cloud Height (m)
Pantex			
A	500	50	359
B	1 000	100	427
C	1 000	100	427
D	300	25	316
E	183	12	280
F	183	12	280
G**	114	8	248
H	2 000	30	508
I	420	120	344
J++	1.3	0.056	67***
K	19.6	0.625	135***
Iowa Army Ammunition Plant			
L	183	12	280
M++	6	0.460	119
N	114	8	248
O	114	8	248
P	300	25	316
Q	2 000	30	508
R	420	120	344
S	19.6	0.625	135***
Hanford			
T	19.6	0.625	135***

*Accident descriptions corresponding to these accident designations are provided by Chamberlin (1982).

**Dispersion and deposition values were not calculated directly for accidents involving this facility. The amounts of high explosive and plutonium (or the impact of these accidents) will not be greater than those from Accident E or F (Pantex) or L (IAAP).

***The cloud height has been calculated based on one-half of the high explosive involved due to the release of the cloud through two separate points (Chamberlin 1982).

+The effective amounts of high explosives detonated, representing the amount of energy that escapes from the facility and causes the initial cloud rise (Chamberlin 1982).

++Dispersion and deposition values were not calculated directly for this accident. The amounts of high explosive and plutonium (or the impact) of this accident will not be greater than those from Accident S.

A brief description of terminology related to radiation dose is considered appropriate in the introduction of this support document. Doses of ionizing radiation are measured in units expressing energy absorbed per unit mass of tissue (the rad) or in units of damage or injury equivalent to damage from 1 rad of gamma or x rays. This latter unit is the rem (roentgen equivalent man). The rad, defined as 100 ergs per gram of tissue, is a basic unit of absorbed dose. A rad from one type of radiation cannot be directly compared to or added to a dose from other types of radiation present at the same time unless a conversion to rem is performed. This conversion is accomplished by multiplying the dose in rad by one or more modifying factors. The chief modifying factor is the quality factor (QF), an experimentally or statistically derived value assigned to a radiation type according to the amount of damage it causes in relation to an equal dose (in rads) of gamma or x ray. The quality factors used in calculations for this document follow.

x rays, gamma rays, and beta particles	QF = 1
neutrons	QF = 10
alpha particles	QF = 20

These factors correspond to the most recent recommendations of the International Commission on Radiological Protection (ICRP 1977). The term "dose" as used in this report will refer to dose equivalent in rem unless otherwise specified.

II. RADIOLOGICAL INHALATION HAZARDS

A. Radionuclides Dispersed

1. Weapons-Grade Plutonium. Weapons-grade plutonium is a mixture of several radionuclides, with the major ingredient being ^{239}Pu. Alpha-particle emission is the primary mode of decay for plutonium. A beta-particle emitter, ^{241}Pu, contributes a large fraction (0.84) of the total activity of weapons-grade plutonium but is not as biologically significant as the alpha emitters (Poston 1977). Other alpha emitters in the mixture, particularly ^{240}Pu, are present in sufficient quantities to increase the organ dose approximately 35% above that of ^{239}Pu alone. Americium (^{241}Am), an alpha-particle and x-ray emitter, is a decay product from the beta decay of ^{241}Pu. It gradually gains in significance as a contributor to dose; ^{241}Am inhaled about 10 years after the purification process contributes only 2% of the ^{239}Pu + ^{240}Pu bone dose after 50-years' dose accumulation. The activities of alpha and beta emitters in weapons-grade plutonium are listed in Table II. The dose factors (rem/μg inhaled) resulting from the total activity are presented later.

Plutonium-239 emits energetic alpha particles (5.15 MeV). Plutonium also emits x or gamma rays mostly of low energy (<40 keV). However, ^{241}Am and ^{237}U

TABLE II

ACTIVITY OF RADIONUCLIDES IN WEAPONS-GRADE PLUTONIUM

Total activity of alpha emitters	0.081 μCi/μg
Total activity of beta emitters	0.45 μCi/μg
Total activity in the mixture	0.531 μCi/μg

(decay products of ^{241}Pu) emit 60-keV x and gamma rays, respectively. Compared to the alpha particles emitted with greater energy and abundance, the gamma rays are of little biological significance. Neutrons are also emitted from weapons-grade plutonium, either by spontaneous fission or by an alpha-neutron reaction in PuO_2. However, these neutrons are small in number and of limited biological significance compared to the alpha radiation. The quality factor of alpha particles in the energy range for plutonium (4.8 to 5.5 MeV) is approximately 20 times that of gamma radiation and twice that of neutrons (ICRP 1977).

2. Other Radionuclides and Beryllium. Other materials, radioactive and inert, would be dispersed along with the weapons-grade plutonium by the detonation accident. Some nuclear explosives may also contain other potentially toxic materials: uranium, tritium, and beryllium (a highly toxic nonradioactive material). The accidental detonation would also be expected to produce small amounts of fission products (notably iodine, strontium, and cesium) from a fission yield not exceeding 2.5×10^{17} fissions per device. Organ doses acquired by inhalation of these fission products are negligible fractions (<0.001) of the plutonium dose. As described in Appendix A, the radiological risk from the fission product dose is also negligible compared to the plutonium risk.

Enriched uranium components dispersed either as part of a plutonium device or as the primary material of a uranium device would produce relatively minor doses. Uranium, like plutonium, is a bone seeker. Besides its direct dose to lung, uranium also presents a dose to kidneys. Dose factors (rem/μg) for inhalation of equivalent amounts of enriched uranium were calculated for each

important organ (50-year dose accumulation) and found to be less than 10^{-5} of the plutonium bone dose factor, 10^{-3} of the plutonium lung dose factor, and 10^{-5} of the plutonium kidney dose factor. The ratio of total amounts of uranium to plutonium present would not exceed 3, which limits the uranium lung dose to less than 3×10^{-3} of the plutonium lung dose. Particles containing a plutonium-uranium mixture probably exist in the detonation aerosol, but this is not considered significant. Bair studied the effect on plutonium distribution in hamsters by producing plutonium-uranium oxide aerosols by the exploding wire method (Bair 1973). Retention and translocation of plutonium in the mixture after 1 year were very similar to what was observed after inhalation of PuO_2 alone.

Tritium may also be released as tritiated water by the detonation accident, although in gram quantities rather than the kilogram quantities of plutonium. Tritium emits a weak beta particle (6-keV average), which causes only limited biological damage per transformation of an atom. Tritium inhaled as tritiated water would go anywhere in the body that normal water would go, that is, into all the soft tissues. It would be eliminated as urine at the same rate as normal water. The lung dose factor for tritium is less than 2% of the weapons-grade plutonium dose factor; the whole body dose factors are approximately equal. Therefore, the large difference in source amounts allowed tritium to be discounted as a significant contributor to dose.

Because of its toxicity and suspected cancer-causing potential, beryllium was examined along with the radionuclides for potential health effects. Although no criteria for accidental exposure to beryllium exist, the Occupational Safety and Health Administration (OSHA 1978) restricts weekly occupational exposure to maximum peak of 25 $\mu g/m^3$ for 30 minutes. Calculations in which beryllium was dispersed similarly to plutonium by the DIFOUT model (Dewart 1982) showed the maximum offsite exposure from the unfavorable dispersion case to be approximately 10% of the 3.2-μg exposure received if exposed to a 25-$\mu g/m^3$ concentration for 30 minutes as allowed by OSHA. We concluded that a once-in-a-lifetime accidental beryllium exposure would be only 1/10 of an exposure that may be allowed repeatedly under current OSHA limits.

B. Plutonium Particle Retention

1. Particle Formation. Particle formation probably occurs by more than one process following initiation of a detonation. Detonation of the high explosives surrounding the plutonium components vaporizes, melts, or fragments the plutonium metal. Whether in droplet or small fragment form, the dispersed plutonium will readily react with oxygen to form PuO_2 particles. The actual processes involved in the detonation are quite complex, because other heated gases are present (H_2, N_2, and some hydrocarbons) and other materials are combined as

the aerosol cloud is formed (probably nitrates, soil, and metallic parts of the device). The particulate material from the Roller Coaster tests (Shreve 1965) showed no marked increase in solubility compared to PuO_2 (Ballance 1965). The major portion of the resulting aerosol appeared to be PuO_2 (Stewart 1969). Depending on the availability of inert particles as soil overburden, varying ratios of plutonium to inert carrier particles were observed.

2. Plutonium Retention. Retention of weapons-grade plutonium in the human body depends on the size distribution of the particles and their chemical form and crystalline structure. Sources agree that PuO_2 has low solubility in body fluids (Bair 1970, Morrow 1967, and ICRP 1980). It has been classified as a Class Y compound (pulmonary retention half-time is years, rather than weeks or days). PuO_2 formed at high temperature (>350°C) has shown lung retention half-times of 400 to 1000 days in beagle dogs (Bair 1968 and Guilmette 1980). Animal studies in conjunction with the Roller Coaster tests showed lung retention half-times of only 155 to 400 days (Wilson 1968). A clear reason for this difference was not found but may be attributable at least partially to higher surface-to-mass ratio of small particles or formation of at least some of the particles at lower temperatures in the Roller Coaster field studies. An intermediate half-time, such as the 500 days chosen by the ICRP Task Group on Lung Dynamics, appears to be a suitable choice (ICRP 1966). The Task Group Lung Model as used in dose modeling will be described later.

Particle size enters into several aspects of plutonium retention: first, the aerodynamic equivalent diameter (D_{ae}) of a particle influences how far it travels along the respiratory tract before being deposited; second, its physical diameter determines how much surface area is available for dissolution in a given mass of material; and third, its physical diameter affects the phagocytic process that attempts to clear the lung of foreign material. Without going into the detail required to describe each process, the experimental animal data in Fig. 1 are included to show the large changes (factors of 2 to 10) in various retention or transfer processes that accompany a change of even a few micrometers in particle diameter. The important feature to note in Fig. 1 is the relative consistency of the PuO_2 percentage retained in the lung as a function of particle size. Analysis of Roller Coaster data, particularly the Double Tracks test data for detonations without soil cover and Clean Slate 2 for bunkered or igloo detonations, provided valuable information directly applicable to the postulated accidents. As described in a related document (Dewart 1982), the recommended particle size to be used in the ICRP Task Group Lung Model was 2.0-μm activity median aerodynamic diameter (AMAD). For PuO_2 with a particle density of approximately 10, this converts to a mass median diameter (for comparison with Fig. 1) of 0.7 μm.

Clearance of PuO_2 from the human body occurs in early and late phases. A major portion (60 to 70%) of inhaled PuO_2 is cleared from the nasopharynx and tracheobronchial portions of the respiratory tract within the first few days

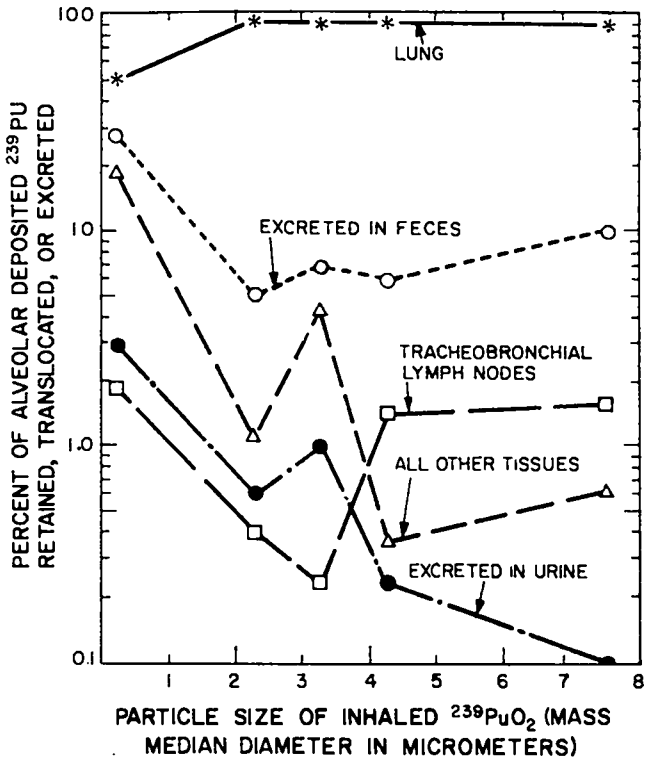

Fig. 1. Effect of particle size on retention, transfer, and excretion of inhaled $^{239}PuO_2$ in beagle dogs 30 days after exposure (Bair 1970).

after inhalation. These particles are deposited in the ciliated epithelium, trapped in mucus, and transported by ciliary action upward to the throat for ultimate elimination by the gastrointestinal tract (Morrow 1967). Particles deposited beyond the ciliated epithelium are cleared from the body very slowly. A small amount of dissolution occurs, plus some translocation of whole particles by phagocytosis to pulmonary and bronchial lymph nodes and possibly to ciliated portions of the respiratory tract. Phagocytosis is a scavenging action performed by macrophage cells, which engulf foreign particles to isolate them in place or move them to the lymph system or to ciliated airways. Plutonium reaching the bloodstream is transported to the bone and liver (approximately 45% each) and the remainder to other body tissues and excreta. Once in the bone and liver, plutonium remains for long periods, retention half-times of 100 and 40 years, respectively (ICRP 1972).

