AAPM REPORT NO. 18

A PRIMER ON LOW-LEVEL IONIZING RADIATION AND ITS BIOLOGICAL EFFECTS

Published for the
American Association of Physicists in Medicine
by the American Institute of Physics

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A Report of the Biological Effects Committee
American Association of Physicists in Medicine

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July 1986

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American Association of Physicists in Medicine
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American Institute of Physics
c/o AIDC
64 Depot Road
Colchester, VT 05446

(1-800-445-6638)

Library of Congress Catalog Card Number: 86-072715
International Standard Book Number: 0-88318-514-8
International Standard Serial Number: 0271-7344

Published by the American Institute of Physics, Inc.,
335 East 45 Street, New York, New York 10017

Printed in the United States of America
third printing, 1992

Introduction

During the past few years widespread concern regarding the health effects of low levels of' ionizing radiation has developed. Lou radiation levels include patient exposures in medical diagnostic procedures using x-rays or radioactive materials, occupational exposures from radiation sources used in medicine and industry, end public environmental exposures due to the presence of x-ray sources or contamination with radioactive substances. The information in this primer is intended to provide an up-to-date review of the scientific background and current estimates of the potential risk of adverse effects of such exposure. The effects of high radiation levels such as those occurring in a serious accident with large radiation sources or in nuclear warfare may include rapid appearance of radiation damage and early death, but these are not discussed here because the early damaging effects cannot be produced by low-level exposures.

This primer is principally directed to the professional staffs of medical institutions, particularly medical physicists, radiologists and nuclear medicine specialists, but also to other physicians who refer patients for examinations involving low levels of radiation exposure, or who may be exposed to radiation in a clinical or research environment. It is hoped that the information contained herein will also be educational to diagnostic x-ray, radiation therapy, and nuclear medicine technologists. and to nurses, particularly that in Section II which lists a large number of questions commonly asked by patients and medical personnel who are exposed to radiation, together with answers based on current assessments of radiation risks.

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ACKNOWLEDGEMENTS

The preparation of this primer was a project of the Biological Effects Committee of the American Association of Physicists in Medicine (E. W. Webster, Chairman). Significant critical contributions to the text and particularly to the questions in Section 2, were made by the following members of the Committee: R. O. Carson, C. C. Ling, A. Norman. C. G. Orton and A. J. Schneider. Other members of the Committee serving for varying periods during preparation of the text were: A. B. Ashare, R. J. Baker, A. B. Brill, C. C. Chamberlain, E. C. Gregg, D. E. Herbert, E. C. Krishnan, L. H. Lanzl, J. Ovadia, A. J. Schulz, R. J. Shalek. and R. L. Witkofski.

SECTION 1

THE BASIC FACTS

IONIZING RADIATION

1. Definitions

Ionizing radiation, as distinct from non-ionizing radiation, has sufficient energy to ionize atoms or molecules in biological and other systems. An atom is ionized when one or more of its electrons is separated from the atom. The electrons which are released and the remaining positively-charged ions are chemically reactive and the reactions they produce can damage the chemical constituents of living matter. Other kinds of radiation such as radio waves, visible light, and ultrasound do not have the capability of producing ionization, although they may cause biological damage by other mechanisms if the energy deposited is sufficiently great.

2. Types

The principal kinds of ionizing radiation encountered in our society are:

a. gamma rays and x-rays (essentially identical)
b. beta particles (high speed electrons)
c. neutrons (heavy uncharged particles. mass nearly equal to hydrogen atom)
d. alpha particles (heavy charged particles, mass about four hydrogen atoms)

These four kinds of radiation differ in methods of production, details of their interactions with atoms, and their penetration ability through matter.

Gamma rays and x-rays, like radio waves, both consist of tiny packets of energy known as photons, which travel with the speed of light. They have Identical properties and are distinguished only by the mechanism which produce them. Photons are not usually regarded as particles since they have no mass. In contrast. beta particles, neutrons and alpha particles all possess mass and are basic components of the atoms of which matter is composed.

The energy carried by the photons and particles in a stream of radiation is measured in units of kilo-electron-volts (keV) or mega-electron-volts (MeV), where one electron-volt (eV) is the energy gained by a particle having one electronic charge moving between two surfaces (in a vacuum) with a potential difference of one volt: one keV and one HeV are 1000 and 1,000,000 times greater, respectively. One keV and one MeV are much greater than the amount of energy necessary to cause ionization of an atom, which is about 10 eV. Therefore photons or particles with these high energies will produce a large number of ionizations when they are absorbed in matter.