Long-term retention and transfer of $^{239}PuO_2$ deposited in the deep lung (alveolar sacs) of beagle dogs are shown in Fig. 2 (Bair 1970). For dogs, the lung clearance half-time is about 1000 days (2.7 years). Translocation to the tracheobronchial lymph nodes begins soon after exposure, with more gradual transfer to the liver and bone. Approximately 10 years after exposure, all but 10% of the plutonium deposited in the deep lung has been transferred to other

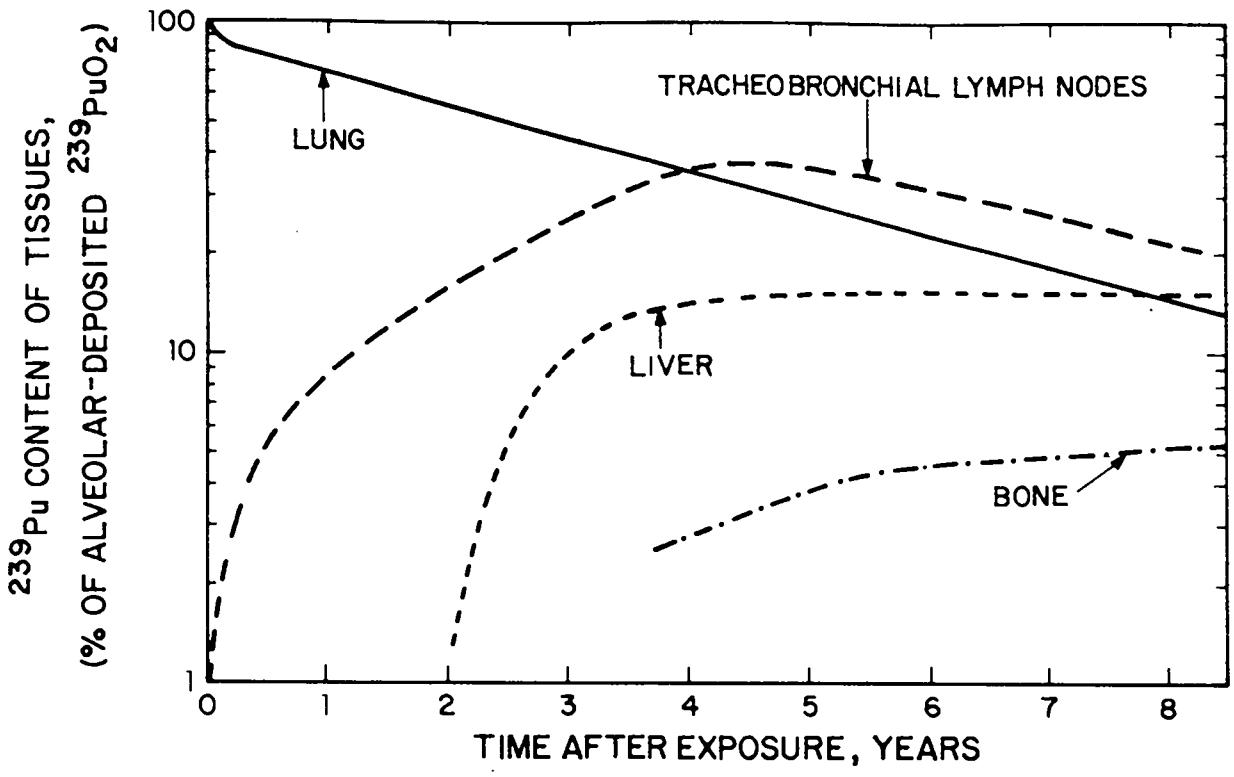

Fig. 2. Retention and transfer of alveolar-deposited $^{239}PuO_2$ in dogs (Bair 1970).

organs either as particles or monomer or polymer molecules. The liver retains 15%; the skeleton, 5%; the lymph system, 40%. As discussed later, the appearance of higher amounts (and doses) in a given organ does not necessarily mean more health effects in that organ. Sensitivity of the organ to radiation enters into estimation of effects. The relative sensitivity of each organ is discussed in Sec. IV.

3. Lung Model. The ICRP Task Group on Lung Dynamics proposed a lung model for computing particle deposition in and clearance from the human respiratory tract (ICRP 1966). The model was based on laboratory animal experiments and a few human cases. Since its initial introduction, the model has been revised by the ICRP twice, first in ICRP 19 (1972) and recently in ICRP 30 (1979). The model includes an anatomical description of the respiratory tract in three major regions (nasopharynx, tracheobronchial, and pulmonary). Its transfer fractions among regions and a graphical description of pathways are shown in Fig. 3.

The Fig. 3 diagram shows the removal pathways from the three major lung regions to the bloodstream (the secondary pathway to bone, liver, and other organs) or to the gastrointestinal tract, the primary pathway for elimination from the body. In the accompanying table, the removal fractions are listed for

Region	Pathway	Class (D)		(W)		(Y)	
N-P	(a)	0.01d	0.5	0.01d	0.1	0.01d	0.01
	(b)	0.01d	0.5	0.40d	0.9	0.40d	0.99
T-B	(c)	0.01d	0.95	0.01d	0.5	0.01d	0.01
	(d)	0.20d	0.05	0.20d	0.5	0.20d	0.99
P	(e)	0.50d	0.8	50.d	0.15	500.d	0.05
	(f)	-	-	1.d	0.4	1.d	0.4
	(g)	-	-	50.d	0.4	500.d	0.4
	(h)	0.50d	0.2	50.d	0.05	500.d	0.15
L	(i)	0.50d	1.0	50.d	1.0	1 000.d	0.9
	(j)	-	-	-	-	∞	0.1

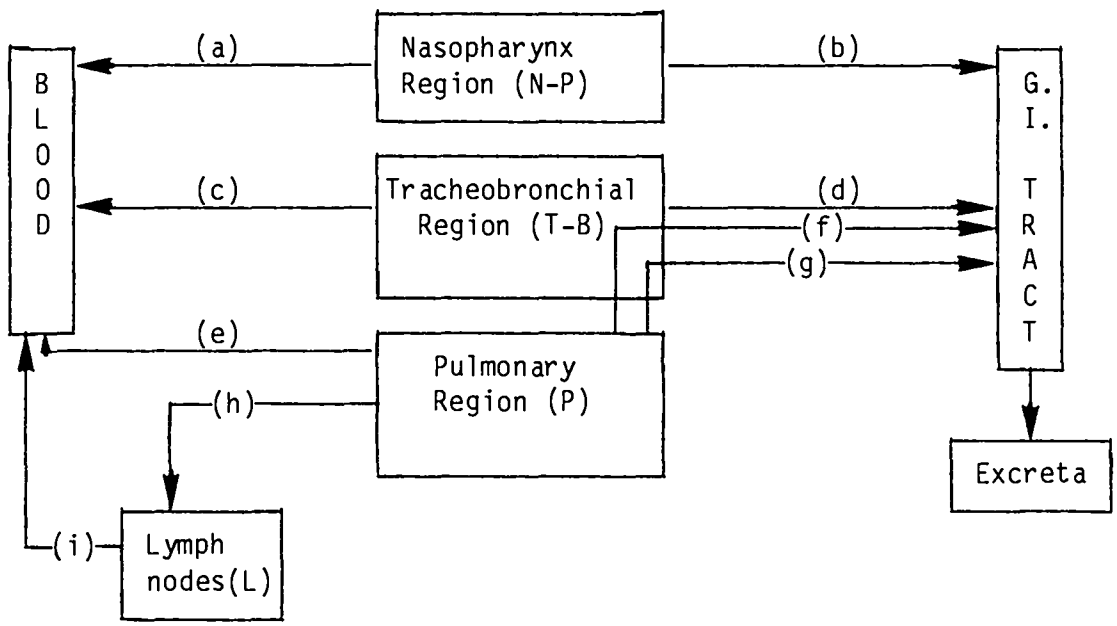

Fig. 3. Amended constants for use with ICRP Task Group Lung Model (ICRP 1979). The first value listed is the removal half-time in days (d); the second is the fraction of removed material assigned to each pathway.

each pathway depending on the clearance class of the compound. The PuO_2 clearance class is Y, which is assigned a 500-day removal half-time. As described earlier, particle size plays an important part in removal of radionuclides from the body. The Task Group Lung Model allows calculation of specific regional fractions for any particle size. This size is the activity median aerodynamic diameter (AMAD), given in micrometers. Table III shows deposition fractions for several particle sizes including the 2-μm AMAD selected to represent the particle size produced by the accidental detonation. The pulmonary fraction, the fraction of greatest biological concern, increases as particle size decreases. It decreases as breathing rate increases, primarily because of increased deposition by impaction of particles in the nasopharynx region. It should be noted, however, that the pulmonary fraction is a fraction of the total inhaled material and does not account for higher total material inhaled at higher breathing rates.

C. Plutonium Pathology

1. Somatic Effects. Somatic effects (direct effects in the exposed person as opposed to indirect or genetic effects in future generations) have been discussed in detail in ICRP (1980). No cancers attributable to plutonium have been recorded in man (ICRP 1977), although in some cases the body burdens have been as high as 0.42 μCi (Hempelmann 1973), about the same as the regulatory value for maximum permissible body burden of 0.4 μCi (ICRP 1959). Recent nationwide epidemiological studies of occupationally exposed plutonium workers indicate no detectably high death rate in the categories of all causes of death or malignancies (Voelz 1982). However, cancer induced by other internally deposited alpha emitters has been observed in other studies; therefore, risk of cancer in a population accidentally exposed to $^{239}PuO_2$ cannot be ruled out. The following paragraphs discuss the organs at risk to cancer.

TABLE III

TASK GROUP LUNG MODEL DEPOSITED FRACTIONS*

Region	AMAD** (μm)		
	1	2	5
Nasopharynx	0.29	0.50	0.77
Tracheobronchial	0.08	0.08	0.08
Pulmonary	0.23	0.17	0.11

*Deposited fraction of the total mass of material inhaled; some material is exhaled (the sum of the three regional fractions does not equal 1.0).
**Activity median aerodynamic diameter.

Animal studies using inhaled $^{239}PuO_2$ particles show the lungs to be a prominent site for development of cancer (Bair 1973). The thoracic lymph nodes around the lungs receive much higher dose but apparently are more radiation resistant than pulmonary lung tissue (ICRP 1980). The ICRP suggests combining the thoracic lymph nodes and the lungs into a single organ (ICRP 1977). Mortality can be expected in approximately 80% of lung cancer cases (BEIR III 1980).

We assume that approximately 45% of plutonium reaching the bloodstream is deposited in the bone (ICRP 1980). Mortality can be expected in almost all bone cancer cases. Bone cancer has been observed only in animal studies where soluble forms of plutonium were inhaled, causing a large bone dose much sooner than would be expected from PuO_2 (Bair 1973). The short life of laboratory animals prevents onset of bone cancer before the animal dies of old age or some other disease. However, human experience with other internal sources, such as ^{224}Ra, indicates bone sarcomas may occur if a volume bone dose of 90 rads or larger is received (Mays 1978). A dose this large is not likely to be received in an accident involving $^{239}PuO_2$, as will be noted later.

Leukemia resulting from exposure of the bone marrow to alpha radiation from $^{239}PuO_2$ deposited in bone was reviewed as a possibly serious consequence. Although leukemia, particularly myeloid leukemia, has a high sensitivity to radiation induction, it has been observed with moderate frequency in patients injected with radium-alpha emitters, at approximately one-tenth the frequency at which bone cancer occurs (Spiers 1976). The risk of leukemia from plutonium may be greater than is the risk of bone cancer because plutonium is retained in marrow as well as in bone (Morrow 1967). However, risk of leukemia from inhalation exposure to PuO_2 is poorly defined and is considered to be lower than is the risk of lung, bone, and liver cancer.

Approximately 45% of plutonium entering the bloodstream concentrates in the liver (ICRP 1980). Mortality is expected in almost all cases of liver cancer. Liver cancer has been observed with high frequency in persons injected with alpha emitters (Van Kaick 1978). Although bone sarcomas are more common than liver cancer in animals exposed to plutonium, the relative sensitivity of bone in man is lower (Mays 1976) than is the sensitivity in animals. This sensitivity indicates that a liver cancer risk probably exists in a population accidentally exposed to plutonium.

Risk of cancer in other organs or soft tissue exists, such as the kidneys and the female breast. The probability of cancer in these tissues is expected to be lower than that for other organs discussed because of lower concentration of plutonium. Relative risk of all major-organ cancers is discussed quantitatively in Sec. IV.

2. Genetic Effects. An effect of ionizing radiation is to increase the frequency of mutation, a direct effect of damage in the genetic material. Rather than including a description of the complex nature of genetic material and genetic disorders, the reader is directed to a compilation of identified disorders (McKusick 1978) and a discussion of how disorders are induced by radiation (Chadwick 1981).

Genetic disorders arising as a result of a plutonium-dispersion accident that causes increased gonad dose would not differ from those occurring naturally throughout history. However, genetic effects capable of causing serious handicap at some time during the lives of offspring could be expected to increase according to some function of the increased dose. The increased incidence probabilities are discussed in Sec. IV.

3. Acute Exposure. Animal studies have shown that PuO_2 can be inhaled in amounts large enough to cause acute toxicity and death within a week (Bair 1970). Earlier, the belief was that not enough PuO_2 could be breathed to cause this much damage. Most animal experiments showed that animals receiving >0.1 μCi/g (of lung) died within 1 year after exposure. The maximum deposit in the lung of an ICRP "reference man" (ICRP 1974), positioned at the site boundary at Pantex, was calculated to be <0.001 μCi/g (of lung). Therefore, no acute exposure mortalities would be expected from the most severe exposures resulting from the postulated accidents.

III. DOSE CALCULATIONS

A. Basic Dose Equation

Calculation of dose to a person who contacts the debris cloud from a postulated accident was based on the following equation.

$$\text{Dose} = \text{Dosage}\left(\frac{\mu g \cdot s}{m^3}\right) \times \text{organ dose factor}\left(\frac{\text{rem}}{\mu g}\right) \times \text{breathing rate}\left(\frac{m^3}{s}\right) \times \text{respirable fraction.} \qquad (1)$$

The dosage is the integrated air concentration of weapons-grade plutonium from the DIFOUT computer model described by Dewart (1982); the dose factor is the internal dose accumulated over 50 years in a specific organ per microgram of plutonium (each organ has its own dose factor); the breathing rate is a volume of air exchanged in a unit time interval; and the respirable fraction is the mass fraction of the particle cloud associated with particles less than 10-μm

D_{ae} (determined to be approximately 0.20 for plutonium aerosol in the Roller Coaster test series).

The ICRP has established a series of breathing rates consistent with analysis of doses from inhalation of radioactive material by the reference man (ICRP 1974). These standard breathing rates are as follows.

Resting	1.25×10^{-4} m^3/s
Light Activity	3.33×10^{-4} m^3/s
Heavy Work	7.1×10^{-4} m^3/s
Heavy Exercise	20.0×10^{-4} m^3/s

A moderate work level of 3.5×10^{-4} m^3/s was chosen to simulate with some conservatism the breathing rate of the person contacted by the detonation cloud. This rate implies that the accident occurs in daytime. The cloud contacts all members of the exposed population for a brief period, probably less than 30 minutes. Actual uptake time is not important because the airborne dosage ($\mu g \cdot s/m^3$) from DIFOUT provides inhaled mass when multiplied by breathing rate.

The airborne dosage was used in two ways in the accident analysis. First, the maximum dosage at each of three important locations along the cloud path was determined, that is, at the site boundary, at the nearest residence, and at the nearest population center. The dosage in Eq. (1) provided the organ dose to the maximum-exposed person at each location. Coupled with the appropriate risk factor, this dose provided an estimate of health risk to a maximum-exposed person at each of these locations. This risk was expressed in the form of odds, that is, the chances in 100 000 or the chances in 1 million that a life-threatening illness would result from the exposure.

A second way of using the dosage was to obtain an average of dosages along the inner and outer arc of a geographic sector contacted by the debris cloud. This calculation involved averaging a minimum of 10 dosage values around each sector or subsector containing people. Sector boundaries were selected to fit the city limits of the population center; this selection avoided dilution of the result by including large low-population areas with low dosage. The average dosage in the sector times the population in the sector was then applied to Eq. (1). The sum of all sectors yielded the total population dose in terms of person-rem. Dividing this population dose by the total number of people in the population yields an average dose to individuals that is useful in estimating the most likely rate of health effects in the population.

B. DACRIN Dose Model

The computer model used to calculate organ doses from inhaled radioactive material (weapons-grade plutonium) was the DACRIN model (Houston 1974), modified to include current values of quality factor for alpha radiation (20) and organ masses (5000 g bone, 1800 g liver). These modifications brought the DACRIN

results into consistency with the latest recommendations of the ICRP. DACRIN as modified adheres to the ICRP reference man (ICRP 1974), which specifies average organ masses and other physical dimensions for radiological protection purposes.

Selection of a uniform population, composed of the ICRP reference man rather than a mixed population of various ages and both sexes, was justified on the basis of several studies on the effect of population makeup. A summary of this justification is presented in Appendix B. The population dose ratio calculated for the mixed population versus the reference man population for ^{239}Pu exposure follows.

Organ	Ratio
Liver	0.94
Bone	0.94
Lung	1.15

This ratio indicates a minor underestimation of lung dose in the population dose calculation and a more conservative estimate of the bone and liver dose than when the reference man population is assumed. Although dose factors for each organ are higher for infants and children than the dose factors for teenagers and adults, this difference is partially or completely offset by the higher breathing rate of the teenagers and adults. Therefore, the assumption of a reference man population appears to be adequate in situations where the exact population distribution is unknown.

With DACRIN (as well as most of the other dose models) the following approach is used. (1) Use the ICRP lung model described in Sec. II.B.3 to determine how much of the inhaled radionuclide remains in each region of the lung and how much is transferred by the lymph system, gastrointestinal tract, and bloodstream. (2) Once a radionuclide is in the bloodstream, determine how much radionuclide is deposited in the organ of interest. (3) While in the organ, determine how much energy or dose the nuclide and its daughters deposit there. Any length of dose period can be selected; as stated earlier, we chose 50 years as a period spanning the remaining lifetime of most of the exposed population. This assumption provides an element of conservatism because total doses are not accumulated until old age when most of the population has higher risk of death from many other causes. A more realistic (shorter) accumulation time might have been chosen; it was not chosen because specific data on increased radiation-caused cancer versus age is lacking.

Input to the DACRIN code, in its simplest form, consists of a few program control variables: the duration of accumulation (50 years, as above), the organs of interest (lung, liver, bone), the quantity of radionuclide inhaled (unit mass of weapons-grade plutonium), its solubility class (Y), and its particle size (2.0-μm AMAD). Output is the dose factor in rem/μg of inhaled weapons-grade plutonium for the particular organ.

The equations on which organ doses are based are listed in detail by Houston (1974). The basic equation for a long-lived radionuclide with a brief uptake period followed by a long dose-accumulation time is

$$D(t) = \frac{5.92 \times 10^{-4} E_n}{M_n} \int_0^T Q(t)\, dt. \qquad (2)$$

$D(t)$ = the dose in rem received by the organ by time T in seconds;

E_n = effective absorbed energy per disintegration of an atom

in $\frac{\text{MeV}}{\text{dis}}$ $\frac{\text{rem}}{\text{rad}}$;

M_n = mass of the organ in grams;

$Q(t)$ = the activity (in microcuries) of the radionuclide present in the organ as a function of time; and the constant is a combination of the conversion factors

$$3.7 \times 10^4 \left(\frac{\text{dis/s}}{\mu\text{Ci}} \right) 1.6 \times 10^{-6} \left(\frac{\text{ergs}}{\text{MeV}} \right) 10^{-2} \left(\frac{\text{rads}}{\text{erg/g}} \right).$$

Depending on the organ, the time integral $Q(t)$ may be quite complex in the DACRIN calculation. Because plutonium is long lived and has only minor dose from daughter products, a simplified example of the integral of $Q(t)$ becomes

$$Q_I \sum_j \frac{f_j}{\lambda_j} \left(1 - e^{-\lambda_j T}\right), \qquad (3)$$

where

Q_I = total activity inhaled (μCi),

f_j = fraction of total intake deposited in the organ or organ compartment j,

λ_j = effective removal rate (s^{-1}), and

T = time after inhalation (s).

C. Comparison of Dose Models

More recent dose models such as INREM II and ICRP 30 provide the refinement of adding dose from radioactive materials in nearby organs to the dose from the radioactive material in the organ itself (Dunning 1979, ICRP 1978). This refinement was not needed for dose from an alpha emitter like plutonium because alpha particles cannot penetrate from one organ to another. Other variations, particularly in the choice of quality factor, caused differences that prompted a few modifications to DACRIN. The modifications achieved consistency among the results from the available dose models without a radical departure from the familiar and widely accepted DACRIN model. As stated earlier, the modifications made to DACRIN were as follows.

1. QF for alpha particles was increased from 10 to 20 in accordance with ICRP recommendations (ICRP 1977).

2. Bone mass was reduced from 7000 to 5000 g.

3. Liver mass was increased from 1700 to 1800 g.

Table IV lists the doses to the major organs calculated by each of the available dose models based on the ICRP Task Group Lung Model, including the modified DACRIN, and shows the basic differences in the models. Although the approach to dose calculation is different in several cases, modifications have brought the DACRIN dose factors into reasonable agreement with both INREM II and ICRP 30.

1. Bone Dose Factor. DACRIN does not calculate bone surfaces or red marrow dose; rather, bone doses are calculated for the bone volume and adjusted by a distribution factor n = 5 if the radionuclide is an alpha-emitting bone surface seeker, such as plutonium (ICRP 1959). This factor is based on animal experiments that showed some bone-seeking radionuclides produce greater damage to bone than ^{226}Ra, an alpha-emitting bone volume seeker. ICRP 30 and INREM II use a more elaborate model that considers the active red bone marrow cells and bone surface cells (endosteum) as components of bone at highest risk from cancer. Because irradiation of bone marrow is related to leukemia and irradiation of the endosteum dominates for ^{239}Pu, solid bone cancer rather than leukemia is the dominant health concern for bone. As described later, either the volume bone dose or the surface bone dose is appropriate in estimating health effects if the applicable risk factor is used.

2. Lung Dose Factor. Reasonable agreement among the three models was expected since all three used the Task Group Lung Model (ICRP 1966). The difference noted in Table IV arises in the various interpretations of what constitutes lung tissue. ICRP 30 considers the tracheobronchial region, the pulmonary region, and the pulmonary lymph nodes as one composite organ of mass 1000 g. The 1000-g weight represents the weight of total lung tissue for

TABLE IV

COMPARISON OF ORGAN DOSE FACTORS BY AVAILABLE DOSE MODELS (rem/μCi)*

Organ	DACRIN QF = 10	MODIFIED DACRIN*** QF = 20	INREM II QF = 20	ICRP 30 QF = 20
Lung	483	970	1 183+	1 184
Liver	387	737	797	777
Bone	595	1 590**	1 824**	NC
Bone surfaces	NC	NC	4 160	3 515
Red marrow	NC	NC	303	281
Gonads	NC	NC	40	NC

*rem/μCi based on inhaled 1-μm particles of $^{239}PuO_2$ (Class Y) and a 50-yr accumulation time.