Radiation is emitted by all radioactive substances. The disintegration of radioactive atoms is the source of gamma rays, some x-rays, beta particles and alpha particles. An important characteristic of all radioactive materials is a gradual reduction with time In the emission of radiations. The time required for the emissions to be reduced to one-half of their Initial level Is known as the half-life. After 10 half-lives the initial radiation level Is reduced by a factor of about 1,000. The half-life for different radioactive materials may be shorter than 1 second or as long as billions of years. Those used in diagnostic medicine have half-lives ranging from a few hours to a few weeks so that the radiation emission falls to negligible values In times ranging from a few days to a year.

X-rays are produced by the absorption of high-speed charged particles, usually electrons. The most common source of x-rays is an x-ray tube in which a beam of electrons is directed toward, and is absorbed in, a metal target. The x-ray tubes most commonly used in medicine or industry do not produce radioactivity. The radiation from ordinary x-ray tubes is therefore terminated when the x-ray tube is switched off. Only very high energy x-rays (many MeV) generated by special equipment can produce radioactivity and that usually has a short half-life.

Neutrons are released when x-rays, gamma rays or high-speed charged particles with very high energies interact with atomic nuclei, and when the nuclei of certain elements such as uranium and thorium are caused to split, a process known as nuclear fission.

3. Linear energy transfer.

The four kinds of radiation listed above can be classified according to the average amount of energy transferred to an absorber per unit distance along the path of the radiation, a quantity known as the "linear energy transfer" (LET). LET is commonly expressed in units of keV per micron (0.001 mm). Table 1 provides typical values of LET. High values of LET Signify that ionizations are produced much closer together in an absorber than is the case with low values of LET. LET is an important radiation characteristic since it affects the amount of damage produced by the absorption of a given amount of radiation energy.

X-rays and gamma rays are absorbed by collisions with electrons to which some or all of their energy is transferred. These electrons lose their energy in ionizing events which are relatively widely spaced. X-rays, gamma rays and electrons (including beta particles) are, therefore classified as low LET radiations. Electrons and beta particles will penetrate distances in the body ranging from a small fraction of a millimeter up to a few centimeters depending

on their energy. In contrast, x-ray and gamma ray photons will penetrate considerably greater distances than electrons possessing the same energy. Typical photons employed in x-ray diagnosis will travel several centimeters into the body before an electron collision occurs. Some will penetrate the entire thickness of the human body without a collision and can therefore produce an anatomical image on a suitable recording device. The Fraction of the photons which reach a given depth in the body or penetrate the entire body, increases as the photon energy increases.

TABLE 1

LET for 1 MeV Radiations

Radiation Type	LET in water (keV per micron)
Low LET:	
x-ray, gamma ray or beta particle	approx. 0.25
High LET:	
Proton or Fast neutron	approx. 40
Alpha particle	approx. 200

Alpha particles compared with beta rays, deposit their energy in much shorter distances -- their energy loss per unit distance is about 1000 times greater as shown in Table 1 and they are said to have a high LET. Alpha particles with a Few MeV of energy are absorbed in a Few centimeters of air or in a Few contiguous cells. They will not penetrate the skin of the body and only constitute a hazard when the radioactive material emitting them is inside the body.

Fast neutrons are also classified as high LET radiation because their interactions with matter produce heavy charged particles, such as protons, which have a high LET. However, because the neutrons themselves carry no electrical charge, they can travel relatively large distances between interactions and can, therefore, penetrate many centimeters of the body (as do x and gammma rays.)