**Based on QF = 20, n = 5.

***Modifying factors other than QF (20/10) are bone mass (5 000 g/7 000 g) and liver mass (1 800 g/1 700 g).

+Dose obtained by mass averaging the separate lung dose (580 rem in a 1 000-g lung) and lymph dose (41 400 rem in 15-g lymph node tissue).

NC not calculated.

reference man plus arterial (200 g), venous (230 g), and capillary blood (100 g). The total weight of pulmonary blood is 530 g. The 15-g mass of lymph nodes is not included. INREM II also uses a lung mass of 1000 g but treats the pulmonary lymph nodes as a separate organ. DACRIN uses a lung mass of 570 g for alpha emitters and 1000 g for beta and gamma emitters. The 570-g mass does not include arterial or venous blood, which suggests that deposited alpha energy to the lung should not be averaged over the mass of blood, because dose to blood does not contribute to a serious health effect except temporary drop in lymphocyte count. However, DACRIN does not include the pulmonary lymph nodes in the lung dose calculation. This seems reasonable because the material deposited in the lymph nodes appears to contribute very little to the incidence of lung cancer and very little to lymphosarcomas when exposed to insoluble plutonium particles (ICRP 1977). It should be noted that the lower lung mass used in DACRIN is offset when the plutonium in the lymph nodes (about 50% of that in the lung) is neglected. Therefore, the lung doses calculated by these models agree reasonably well when adjusted for the differences.

3. Liver Dose Factor. All three models treat the liver dose approximately the same, except for the quality factor and minor organ mass differences. The modified DACRIN model was used with a QF of 20 and a liver mass of 1800 g.

4. Gonad Dose Factor. The number of genetic effects depends on the dose received by the reproductive organ (gonad) of either parent. This dose is called the gamete dose and is expressed in gamete-rem as opposed to person-rem in the case of organ dose to the average individual in the population. Whether a difference exists between the gamete-rem dose and the person-rem dose to gonads is not clear. Animal studies indicate that the sperm-producing region of the male mouse gonad would receive 2 to 2.5 times the dose averaged over the whole organ (Green 1975). However, another study relating animal and human data indicates the dose to the sperm-producing region of the human to be half that of the mouse (Brooks 1979). Because this factor of 0.5 essentially offsets the factor of 2 from the animal data of Green, no distinction between gamete dose and average gonad dose is made in this report.

Neither has it been found necessary to suggest a quality factor other than the ICRP recommended value of 20 because the observed range appears reasonably represented by a quality factor of 23 (Searle 1976). Because a gonad dose calculation is not included in the DACRIN code, the dose factor for gonads was calculated using the INREM II dose model (Dunning 1979).

D. Dose Factors for Important Organs

The organs considered most important in the evaluation of health effects are the lung, liver, and bone. Each organ receives dose from inhalation of plutonium oxide and exhibits risk of a health effect. The gonad dose factor is included for illustration (Sec. V) of its role in estimating genetic effects. The dose factors used in Eq. (1) were as follows.

Lung	58 rem/μg
Liver	52 rem/μg
Bone	110 rem/μg
Gonads	2.6 rem/μg

These factors are based on weapons-grade plutonium, solubility class Y, particle size 2.0-μm AMAD, and dose accumulation time 50 years.

E. Population Dose Estimates

The use of a population dose in person-rem to obtain the dose to the average-exposed person was described in Sec. III.A. Estimation of the summed dose to the exposed population required data on the number of people at various distances and directions. Population data were projected to the year 1990 for each of the alternative sites from preliminary 1980 census data (LATA 1982). Population increases between 1980 and 1990 are expected to be approximately 14% in the Amarillo area, 4% in the Burlington area, and 20% in the Hanford Reservation area. Roughly proportional increases in population dose occurring over other time intervals could be estimated from this rate of increase.

IV. HEALTH RISK ESTIMATION

Estimation of health risk for an individual or for a population exposed to radiation can take several forms. If a rate of mortality from a unit dose is known, the risk from an exposure (or a series of exposures) can be expressed as an increase in chance of mortality (chances per 100 000 for instance) each year per unit dose. This rate applies only after a latency period expires. This risk rate multiplied by a period after the latency period (the period at-risk) yields an average lifetime risk. Another approach to estimation of the lifetime risk is used if the risk rate changes with age or sex, requiring specific knowledge of the age and sex makeup of the exposed population. This "life table" calculation adjusts the risk factor for depletion of the population by causes of death other than radiation. The following sections describe the application of these approaches in determination of organ risk factors.

Specific organ risk factors based on a linear dose-effect relationship and an absolute model for projecting lifetime risk have been adopted for the estimates of health effects caused by deposition of weapons-grade plutonium in the liver, bone, and lungs. Further, a single best estimate of health effects based on experimentally derived coefficients for each organ was chosen, rather than a broad range of possible values from several proposed dose-response models. Specific reasons for these choices are discussed in following sections.

A. Dose-Response Modeling

Various models have been developed over the years for calculating somatic health effects resulting from radiation above normal background. These models were hampered by a lack of definitive data at low doses and difficulty in relating observed somatic effects, such as cancer, unequivocally to the radiation doses. The spontaneous or normal rate of cancer incidence (all types) is quite high, approximately one in six, making any increase or excess above this normal incidence very difficult to assess statistically. Only in cancers where the normal incidence is quite low, such as bone cancer, might a radiation-induced increment be readily detectable. Figure 4 shows qualitatively how cancer incidence or mortality varies with age in an unexposed population and in an exposed population. If the radiation-induced increment is small, the likelihood of detecting the change is also small.

Studies involving human populations have been troubled by insufficient numbers of subjects in the population, inadequate dosimetry, inadequate cause of death records, and nonuniformity in distribution of dose levels over the range of interest, say 100 mrem to several hundred rem. The rate at which the dose is administered and the subject's age are also variables with important effects on dose response. Knowledge of radiation effect on lung cancer incidence in smokers has been skimpy among human subjects. Consequently, much of the work on

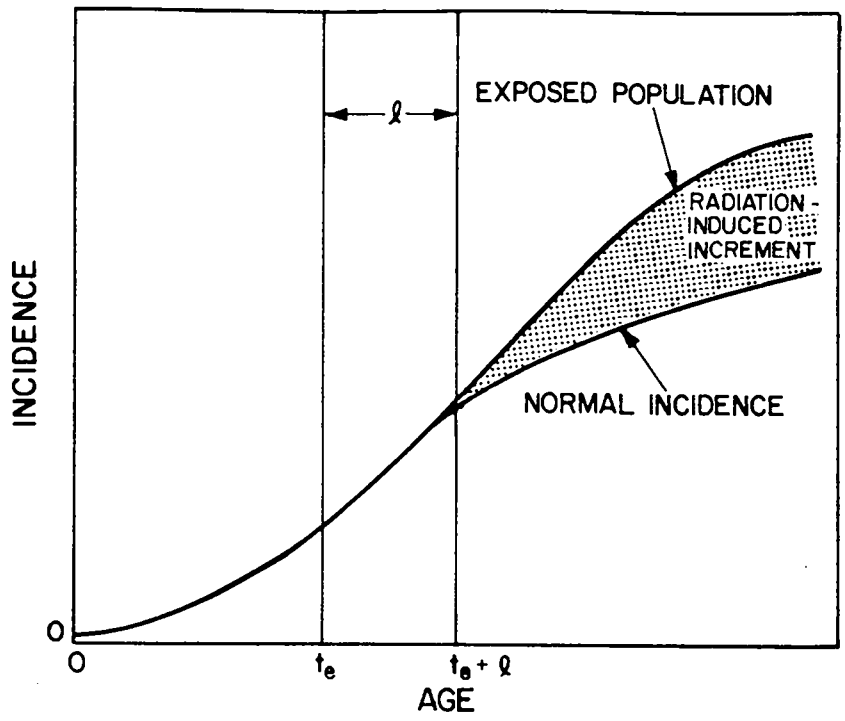

Fig. 4. Radiation-induced effect above the normal cancer incidence: t_e is age at exposure; ℓ is the latent period.

verifying dose response modeling is based on results of mammalian animal experiments, which leave the question of relevancy of animal data in estimating human dose response. However, much of the animal data has been obtained from experiments using large mammals such as beagle dogs and is believed to have relevancy in predicting potential health effects in humans.

Three major radiation protection advisory bodies have published risk factor estimates. These are

(1) the National Academy of Sciences (BEIR III 1980),

(2) the International Commission on Radiation Protection (ICRP 1977), and

(3) the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1977).

The BEIR Committee report (Committee on Biological Effects of Ionizing Radiation) was used as a primary reference in this report. However, agreement among these sources is generally good when uncertainties in each of their analyses are considered.

1. BEIR III Dose-Response Modeling. The report of the Committee on the Biological Effects of Ionizing Radiation of the National Academy of Sciences presents a detailed and comprehensive collection of dose response analyses (BEIR III 1980). Despite the controversy within the Committee itself, the differing views presented in the report, and its treatment primarily of low doses of radiation with low-linear energy transfer (LET), the BEIR III report provides information on risk factors applicable to internal alpha emitters. Although portions of the dose-effect conclusions made by the BEIR Committee and other evaluating bodies have been questioned recently due to revised Hiroshima/Nagasaki neutron dosimetry data (Loewe 1981, Straume 1981), the conclusions regarding alpha emitters are not expected to change.

In studying the proposed shapes of dose response curves, the BEIR Committee chose a linear-quadratic form to yield intermediate values between the linear and pure quadratic forms. Although not of particular concern in the postulated accident case of irradiation by internal alpha emitters (high-LET radiation), the linear-quadratic form provides a reasonable approach to dose response analysis for low-LET radiation (beta, gamma, and x ray).

2. Projection Models. Further variation in health risk estimates comes from use of two projection models designed to describe the fate of the exposed population beyond the observation time: the absolute and relative risk projection models. The absolute risk model, shown in Fig. 5 as a plateau period beginning after a latent period, can be defined as the number of radiation-induced cancer cases per unit of population, per unit of time, and per unit of radiation dose. The relative risk model, shown in Fig. 5 as the gradually increasing risk with advancing age, expresses the radiation-induced cancer risk as a multiple of the natural age-specific cancer risk for that population. The chief difference between the two models is that the relative risk model takes into account the increasing susceptibility to cancer related to advancing age. The relative risk projection yields life-time numbers of health effects substantially larger than the absolute model (approximately 3 to 5 times larger). This projection is considered an overestimation, whereas the absolute projection might be an underestimation, unless the risk rate on which the absolute projection is based was obtained over a long period of time (a major portion of a lifetime). The absolute risk projection therefore becomes the preferred model for any health effect whose risk coefficient is based on observation of exposed subjects over a long time.

B. Organ Risk Factors

BEIR III recommends a linear dose-effect relationship for high-LET radiation. This relationship allows direct application of risk rate factors in terms of cancer risk (either mortality or incidence) per person in a population (usually 10^6) per rem of radiation to the average-exposed person of that population. As previously noted, the dose to the average-exposed person

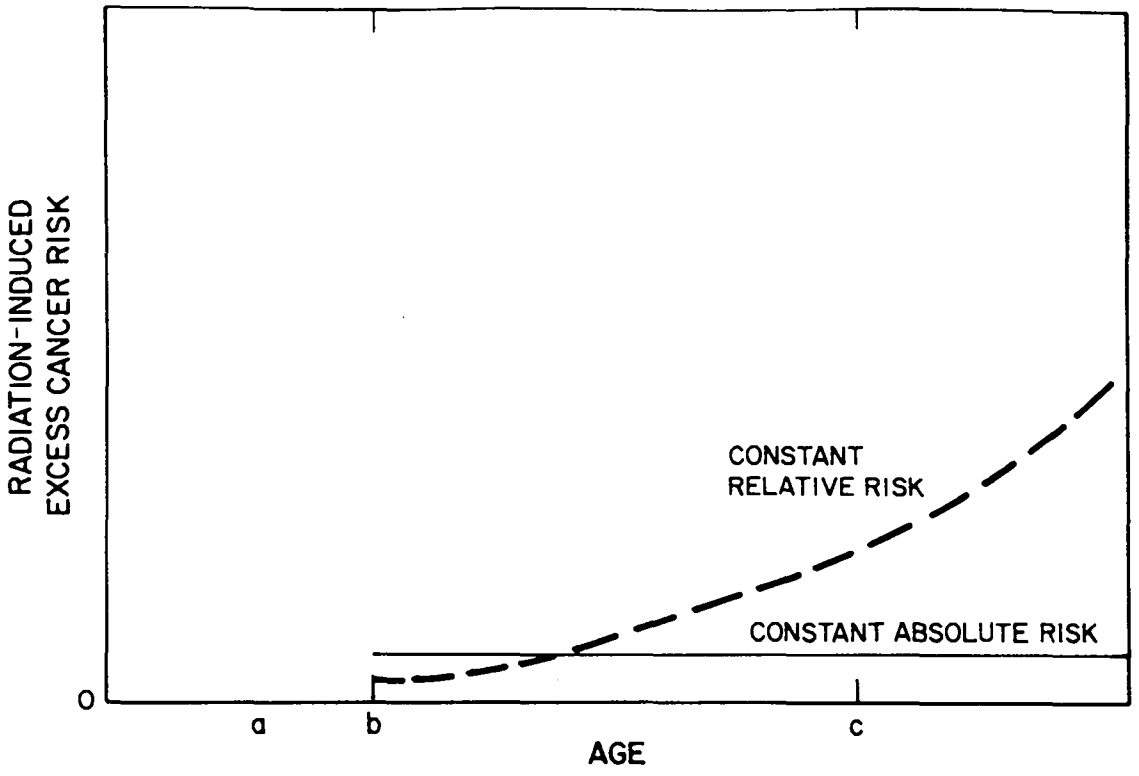

Fig. 5. Cancer risk following irradiation, absolute and relative risk models: a, age at irradiation; b, age at end of latent period; c, any age after age b (BEIR III 1980).

is obtained by dividing the integrated (population) dose by the number of persons in the population. As described in Sec. III, doses to each important organ as calculated by DACRIN in dose equivalent units (rem) can be converted to health effects by a simple multiplication.

Risk factors for high-LET radiation to each organ were obtained from BEIR III, Ch. V, Appendix A, "Site Specific Data Concerning Radiation-Induced Cancers."

We prefer to use a single best estimate value based on the experimentally derived recommendations of BEIR III, Ch. V, Appendix A, rather than a broad range of possible values such as BEIR III provided in Ch. V, Tables V-1 through V-4. This preference is based primarily on the fact that (1) the difference between the absolute and relative risk coefficients is at least a factor of 4 (relative ≈4 times absolute) and (2) neither model is specifically recommended by the BEIR Committee.

1. Liver Risk Factor. Liver cancer risk factor was based on lengthy observations of patients receiving Thorotrast. Thorotrast is colloidal $^{232}ThO_2$, an alpha emitter, used in this case as an x-ray contrast medium. The

average period at-risk of the patients at the time of observation exceeded 23 years, long enough to establish a risk factor suitable for use in estimating cancer incidence by the absolute projection model. This period at-risk (23 years) times the recommended risk rate of 13 x 10^{-6}/yr/rad or 0.65 x 10^{-6}/yr/rem yielded a lifetime risk factor of 15 x 10^{-6}/rem.

2. Lung Risk Factor. Lung risk estimates are based on data from miners exposed to radon-thoron daughters (mostly alpha emitters) at fairly high doses (>100 rem average). A major difference in administering the dose causes some uncertainty, that is, dose mostly to the bronchial epithelium by radon-thoron daughters as gas or gas molecules attached to inert particles versus dose deeper in the lung by PuO_2 particles. In the absence of human experience with lung cancer caused by plutonium and conflicting data from animal studies, we used the miner data.

Risk estimates for lung cancer depend on the age of the subject at the time of radiation exposure, as well as the age at the time of appearance of the cancer. There is little evidence of an increased risk before age 35, regardless of the age at exposure, but the risk at later ages rises steeply. The observation time was generally late in life for a large number of these miners. Continuing follow-up time greater than 17 years offers reasonable assurance that an absolute projection model can be applied to lung estimates.

BEIR III predicts risk of lung cancer mortality as follows.

Age at Diagnosis	
Under 35	0
35-49	1.5 x 10^{-6}/yr/rem
50-65	3.0 x 10^{-6}/yr/rem
over 65	7.0 x 10^{-6}/yr/rem

The latent period from radiation exposure to diagnosis is generally 10 years or more. For estimating the lung cancer risk factor, BEIR III assumed an average latency period as follows.

Exposed under age 15	25 years
Exposed between age 15-34	18 years
Exposed over age 35	10 years

A lifetime lung cancer risk factor of 43 x 10^{-6}/rem was used. To obtain this single risk factor adjusted for age, a life-table calculation was performed by the Harley and Pasternak method (Harley 1981). This method depleted the population according to the US population, providing a year-by-year count of male and female deaths in various age groups. The life-table approach produces numbers of health effects, showing that fraction of people who might die from radiation-induced cancer but actually die earlier from competing causes.

3. Bone Risk Factor. The best information on bone cancer risk comes from patients who received ^{224}Ra injections and persons exposed to intake of ^{226}Ra and ^{228}Ra. Although radium is a bone volume seeker and plutonium is a surface seeker, the assumption has been made that ^{224}Ra with its short 3.6-day half-life deposits most of its energy at the surface of bone (Spiess 1970). Therefore, the bone cancer (sarcoma) experience among ankylosing spondylitis and tuberculosis patients injected with ^{224}Ra appears to be applicable to plutonium-induced health effects. The BEIR Committee used 200 cases per million persons per rad of average skeletal (volume) dose as a basis for its bone risk factor. This factor was based on a bone mass of 7000 g. To maintain consistency with the ICRP reference man assumption of 5000 g, the risk factor was multiplied by the ratio 5000/7000, yielding a revised risk factor of 143 cases per million persons per rad. Conversion to risk per rem follows by division of the revised risk factor by the quality factor (20) and the distribution factor (5) to yield 1.4 cases per million persons per rem. Although obtained differently, this is the same per rem value recommended by the BEIR Committee. The risk factor for bone cancer was also treated by the absolute projection model due to its shorter period at risk and the lengthy observation time of the spondylitis patients.

4. Risk of Genetic Effects. Estimates of genetic effects, shown in Table V, were taken from BEIR III (1980), which showed the expected effect in the first generation and in the number of generations required to reach equilibrium

TABLE V

GENETIC EFFECTS OF AN AVERAGE POPULATION EXPOSURE OF 1 rem PER 30-yr GENERATION (BEIR III 1980)

Type of Genetic Disorder*	Current Incidence per Million Liveborn Offspring	Effect per Million Liveborn Offspring, rem per Generation	
		First Generation	Equilibrium
Autosomal dominant and X-linked	10 000	5-65	40-200
Irregularly inherited	90 000	--	20-900

*Includes disorders and traits that cause serious handicap at some time during lifetime.

(when the rate of appearance of new mutations equals the disappearance of old mutations). This equilibrium value may be used in two ways: (1) to estimate genetic effects in all offspring of an average population that receives an increased level of radiation in every generation (as with a relatively fixed increase in background radiation) or (2) to estimate total genetic effects over all time in the offspring of a specific group of people that received a single accidental exposure (applicable to the accidental exposures postulated for the Pantex EIS). In the latter case, equilibrium is not reached, and the exposure effects on the original generation tend to disappear much earlier than in the continuing exposure case. The BEIR Committee, therefore, suggests that the total of all genetic effects expressed over all future generations as a consequence of exposure limited to a single generation is numerically equal to the total for the first generation reaching the equilibrium situation. This assumption permits using the equilibrium value to estimate the over-all time effects caused by an accident.

C. Comparison with Other Recommended Risk Factors

A summary of recommended lung, liver, and bone cancer risk factors from the major radiation protection advisory bodies (ICRP, UNSCEAR, and BEIR) is presented in Table VI. Agreement for lung and liver is within a factor of 2 and makes the selection of appropriate risk factors relatively clear. For bone, the analysis by the BEIR Committee yielded 1.4 deaths per million persons per rem as opposed to 2-5 recommended by the other two bodies. The better choice for bone is not clear, but the 1.4 value appears to have a strong basis.

TABLE VI

SUMMARY OF LIFETIME MORTALITY RISK FACTORS
RECOMMENDED BY RADIATION PROTECTION ADVISORY ORGANIZATIONS
(per million persons per rem)

Organ	BEIR III	ICRP Publ. 26	UNSCEAR
Lung	43*	20	25-50
Liver	15	Under 10	10-15
Bone	1.4	5	2- 5

*Assumptions regarding latency perod and risk in intervals of age are included in Sec. IV.B.2.

D. Comparison of Postulated Accident Risks and Common Risks

A perspective on these estimates of potential risk can be gained by a simple comparison with other risks common in day-to-day living. For example, cigarette smoking (one pack or more per day) carries an equal increase in chance of death (0.15) from lung cancer as a lung dose of 3600 rem from inhalation of plutonium. As will be seen in the consequences section, this dose is approximately 14 times the highest estimate of dose to the lung of the maximum-exposed person. This example and others are shown in Table VII in terms of increase in chance of death from common risks (Wilson, 1979). An assumed risk from a lung dose of 100 rem (430 chances of death in 100 000) ranks high among these risks; however, it is lower than some risks accepted willingly by individuals in today's society.

The possible significance of health effects that could occur in the large number of people receiving doses can be evaluated by comparing the estimated

TABLE VII

RISK COMPARISON
(increase in chance of death from various activities)

Activity or Event	Chance of Death (Per 100 000)	Lung Dose** Yielding Equivalent Chance of Death (rem)
Cigarette smoking* (cancer and heart disease)	15 000	3 600
Working 10 yr as a coal miner (black lung)	1 900	460
Working 10 yr as a coal miner (accident)	640	150
Accidental lung dose of 100 rem (see text)	430	100
Dwelling in a large eastern city for 20 yr (pollution-related diseases)	360	87
Traveling 300 000 miles by automobile (accidents)	100	24
Traveling 300 000 miles by commercial jet (accident)	30	7
Traveling 300 000 miles by commercial jet (cosmic radiation)	5	1

*Moderate-to-heavy smoking (1 pack/day or more) for 40 yr.
**Risk coefficient 0.000043/person-rem.

number of potentially accident-related health effects with the normal incidence of the same cancer types in the same population. The normal incidence of cancer death in the US is approximately as follows (NCI 1975).

	Annual Number of Deaths per 100 000 Persons	Total Deaths in Average Lifetime per 100 000 Persons	
		Total	Percent
Lung cancer	42	3120	3.1
Liver cancer	2.4	256	0.26
Bone cancer	0.8	50	0.05

Consequences of the postulated accidents compared with these values are made in Sec. V. Estimates of accident-caused health effects are expressed as total numbers and as percentages of the normal incidence.

V. RADIOLOGICAL CONSEQUENCES OF POSTULATED ACCIDENTS

Methods used to estimate doses from inhalation of plutonium and the resulting health effects have been described in Secs. III and IV. We made the following assumptions.

(1) No credit was taken for mitigation measures such as evacuation or remaining indoors during debris cloud passage or for any follow-up medical procedures undertaken to minimize health effects. Although such action would reduce the chance of health effects in individual cases, it was not expected to have substantial effect on these estimates of health effects in an exposed population.

(2) The doses are caused by immediate inhalation of plutonium from the passing debris cloud. Doses from inhalation of resuspended plutonium or plutonium ingested by way of food chains or with water were treated separately and appear to be less than the immediate inhalation doses even assuming no decontamination (Wenzel 1982E).

The estimated doses and potential health effects from plutonium are believed to be overestimations and represent the upper limit of a range of conceivable consequences. These calculations include major uncertainties that may overstate consequences by factors of as much as 10 to 100.