4. Radiation dose units.

The amount of energy absorbed by tissue elements From the passage of radiation is usually expressed in terms of absorbed dose, of which the unit is the rad.* Dose represents the amount of energy absorbed per gram of absorber -- it signifies, therefore, the concentration of the absorbed energy. The rad is an energy absorption of 100 ergs per gram. The amount of biological damage increases as the dose (rads) increases, but it is not necessarily proportional to the dose. The type of radiation also affects the amount of damage produced by a given dose because of differences in LET. It has been found that high LET radiations (alpha particles and neutrons) generally produce more damage per rad of dose than do low LET radiations (x-rays, gamma and beta rays). This is because they deposit more energy in the small sensitive volumes of a cell nucleus when traversing a cell. Another dose unit, the rem** (rad equivalent man), has therefore been introduced to take account of the greater effects of high LET radiations. The rem is simply the rad multiplied by a Quality Factor (Q) which expresses the effectiveness of' a particular kind of ionizing radiation relative to that of x-rays. The Quality Factor depends on the LET. These

*A new international unit of radiation dose, the gray (Gy) has been introduced recently as one of the S.I. (Systeme Internationale) units. 1 gray = 100 rads.

**A new international unit of radiation dose equivalent, the sievert (Sv), has been introduced recently as one of the Systeme Internationale (S.I.) units. 1 sievert = 100 rem. In the interest of simplicity the S.I. units are not used in this primer.

factors allow all ionizing radiations to be described in equivalent dose units when discussing human radiation hazards. Conventional values for Q of 10 For Fast neutrons and 20 for alpha particles have been recommended by the National Council on Radiation Protection and Measurements (NCRP) (1).

Thus 1 rad of gamma or x-rays = 1 rem (Q = 1)
1 rad of Fast neutrons = 10 rem (Q = 10)
1 rad of alpha particles : = 20 rem (Q = 20)

In this primer we will describe doses† in rem or millirem (mrem) (one thousandth of a rem), and dose rates† in rem per minute, per hour or per year or millirem per hour or per year.

5. Examples of radiation dose levels.

A good yardstick for appreciating the size of the rem unit is the background radiation level. This is contributed by three components which are variable with location. Average values in the U.S.A. are shown in Table 2:

TABLE 2

Average U.S. Levels of Radiation Background (2)

Radiation Source	mrem/year
Cosmic rays	28*
Terrestrial radioactivity	26**
Internal radioactivity	26
Total	80

*after 10% reduction For building shielding
**after 20% reduction For building shielding and a further 20% reduction For body shielding

†more precisely, the rem end millirem are units of "dose equivalent."

The cosmic radiation component increases with altitude (it is doubled at 6500 ft compared to sea level) and at higher latitudes. At cruising altitude in a jet plane it is about 100 times greeter then et ground level (0.5 mrem per hour). In Denver, Colorado, it is 50 mrem/year. The terrestrial component is due to radioactivity in the soil (end in buildings), particularly that of uranium, thorium and potassium-40, and is much higher on the Colorado plateau (57 mrem/year) than on the Atlantic/Gulf coastal region (15 mrem/year) or the Middle West region (29 mrem/year). The internal radioactivity is produced primarily by potassium-40 supplied naturally in the diet (19 mrem/year) but also by carbon-14, and the radioactive decay products of thorium end uranium, including radium. Table 3 shows extra background doses to the whole body received in various situations.

The lung receives en additional background dose averaging about 700 mrem/year from normal concentrations outdoors of radon in the atmosphere (4). Radon is a derivative of uranium in soil end rocks. Since radon levels are higher indoors, the average dose to the lung is estimated at about 3 rem/year increasing in some areas with higher uranium concentrations (e.g., Grand Junction, Colorado and in phosphate mining areas of Florida to the range 7-14 rem/year. In some energy-efficient homes with minimal ventilation the lung dose may be as high as 28 rem/year (4).

TABLE 3

Increased Background Doses (3)

	millirem
One round-trip Flight New York to Los Angeles	5
Moving Free East Coast to Denver for one year	50
Moving from frame house to brick house, one year	30
To flight crew From cosmic rays per year	150

Another common yardstick is the dose received from medical x-ray: procedures. About one-half of medical diagnostic examinations are of the chest. In all of these procedures the exposure is to a part of the body, not to the whole body; moreover, the tissues In the x-ray beam receive different doses with the maximum on the skin at the entrance area of the beam and typically only 1% to 10% of the maximum at the exit area. Therefore these medical doses are not strictly comparable with background radiation levels which expose the whole body. Some typical maximum doses (to the skin) are given in Table 4.

TABLE 4

Some Typical Skin Doses from Diagnostic X-ray Examinations*

Chest:	anterior-posterior	30 mrem
	lateral	100 mrem
	ovaries	1 mrem
Dental film:	periapical	350 mrem
Skull:	lateral	250 mrem
Lumbar spine:	anterior-posterior	850 mrem
Abdomen:	anterior-posterior	750 mrem
	ovaries	125 mrem
Fluoroscopy:	abdomen	3000 mrem/minute
Computerized body tomography:		4000 mrem

* Mainly from Ref. 5 including backscattered radiation

Medical examinations providing information on the functioning of specific organs are also performed by administering to patients small amounts of radioactive materials. Typical doses to the whole body and to organs which receive the maximum dose are listed in Table 5. More than 90% of nuclear medicine procedures employ technetium-99m. The typical maximum organ dose with this radionuclide is 1000 to 4000 mrem while the average dose to the whole body is of the order 100 mrem.

In x-ray treatments for cancers much higher localized doses are given - typically about 6,000,000 mrem to the tumor-bearing tissue over a period of 6 weeks. These doses are intended to destroy all the malignant cells in the tumor.

The x-ray or gamma ray dose delivered in a short time to the whole body which is fatal for most people after a time lapse of about 30 days is estimated at about 350,000 mrem(7).

Figure 1 indicates the range of doses for several medical procedures and compares them with the doses for which various biologic effects have been observed.

BIOLOGICAL EFFECTS OF IONIZING RADIATION

6. Mechanisms and characteristics.

The biologic damage produced by radiation occurs first at the chemical and biochemical level due to the disruption of some of the molecules within the cells of living matter; this disruption may be produced by direct cleavage of the parts of a molecule or by chemical attack by abnormally active chemical agents produced by the radiation -- for example, in decomposing the water molecules in the cell. The most critical targets in the cell are the DNA

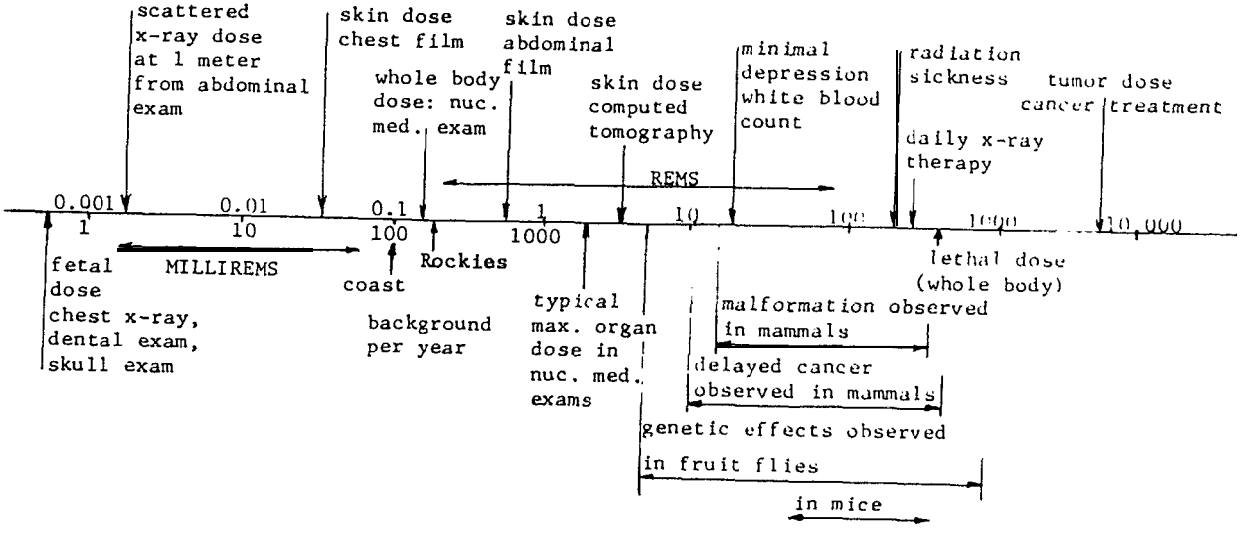

Figure 1. Scale of radiation doses and effects.