Rather than provide an estimate of genetic effects from each accident, we decided to provide detailed estimates of cancer deaths in the exposed population and compare the likelihood of comparable numbers of genetic effects in future generations. Comparing the relative impact of genetic and somatic effects is tenuous at best when it is recognized that the populations involved are not the

same and the results of genetic effects differ broadly. The upper limit estimates of genetic effects potentially resulting from the worst of the postulated accidents are presented in Table VIII. Accident I at the Pantex Plant or Accident R at the Iowa Army Ammunition Plant (see Table I) would result in an average gonad dose of approximately 0.37 rem. This dose was combined with the risk factors of Table V to provide a range of genetic effects. The sum of effects over all time from the 0.37-rem dose ranges from 2 to 27 in the offspring of the exposed population. This number of genetic effects would not exceed approximately 0.4% of the normal incidence. As will be seen later (Sec. V.A.1), this maximum number of genetic effects is of the same order of magnitude as the number of cancer deaths potentially resulting from the worst case accident (49 lung cancers, 16 liver cancers, and 3 bone cancers).

A. Pantex

This section presents the results of the radiation-induced cancer risk evaluations for postulated accidents in the major types of facilities included in one or more of the Pantex options. For each facility, tables give results of risk estimates based on immediate inhalation of plutonium from the debris cloud

TABLE VIII

POTENTIAL NUMBER OF GENETIC EFFECTS RESULTING FROM A POSTULATED ACCIDENT*

Type of Genetic Disorder	Estimated Normal Incidence per Generation**	Accident Related Effects	
		First Generation	Over All Time
Autosomal dominant and X-linked	670	0.1-1.6	1.0-5
Irregularly inherited	6 000		0.5-22

*Based on an average gonad dose of 0.37 rem from the worst case accident at Pantex. US population (1979) figures show 15 700 live births per year per million population. From an exposed population of 142 000, this liveborn rate in a stable population would produce 67 000 liveborn offspring in each 30-yr generation. The total of effects over all time is assumed to be equivalent to the total in the first generation reaching equilibrium (BEIR III, p. 128).

**Estimated from current incidences of 10 000 per million liveborn (autosomal) and 90 000 per million liveborn (irregularly inherited), as scaled to the liveborn rate of 67 000 per generation.

resulting from a postulated accident. Information relating accident number designations and detailed accident descriptions is available in Chamberlin (1982).

1. Accident A. Potential health consequences resulting from inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table IX. Under unfavorable dispersion conditions, health effects would not be expected to exceed 18 cases of lung cancer, 6 cases of liver cancer, and 1 case of bone cancer in the exposed population (142 000). This number of lung cancers would be approximately 0.42% of the normal incidence; liver cancers, 1.6%; and bone cancers, 1.6%.

2. Accident B. Potential health consequences resulting from inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table X. Under unfavorable dispersion conditions, health effects would not be expected to exceed 28 cases of lung cancer, 9 cases of liver cancer, and 2 cases of bone cancer in the exposed population (142 000). This number of lung cancers would be approximately 0.64% of the normal incidence; liver cancers, 2.5%; and bone cancers, 2.7%.

3. Accident C. Potential health consequences resulting from inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XI. Under tornado dispersion conditions, zero or 1 case of lung cancer might occur in the exposed population (13 540).

4. Accident D. Potential health consequences resulting from inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XII. Under unfavorable dispersion conditions, health effects would not be expected to exceed 10 cases of lung cancer, 3 cases of liver cancer, and 1 case of bone cancer in the exposed population (142 000). This number of lung cancers would be approximately 0.26% of the normal incidence; liver cancers, 1.0%; and bone cancers, 1.0%.

5. Accidents E and F. These accidents have similar releases and consequences. Potential health consequences resulting from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XIII. Under unfavorable dispersion conditions, health effects would not be expected to exceed 6 cases of lung cancer and 2 cases of liver cancer; none or 1 case of bone cancer might occur in the exposed population (142 000). This number of lung cancers would be approximately 0.13% of the normal incidence; liver cancers, 0.40% of the normal incidence.

6. Accident G. The Accident G doses in Table XIV were obtained by applying a simple scaling factor (0.78) to the Accident E doses. The scaling factor accounted for the reduced cloud height and the reduced mass of dispersed plutonium. Under unfavorable dispersion conditions, health effects would not be

TABLE IX

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Pantex
Option: 1 and 4

Accident

A

Release: 50 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion Wind; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung	120	140	28	34
	Liver	39	48	26	32
	Bone	8	9	56	67
(distance, km)		(5.0)	(5.5)	(5.0)	(5.5)
Nearest Residence	Lung	120	130	28	30
	Liver	39	40	26	27
	Bone	8	8	56	59
(distance, km)		(5.2)	(6.5)	(6.5)	(6.5)
Major Population Center	Lung	8	36	1.9	8.6
	Liver	3	11	1.7	7.6
	Bone	1	2	3.9	17
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung	2	13	0.36	3.0
	Liver	<.5	4	0.32	2.7
	Bone	<.5	1	0.73	5.9
Total Population Exposed		13,540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung	<.5 (--)*	18 (0.42)*		
	Liver	<.5 (--)	6 (1.6)		
	Bone	<.5 (--)	1 (1.6)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE X

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative: Site: Pantex, Option: 1 and 4 — Accident: B

Release: 100 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion: Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung	160	160	38	38
	Liver	54	54	36	36
	Bone	11	11	78	78
(distance, km)		(5.0)	(5.5)	(5.0)	(5.5)
Nearest Residence	Lung	160	150	38	36
	Liver	54	48	36	32
	Bone	11	10	78	73
(distance, km)		(5.2)	(6.5)	(5.2)	(6.5)
Major Population Center	Lung	17	70	4.0	17
	Liver	5	22	3.6	15
	Bone	1	5	8.1	34
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung	2	20	0.48	4.8
	Liver	1	6	0.44	4.2
	Bone	<.5	1	0.98	9.5
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung	<.5 (--)*	28 (0.64)*		
	Liver	<.5 (--)	9 (2.5)		
	Bone	<.5 (--)	2 (2.7)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XI

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative	Accident
Site: Pantex Option: 1 and 4	C

Release: 100 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Tornado Storm Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion
Site Boundary (distance, km)	Lung Liver Bone	240 77 16 (5.0)	See Note	56 51 110 (5.0)	See Note
Nearest Residence (distance, km)	Lung Liver Bone	220 71 15 (5.2)	See Note	52 48 110 (5.2)	See Note
Major Population Center (name, distance)	Lung Liver Bone	10 3 1 (Borger, 42 km)	See Note	2.4 2.1 4.8 (Borger, 42 km)	See Note
"Average Individual"	Lung Liver Bone	2 1 <.5	See Note	0.40 0.36 0.78	See Note
Total Population Exposed		13 540		13 540	
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	See note		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

Note: A tornado storm system moving WSW toward Amarillo was not considered credible.

TABLE XII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative — Accident

Site: Pantex — D
Option: 1, 2, and 4

Release: 25 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual or cases/100 000 of exposed population)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion Wind; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung Liver Bone	85 27 6	85 27 5	20 18 42	20 18 39
(distance, km)		(5.0)	(5.5)	(5.0)	(5.5)
Nearest Residence	Lung Liver Bone	85 27 6	76 24 5	20 18 42	18 16 36
(distance, km)		(5.2)	(6.5)	(5.2)	(6.5)
Major Population Center	Lung Liver Bone	4 1 <.5	9 3 1	0.84 0.76 1.7	2.2 2.1 4.5
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung Liver Bone	1 <.5 <.5	7 2 1	0.22 0.19 0.42	1.7 1.5 3.4
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	10 (0.26)* 3 (1.0) 1 (1.0)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XIII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Pantex

Option: 1 and 4

Accident

E and F

Release: 12 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung Liver Bone	51 16 3	51 16 4	12 11 24	12 11 25
(distance, km)		(5.0)	(5.5)	(5.0)	(5.5)
Nearest Residence	Lung Liver Bone	51 16 3	47 15 3	12 11 24	11 10 22
(distance, km)		(5.2)	(6.5)	(5.2)	(6.5)
Major Population Center	Lung Liver Bone	2 1 <.5	11 3 1	0.38 0.34 0.76	2.6 2.3 5.0
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung Liver Bone	1 <.5 <.5	4 1 <.5	0.14 0.13 0.28	0.94 0.86 1.9
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	6 (0.13)* 2 (0.40) <.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XIV

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative: Accident G

Site: Pantex
Option: 2

Release: 8 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung Liver Bone	40 13 3	40 13 3	9.4 8.6 20	9.4 8.6 20
(distance, km)		(5.0)	(5.5)	(5.0)	(5.5)
Nearest Residence	Lung Liver Bone	40 13 3	36 12 2	9.4 8.6 20	8.6 7.8 17
(distance, km)		(5.2)	(6.5)	(5.2)	(6.5)
Major Population Center	Lung Liver Bone	1 <.5 <.5	3 1 <.5	0.30 0.27 0.59	2.0 1.8 3.9
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung Liver Bone	<.5 <.5 <.5	3 1 <.5	0.11 0.10 0.22	0.73 0.67 1.5
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	4 (0.10)* 1 (0.39) <.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

expected to exceed 4 cases of lung cancer and 1 case of liver cancer; none or 1 case of bone cancer might occur in the exposed population (142 000). This number of lung cancers would be approximately 0.10% of the normal incidence; liver cancers, 0.39%.

7. Accident H. Potential health consequences resulting from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XV. Under unfavorable dispersion conditions, health effects would not be expected to exceed 7 cases of lung cancer, 3 cases of liver cancer, and no case of bone cancer in the exposed population (142 000). This number of lung cancers would be approximately 0.16% of the normal incidence; liver cancers, 0.70%.

8. Accident I. Potential health consequences resulting from immediate inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XVI. Under unfavorable dispersion conditions, health effects would not be expected to exceed 49 cases of lung cancer, 16 cases of liver cancer, and 3 cases of bone cancer in the exposed population (142 000). This number of lung cancers would be approximately 1.1% of the normal incidence; liver cancers, 4.3%; and bone cancers, 4.7%. This number of health effects makes this accident the most serious of the postulated accidents at Pantex.

9. Accident J. No potential health consequences would be expected from inhalation of plutonium as the debris cloud passes through populated areas.

10. Accident K. Potential health consequences that result from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XVII based on the upper limit 0.6-kg release. Even under unfavorable dispersion conditions, zero or 1 case of lung cancer might occur in the exposed population (142 000). If the release were reduced by factors of 2 to 4, no health effects would be expected.

B. Iowa Army Ammunition Plant

This section presents the results of the radiation-induced cancer risk evaluations for the major types of facilities included in the two Iowa Army Ammunition Plant options. Tables give results of radiological health risk calculations based on immediate inhalation of plutonium from the debris cloud resulting from the postulated accidents.

1. Accident L. Potential health consequences resulting from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XVIII. Under unfavorable dispersion conditions, health effects would not be expected to exceed 6 cases of lung cancer and 2 cases of liver cancer; zero or 1 case of bone cancer might occur in the exposed population (34 400). This number of lung cancers would be approximately 0.61% of the normal incidence; for liver cancers, 2.3%.