TABLE 5

Radiation doses to adults from radioactive materials used in common medical diagnostic procedures (nuclear medication)+ (6)

Organ of Interest	Radionuclide	Amount in Millicuries*	Typical Doses in Millirem Crit. Organs**	Whole Body
Tumors	Gallium 67 Citrate	5	3000 (spleen)	800
Thyroid	Iodine 123 iodide (scan)	0.2	2000 (thyroid)***	6
Thyroid	Iodine 131 iodide (uptake)	0.005	5000 (thyroid)***	2
Heart	Thallium 201 chloride	2.0	4200 (kidney)	500
Brain	Technetium **99m** -pertechnetate	**20**	4000 (intestine)	260
Brain, kidney	-DTPA	10	4500 (bladder)	160
Cardio-vascular	-HSA	20	1000 (blood)	300
Lung	-MAA	3	600 (1ungs)	45
Blood pools	-red blood cells	15	4000 (spleen)	220
Liver, spleen	-sulfur colloid	3	1000 (liver)	60
gone	-diphosphonate	20	1000 (bone)	260

† imaging tests except for measurements of thyroid uptake with I-131.
* a millicurie is a unit of radioactivity denoting the number of radioactive atoms which change by radiation emission every second.
** the critical organ is the organ with the maximum radiation done.
*** 20% uptake in thyroid gland

molecules which are responsible for cellular development, function and division. Cellular damage is manifested in several ways: death of cells, failure to reproduces, and much more rarely, transformation of cells to new forms, some of which may eventually become malignant and initiate a cancer. In the case of the germ cells, damage to the genes may occur, introducing heritable diseases which may appear in future generations. It high doses (greater than 100,000 mrem) a large fraction of cells is likely to die; at low doses (below 10.000 mrem) few cells are damaged and the harmful effects, if any, on an organism became evident only after a long period of time.

DNA damage is partially repairable. The amount of repair has been shown to depend on the type of radiation, the dose delivered and the time over which delivery occurs (i.e. dose rate). There is a large body of experimental information on rainy forms of life including bacteria, plants, animals, and isolated suspensions of animal cells, which shows that repair is minimal following irradiation with high LET radiation, but is very likely with low doses or low dose-rates of low LET radiation. This has been explained by a theory proposing that two or more repairable sub-lesions must be produced sufficiently close together in space and time to interact with each other in order to yield a permanent effect(*). There is a very high probability that high LET radiations will produce along their tracks two or more such close sub-lesions, but with low LET radiations the sub-lesions are relatively widely separated.

A consequence of this "multiple-hit" mechanism to produce a permanent effect is that the relation between dose and effect for low LET radiation would be expected to be non-linear; the dose-effect curve would be concave upward as illustrated in Figure 2. However. a linear relationship with a small slope would be expected at the lowest doses (below about 10 rem or 10,000 mrem) which would continue as shown in Figure 2 for low dose rates. The relationship for

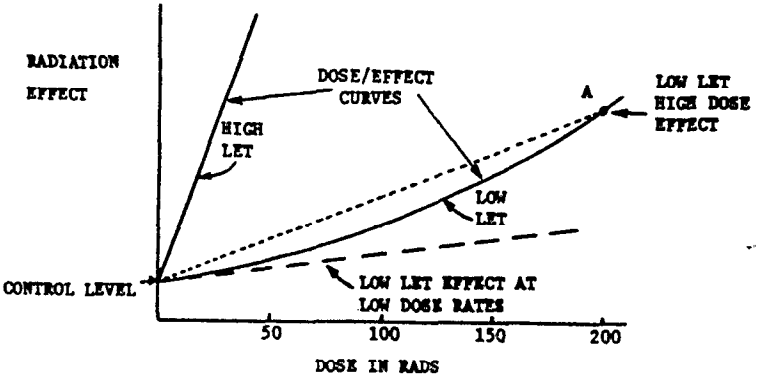

Figure 2. Relation between magnitude or frequency of radiation affects and radiation dose for high LET and low LET radiations. The control level is the normal level of effect in the absence of radiation. The affects of high LET radiations per rad are generally considerably greater (higher slope) than for low LET radiations. The dotted line illustrates that a linear extrapolation from high doses to law doses for low LET radiations may over-estimate the affect of low doses. The dashed line indicates the generally smaller effects of low LET radiation when it is delivered in a fractionated or a protracted time period: that is, when the average dose rate is reduced. This is attributed to repair processes within the cell nucleus which are most affective at low doses or at low dose rates when ionizing events are widely separated in space, or in time, or both.

high LET radiation would be linear but the slope would be much greater than for low LET radiation as shown, when the dose is expressed in rads as in Figure 2.