TABLE XV

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Pantex
Option: 1, 2, 3, and 4

Accident

H

Release: 30 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100,000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung Liver Bone	110 34 7	82 26 5	26 23 50	19 17 39
(distance, km)		(2.2)	(4.0)	(2.2)	(4.0)
Nearest Residence	Lung Liver Bone	94 28 6	74 23 5	22 19 45	17 16 36
(distance, km)		(2.4)	(5.0)	(2.4)	(5.0)
Major Population Center	Lung Liver Bone	5 2 <.5	19 6 1	1.2 1.0 2.4	4.4 4.0 9.0
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung Liver Bone	1 <.5 <.5	5 2 <.5	0.13 0.12 0.27	1.2 1.2 2.5
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	7 (0.16)* 3 (0.70 <.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XVI

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Accident

Site: Pantex
Option: 1, 2, 3, and 4

I

Release: 120 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung	740	590	170	140
	Liver	230	190	160	120
	Bone	51	39	360	280
(distance, km)		(2.2)	(4.0)	(2.2)	(4.0)
Nearest Residence	Lung	690	460	160	110
	Liver	220	150	140	97
	Bone	47	30	340	220
(distance, km)		(2.4)	(5.0)	(2.4)	(5.0)
Major Population Center	Lung	42	94	10	22
	Liver	14	28	9.1	19
	Bone	3	6	20	45
(name, distance)		(Borger, 42 km)	Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung	3	35	0.76	8.2
	Liver	1	11	0.68	7.4
	Bone	<.5	2	1.6	17
Total Population Exposed		13 540	142 000	13 540	142,000
Potential Cases of Cancer Deaths in Exposed Population	Lung	<.5 (--)*	49 (1.1)*		
	Liver	<.5 (--)	16 (4.3)		
	Bone	<.5 (--)	3 (4.7)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XVII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Pantex
Option: 1, 2, and 3

Accident

K

Release: 0.6 kg

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW	Median Dispersion; Wind to NNE	Unfavorable Dispersion; Wind to WSW
Site Boundary	Lung Liver Bone	8 2 1	19 6 1	1.8 1.6 3.6	4.4 4.0 9.0
(distance, km)		(5.0)	(5.4)	(5.0)	(5.4)
Nearest Residence	Lung Liver Bone	7 2 <.5	17 5 1	1.6 1.4 3.4	4.0 3.6 8.1
(distance, km)		(5.2)	(5.6)	(5.2)	(5.6)
Major Population Center	Lung Liver Bone	<.5 <.5 <.5	1 <.5 <.5	0.024 0.022 0.047	0.20 0.19 0.42
(name, distance)		(Borger, 42 km)	(Amarillo, 25 km)	(Borger, 42 km)	(Amarillo, 25 km)
"Average Individual"	Lung Liver Bone	<.5 <.5 <.5	<.5 <.5 <.5	0.0092 0.0082 0.018	0.042 0.038 0.084
Total Population Exposed		13 540	142 000	13 540	142 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	<.5 (---)* <.5 (---) <.5 (---)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XVIII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Iowa Army Ammunition Plant
Option: 1

Accident

L

Release: 12 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung	67	160	16	38
	Liver	21	51	14	34
	Bone	4	11	31	78
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Nearest Residence	Lung	67	160	16	38
	Liver	21	51	14	34
	Bone	4	11	31	78
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Major Population Center	Lung	---	85	---	20
	Liver	---	27	---	18
	Bone	---	6	---	42
(name, distance)		(None)	(Burlington, 8.6 km)	(None)	(Burlington, 8.6 km)
"Average Individual"	Lung	8	19	1.9	4.4
	Liver	3	6	1.7	3.8
	Bone	1	1	3.9	8.7
Total Population Exposed		3 340	34 400	3 340	34 400
Potential Cases of Cancer Deaths in Exposed Population	Lung	<.5 (--)*	6 (0.61)*		
	Liver	<.5 (--)	2 (2.3)		
	Bone	<.5 (--)	<.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

2. Accident M. Potential health consequences resulting from immediate inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XIX. These values were obtained from the Accident S case, which has similar cloud height and amount released. Under unfavorable dispersion conditions, zero or 1 case of cancer might occur in the exposed population (34 000).

3. Accidents N and O. These accidents have similar releases and consequences. Potential health consequences resulting from immediate inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XX. Under unfavorable dispersion conditions, health effects would not be expected to exceed 5 cases of lung cancer and 2 cases of liver cancer; zero or 1 case of bone cancer might occur in the exposed population (34 400). This number of lung cancers would be approximately 0.61% of the normal incidence; for liver cancers, 2.3%.

4. Accident P. Potential health consequences resulting from immediate inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XXI. If median dispersion conditions existed at the time of the accident, health effects would not be expected to exceed 1 case of lung cancer in the exposed population (3 340). Under unfavorable dispersion conditions, health effects would not be expected to exceed 13 cases of lung cancer, 4 cases of liver cancer, and 1 case of bone cancer in the exposed population (34 400). This number of lung cancers would be approximately 1.2% of the normal incidence; for liver cancers, 4.3%; and for bone cancers, 4.8%.

5. Accident Q. Potential health consequences resulting from immediate inhalation of plutonium as the detonation cloud passes through these populated areas are listed in Table XXII. Under unfavorable dispersion conditions, health effects would not be expected to exceed 8 cases of lung cancer, 3 cases of liver cancer, and 1 case of bone cancer in the exposed population (34 400). This number of lung cancers would be approximately 0.80% of the normal incidence; for liver cancers, 3.0%; and for bone cancers, 3.4%.

6. Accident R. Potential health consequences resulting from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XXIII. If median dispersion conditions existed at the time of the accident, health effects would not be expected to exceed 2 cases of lung cancer, 1 case of liver cancer, and zero or 1 case of bone cancer in the exposed population (3 340). Under unfavorable dispersion conditions, health effects would not be expected to exceed 50 cases of lung cancer, 16 cases of liver cancer, and 3 cases of bone cancer in the exposed population (34 400). This number of lung cancers would be approximately 4.5% of the normal incidence; for liver cancers, 18%; and for bone cancers, 20%. This number of health effects makes this accident the most serious of the postulated accidents at Iowa Army Ammunition Plant. Health effect estimates from the same accident at Pantex were very

TABLE XIX

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Iowa Army Ammunition Plant
Option: 2

Accident

M

Release: 0.46 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	100 31 7	26 8 2	24 21 50	6.2 5.5 12
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Nearest Residence	Lung Liver Bone	100 31 7	26 8 2	24 21 50	6.2 5.5 12
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Major Population Center	Lung Liver Bone	--- --- ---	8 3 1	--- --- ---	1.9 1.7 3.9
(name, distance)		(None)	(Burlington, 8.6 km)	(None)	(Burlington, 8.6 km)
"Average Individual"	Lung Liver Bone	1 <.5 <.5	2 1 <.5	0.20 0.19 0.42	0.38 0.34 0.76
Total Population Exposed		3 340	34 400	3 340	34 400
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	<.5 (--)* <.5 (--) <.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XX

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Iowa Army Ammunition Plant
Option: 1

Accident

N

Release: 8 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	53 16 3	130 40 9	12 11 24	30 27 62
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Nearest Residence	Lung Liver Bone	53 16 3	130 40 9	12 11 24	30 27 62
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Major Population Center	Lung Liver Bone	--- --- ---	66 21 5	--- --- ---	16 14 34
(name, distance)		(None)	(Burlington, 8.6 km)	(None)	(Burlington, 8.6 km)
"Average Individual"	Lung Liver Bone	6 2 <.5	15 4 1	1.5 1.4 3.1	3.4 3.0 6.8
Total Population Exposed		3 340	34 400	3 340	34 400
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	5 (0.61)* 2 (2.3) <.5(--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XXI

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Iowa Army Ammunition Plant
Option: 1

Accident

P

Release: 25 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	170 54 11	280 88 18	40 36 81	66 59 130
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Nearest Residence	Lung Liver Bone	170 54 11	280 88 18	40 36 81	66 59 130
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Major Population Center	Lung Liver Bone	--- --- ---	170 54 11	--- --- ---	40 36 81
(name, distance)		(None)	(Burlington, 8.6 km)	(None)	(Burlington, 8.6 km)
"Average Individual"	Lung Liver Bone	15 5 1	37 11 2	3.6 3.2 7.6	8.6 7.6 17
Total Population Exposed		3 340	34 400	3 340	34 400
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	1 (--)* <.5 <.5	13 (1.2)* 4 (4.3) 1 (4.8)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XXII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative — Accident

Site: Iowa Army Ammunition Plant — Q

Option: 1 and 2

Release: 30 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	130 40 9	270 86 18	30 27 62	64 57 130
(distance, km)		(2.45)	(1.8)	(2.45)	(1.8)
Nearest Residence	Lung Liver Bone	130 40 9	270 86 18	30 27 62	64 57 130
(distance, km)		(2.45)	(1.8)	(2.45)	(1.8)
Major Population Center	Lung Liver Bone	--- --- ---	100 31 7	--- --- ---	24 21 50
(name, distance)		None	(Burlington, 6.6 km)	None	(Burlington, 6.6 km)
"Average Individual"	Lung Liver Bone	10 3 1	25 8 2	2.4 2.1 4.8	5.8 5.1 12
Total Population Exposed		3 340	34 360	3 340	34 360
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	8 (0.80)* 3 (3.0) 1 (3.4)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XXIII

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative: Site: Iowa Army Ammunition Plant; Option: 1 and 2

Accident: R

Release: 120 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	1100 340 74	2000 660 130	260 230 530	480 440 950
(distance, km)		(2.45)	(1.8)	(2.45)	(1.8)
Nearest Residence	Lung Liver Bone	1100 340 74	2000 660 130	260 230 530	480 440 950
(distance, km)		(2.5)	(1.8)	(2.5)	(1.8)
Major Population Center	Lung Liver Bone	--- --- ---	850 270 59	--- --- ---	200 180 420
(name, distance)		(none)	(Burlington, 6.6 km)	None	(Burlington, 6.6 km)
"Average Individual"	Lung Liver Bone	68 22 5	140 46 10	16 14 34	34 30 70
Total Population Exposed		3 340	34 360	3 340	34 360
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	2 (2.2)* 1 (8.2) <.5 (--)	50 (4.5)* 16 (18) 3 (20)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

similar to these values. At Pantex, the population exposed would be larger, but the plutonium amounts received at the population center would be lower.

7. Accident S. Potential health consequences that result from immediate inhalation of plutonium as the detonation cloud passes through populated areas are listed in Table XXIV based on the upper limit 0.6-kg release. Under unfavorable dispersion conditions, zero or 1 case of lung cancer, liver cancer, or bone cancer might occur in the exposed population (34 400). If the release were lower because of improved engineering design, no health effects would be expected.

C. Hanford Reservation

Accident T. Potential health consequences resulting from inhalation of plutonium as the debris cloud passes through populated areas are listed in Table XXV. Even under unfavorable dispersion conditions, only zero or 1 case of lung cancer, liver cancer, or bone cancer might occur in the exposed population (119 000). If the releases were lower because of improved engineering design, no health effects would be expected.

VI. SUMMARY AND CONCLUSIONS

Potential health consequences resulting from inhalation of plutonium from postulated nonnuclear detonation accidents at the Pantex Plant and two alternative sites have been estimated. Numbers of somatic effects (cancer) in the exposed population and of genetic effects in offspring of this population are expected to be roughly comparable. Cancer of the lungs, liver, or bone are the most important of the potential health consequences in the exposed population. These consequences were calculated in terms of risk of death from cancer induced by radiation dose to the average-exposed person in the exposed population and to the maximum-exposed person along the cloud path. At the Pantex Plant, the most serious accident occurring under unfavorable dispersion conditions would cause an estimated lung cancer risk of 49 cases in the exposed population of 142 000; liver cancer, 16 cases; and bone cancer, 3 cases. This accident at an alternative site (Iowa Army Ammunition Plant) would cause approximately the same number of cancer deaths in a smaller exposed population (34 400), which would be located closer to the site. Health consequences of an accident at the third alternative site (Hanford Reservation) would be minimal, since credible tornado and aircraft initiating events do not exist there and all facilities would greatly mitigate the only credible detonation involving plutonium.

TABLE XXIV

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Iowa Army Ammunition Plant
Option: 2

Accident

S

Release: 0.6 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E	Median Dispersion; Wind to NE	Unfavorable Dispersion; Wind to E
Site Boundary	Lung Liver Bone	100 31 7	26 8 2	24 21 50	6.2 5.5 12
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Nearest Residence	Lung Liver Bone	100 31 7	26 8 2	24 21 50	6.2 5.5 12
(distance, km)		(1.5)	(3.9)	(1.5)	(3.9)
Major Population Center	Lung Liver Bone	--- --- ---	8 3 1	--- --- ---	1.9 1.7 3.9
(name, distance)		(None)	(Burlington, 6.8 km)	(None)	(Burlington, 6.8 km)
"Average Individual"	Lung Liver Bone	1 <.5 <.5	2 1 <.5	0.20 0.19 0.42	0.38 0.34 0.76
Total Population Exposed		3 340	34 400	3 340	34 400
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	<.5 (---)* <.5 (----) <.5 (----)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

TABLE XXV

SUMMARY OF HEALTH RISKS TO INDIVIDUALS
FROM IMMEDIATE INHALATION OF RADIOACTIVITY DURING CLOUD PASSAGE

Alternative

Site: Hanford Reservation
Option: 1

Accident

T

Release: 0.6 kilograms

Location of Individual	Organ	Incremental Risk of Health Effects (chances/100 000 for an individual)		Radiological Dose Used in Estimate (50-year commitment, rem)	
		Median Dispersion; Wind to SSE	Unfavorable Dispersion; Wind to SSE	Median Dispersion; Wind to SSE	Unfavorable Dispersion; Wind to SSE
Site Boundary (distance, km)	Lung Liver Bone	<.5 <.5 <.5 (35)	1 <.5 <.5 (35)	0.068 0.061 0.14 (35)	0.26 0.23 0.50 (35)
Nearest Residence (distance, km)	Lung Liver Bone	<.5 <.5 <.5 (35)	1 <.5 <.5 (35)	0.068 0.061 0.14 (35)	0.26 0.23 0.50 (35)
Major Population Center (name, distance)	Lung Liver Bone	<.5 <.5 <.5 (Richland, 42 km)	1 <.5 <.5 (Richland, 42 km)	0.040 0.036 0.081 (Richland, 42 km)	0.14 0.13 0.28 (Richland, 42 km)
"Average Individual"	Lung Liver Bone	<.5 <.5 <.5	<.5 <.5 <.5	0.016 0.013 0.031	0.046 0.040 0.092
Total Population Exposed		119 000	119 000	119 000	119 000
Potential Cases of Cancer Deaths in Exposed Population	Lung Liver Bone	<.5 (--)* <.5 (--) <.5 (--)	<.5 (--)* <.5 (--) <.5 (--)		

*Value in ( ) is number of cases as a percentage of normally expected mortality from the same types of cancer in the given population.

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APPENDIX A

A COMPARISON OF INHALATION DOSES FROM FISSION PRODUCTS AND PLUTONIUM FOLLOWING A NONNUCLEAR DETONATION OF SEVERAL NUCLEAR DEVICES

A nonnuclear detonation of a nuclear device may be accompanied by a very small nuclear yield. It has been stated that the internal dose generated from the uptake of the resulting fission products is insignificant compared to that from the plutonium. The validity of this assertion was evaluated using the DACRIN computer code. The assumptions made and the procedure used are as follows.

(1) A nuclear yield equivalent to 40 lb of TNT distributed among several units was assumed (approximately 2.5×10^{18} fissions).

(2) The amount of airborne respirable plutonium was 8.0 kg. All plutonium in the cloud was assumed to be ^{239}Pu.

(3) Since the fission product source term is time dependent, fission product activities were evaluated at 1 min after detonation. Data from UCRL-50243, "Fission Product Decay Chains," were used.

(4) The following biologically significant fission products were selected for study: ^{90}Sr, ^{91}Sr, ^{93}Y, ^{95}Nb, ^{99}Mo, ^{103}Ru, ^{106}Ru, ^{131}Te, ^{131}I, ^{132}Te, ^{132}I, ^{137}Cs, ^{140}Ba, ^{140}La, ^{144}Ce, ^{147}Pm, and ^{155}Pu. We believe that these radionuclides estimate the total fission product dose within a factor of 2.

(5) Internal doses to the following body organs were evaluated: total body, kidneys, liver, spleen, bone, lungs, testes, ovaries, brain, and thyroid.

(6) To determine the lung solubility class (days, weeks, years), we assumed that all fission products were in the oxide form.

(7) We assumed that the meteorological transport of the fission products and plutonium was identical.

The results for whole body, kidney, liver, bone, and lung are summarized in Table A-I as dose ratios (fission product dose/^{239}Pu dose) for 1-, 5-, 10-, and 50-yr dose commitments. None of the dose ratios listed are larger than 8.8×10^{-4}. Because the risk of health effects in those organs (cancers/rem/million persons) does not differ among them more than one order of magnitude, the health effects from fission products would not exceed 10^{-3} of plutonium effects.

TABLE A-I

DOSE RATIOS: FISSION PRODUCT/^{239}Pu

Organ	1 yr	5 yr	10 yr	50 yr
Total body	6.8×10^{-4}	5.1×10^{-5}	1.7×10^{-5}	2.4×10^{-6}
Kidneys	8.8×10^{-4}	6.5×10^{-5}	2.2×10^{-5}	3.3×10^{-6}
Liver	5.6×10^{-5}	4.2×10^{-6}	1.4×10^{-6}	2.5×10^{-7}
Bone	1.2×10^{-4}	9.4×10^{-6}	3.2×10^{-6}	4.9×10^{-7}
Lung	2.6×10^{-5}	1.1×10^{-5}	1.0×10^{-5}	1.0×10^{-5}

For the spleen, testes, and thyroid, the committed dose is due solely to fission products; the significance of these organ doses was evaluated independently. The largest of these organ doses (by a factor of about 100) was the thyroid dose due to uptake of radioiodines and their precursors. The plutonium lung and bone dose commitments were greater than the thyroid dose by a factor of 10^3. Because the risk factors for lung and thyroid are approximately the same (BEIR III 1980, p. 198), it appears justifiable to ignore the thyroid contribution following the nonnuclear detonation of nuclear devices.

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APPENDIX B

EFFECT OF AGE DISTRIBUTION ON POPULATION RADIATION DOSE ESTIMATES

I. BACKGROUND

When calculating a population dose for an organ "j" ($D_{pop,j}$), it is generally assumed that the population is homogeneous and composed entirely of adults with the characteristics of reference man as defined by ICRP 23 (1974). Of course, real populations are heterogeneous and possess a definite age distribution. Four age classes are defined: infant (<1 year); child (1-10 years inclusive); teen (11-16 years inclusive): and adult (17 years and older). All members of the adult age class are assumed to be reference man. Members of any given population can be subdivided into these age classes. The question is what effect does such an age distribution have on the population dose estimate. In mathematical terms the question becomes how much different than 1 is the ratio

$$\frac{D_{pop,j}\ (\text{Heterogeneous})}{D_{pop,j}\ (\text{Homogeneous})} \quad .$$

This appendix shows how this ratio was evaluated for the inhalation of insoluble weapons-grade plutonium causing dose to the bone, lungs, and liver.

II. POPULATION AGE DISTRIBUTION

The test population is assumed to be distributed in the same manner as the US population. The Bureau of the Census (1977) gives three main population projection series that differ essentially in the assumed average number of lifetime births per woman: Series I, 2.7; Series II, 2.1; and Series III, 1.7. The Series I projection is used here because it yields the largest percentages in the younger age classes. Table B-I summarizes the age distribution for the test population using the Series I projection for July 1, 1982.

III. AGE-SPECIFIC RADIATION DOSE FACTORS AND BREATHING RATES

The general methodology and assumptions of Hoenes and Soldat (Hoenes 1977) were followed, except that the DACRIN computer code was used to calculate doses and an acute rather than a chronic intake was assumed.

TABLE B-I

SERIES I, PROJECTION FOR US POPULATION
July 1, 1982

Age Class	Number	Fraction of Total
Infant	4 501 000	0.0196
Child	34 969 000	0.1522
Teenager	21 460 000	0.0934
Adult	168 770 000	0.7348
Totals	229 700 000	1.0000

Radiation dose factors in rem/μg were calculated for each of the age classes defined in Sec. I. With the exception of organ masses and radii, all other biological and metabolic parameters were assumed to be the same in all age groups. It was noted, however, that biological half-lives for adults tend to be greater than those for younger individuals. Therefore, dose factors calculated without using age-specific biological half-lives will generally overestimate the radiation dose for the young age groups.

The age-specific organ masses used in the calculations are listed in Table B-II. The age-specific dose factors for weapons-grade plutonium are summarized in Table B-III. The age-specific breathing rates were taken from Regulatory Guide 1.109 (NRC 1977) and are listed in Table B-IV.

TABLE B-II

AGE-SPECIFIC ORGAN MASSES

Organ	Infant	Child	Teenager	Adult
Lung	110 g	300 g	580 g	1 000 g
Bone	550 g*	1 171 g*	3 500 g*	5 000 g
Liver	212 g**	561 g**	1 271 g**	1 800 g

*NUREG-0172 value $\times \frac{5\ 000\ g}{7\ 000\ g}$.

**NUREG-0172 value $\times \frac{1\ 800\ g}{1\ 700\ g}$.

TABLE B-III

AGE-SPECIFIC DOSE FACTORS FOR INHALATION OF WEAPONS-GRADE PLUTONIUM AEROSOL WITH AMAD = 2 μm (rem/μg)

Age Class	Lung	Bone	Liver
Adult	58	110	52
Teenager	97	120	54
Child	190	170	75
Infant	320	180	80

TABLE B-IV

AGE-SPECIFIC BREATHING RATE

Age Class	m^3/yr	m^3/s
Adult	8 000*	2.5×10^{-4}*
Teenager	8 000	2.5×10^{-4}
Child	3 700	1.17×10^{-4}
Infant	1 400	4.44×10^{-5}

*This adult breathing rate is less than the reference man value for an 8-h workday (3.5×10^{-4} m^3/s) because the breathing rate is averaged over a 24-h period, which includes sleep as well as activity.

IV. METHODOLOGY

For a homogeneous population, the population dose in man-rem for organ j may be summed over all sectors as follows:

$$D_{pop,\ j} = Q(BR)(DF)_j \sum(\overline{X}/Q)_s P_s ,$$

where

Q = source term,
BR = breathing rate of reference man,
$(DF)_j$ = reference man (adult) dose factor for organ j,
$(X/Q)_s$ = the average dispersion factor for a given geographical sector, and
P_s = the population in a given geographical sector.

Similarly, for a heterogeneous population,

$$D_{pop,j} = Q \sum_s \left(\overline{X}/Q)_s P_s \; F_A BR_A DF_{j,A} + F_T BR_T DF_{j,T} + F_C BR_C DF_{j,C} + F_I BR_I DF_{j,I} \right),$$

where Q, $(\overline{X}/Q)_s$, and P_s are as defined above and

F_A = fraction of the population in a given sector, which is in the adult age class,
F_T = fraction of the population in a given sector, which is in the teenager age class,
F_C = fraction of the population in a given sector, which is in the child age class,
F_I = fraction of the population in a given sector, which is in the infant age class,
BR_A = the adult (reference man) breathing rate,
BR_T = the teenager breathing rate,
BR_C = the child breathing rate,
BR_I = the infant breathing rate, and
$DF_{j,A}$, $DF_{j,T}$, $DF_{j,C}$, $DF_{j,I}$ = the age-dependent dose factors for organ j.

Assuming that each F is the same for all sectors, the population dose becomes

$$D_{pop,j} = Q \sum_{i=1}^{4} F_i \left(BR_i\right)\left(DF_{j,i}\right) \sum_s (X/Q)_s P_s \; .$$

The ratio of the two populations is

$$D_{pop,j}\ (\text{Heterogeneous})/D_{pop,j}\ (\text{Homogeneous}) = \frac{\sum_{i=1}^{4} F_i \left(BR_i\right)\left(DF_{j,i}\right)}{BR_A DF_{j,A}} \; .$$

TABLE B-V

POPULATION DOSE RATIO
(HETEROGENEOUS/HOMOGENEOUS)
FOR WEAPONS-GRADE PLUTONIUM INHALATION

Critical Organ	Ratio
Bone	0.94
Lung	1.15
Liver	0.94

V. RESULTS

Population dose ratios for bone, lungs, and liver are shown in Table B-V.

VI. DISCUSSION AND CONCLUSION

It appears that lung dose would not be underestimated more than 15% by the homogeneous population dose calculations for weapons-grade plutonium inhalation. For bone and liver, the homogeneous estimate is the more conservative, overestimating by 5%. The significance of incorporating age-dependent data into population dose estimates is studied by Etnier and Till (1979) for a hypothetical fuel reprocessing facility. Age-dependent data for consumption of vegetables, beef, and milk, as well as inhalation rates were used. For the spectrum of radionuclides considered, the study concludes that "incorporation of currently available age-dependent data has little effect on population dose and does not appear to be worthwhile. Additional research in the future on metabolic behavior of specific radionuclides as a function of age may create a renewed need to include age dependency in dose assessments for populations." Therefore, at the present state of the art, the homogeneous population assumption is adequate in situations where the exact population age distribution is not known.

REFERENCES

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