It is also evident in Figure 2 that if a projection to zero dose from a single high dose point (such as point A) is made for low LET radiation in order to estimate low-dose effects, such a projection (dotted line) will be an overestimate, unless the dose at point A is sufficiently low.

As indicated above, the hazards of radiation to man differ in kind and In degree with dose. At low doses (below 10 rem) there is insignificant cell death and the effects are more subtle in kind, probabilistic (stochastic1 in nature and infrequent in occurrence. The following possible effects are of concern, but at low doses remain to be conclusively demonstrated for mammalian life:

- Induction of malignancy in persons irradiated
- genetic effects in the offspring of persona irradiated
- malformation and retarded development in children following fetal irradiation

The first two may have no threshold dose, i.e., they may occur with diminishing but not zero frequency as the dose is reduced. On the other hand it seems likely that gross structural malformations in a child are produced only when a threshold dose to the fetus is exceeded (see page 32).

CANCER AND RADIATION

7. Cancer

Cancer is the second most likely cause of death after heart disease. In 1983 in the USA 21.6% of all deaths were due to cancer (about 1 in 5) compared with 38.2% due to heart diseases. Estimates of the likelihood during a lifetime

following birth in 1985 of either developing or dying from particular form of cancer are givens in Table 6 (9). In recent years, the highest risks of cancer development in men have occurred in the following organs: lung 8.7%, prostate 8.7% and intestines 6.51; and in women, breast 10.21, intestines 6.95 and uterus 5.05%. Radiation in sufficient dosage is known to cause many types of cancer but only a very small fraction (about 1%) of the total cancer cases in the population can be attributed to radiation (10).

8. Radiation-inducedcancer.

Information on the risk of cancer induction by radiation is of two types: direct observations on human populations and experimental studies of animal populations. Because of the differences in radiation effects on different animal species, the animal information cannot be directly translated into human risk but can only provide a rough guide. Therefore, it would be desirable to place major reliance on human observations. Unfortunately, those observations are sparse and controversial and the low radiation levels which are of principal interest.

Radiation-induced cancer has no special characteristics which differentiate it from cancer produced by other causes. Therefore, human studies of the risks from radiation are conducted by comparing the cancer incidence (or cancer deaths) In an exposed population with the incidence In a caparable population which received no radiation exposure (the control population). Human studies present many problems: cancer occurs in human populations many years after the radiation exposure, at least 10 years ranging up to 40 or more years later. Leukemia (proliferation of the white blood cells) is an exception in that it appears earlier than solid cancers, starting at 2 or 3 years after exposure. The size of the human populations exposed is often small and the number of cancers observed even smaller. This leads to statistical problems in ascertaining the reality of an observed increase.

TABLE 6

1985 Expectation in 10,000 persons* of developing or dying of cancer during a lifetime (9)

Site	Cancer incidence**		Cancer deaths	
	Male	Female	Male	Female
All cancer	3690	3610	2320	2000
Breast	----	1020	----	360
Uterus	----	500	----	90
Lung	870	420	780	340
Prostate	870	----	260	----
Intestine	650	690	290	310
Urinary***	450	190	130	70
Lymphomas****	200	200	100	100
Mouth	160	90	50	30
Stomach	120	80	70	60
Ovary	----	150	----	120
Pancreas	120	130	110	120
Leukemia	120	90	100	80
Esophagus	50	30	50	20
Larynx	90	20	30	10
Thyroid	20	50	0	10

*white persons
**exclusive of skin cancer
***kidney, bladder
****including multiple myeloma

The statistical problem can be demonstrated by the following example. Suppose that the incidence of radiation-induced breast cancer is proportional to dose and that the increased risk in an exposed adult female population, after the lapse of 10 years following a radiation dose, is 5 cases per million per year per rem. Then in a population of 100,000 women each of whom received 10 ram we would eventually expect to find an excess of 5 cases of breast cancer per year; and in a period of 20 years, 100 excess cases. In that population of 100,000 women, over the same period of 20 years, we would normally expect to find about 1 in 20 women with breast cancer (without receiving radiation), that is about 5000 cases. The normal statistical variability of this number is such that the actual number of breast cancer oases is expected to range between 4860 and 5140 cases. Therefore, an additional number of 100 cases over the norma1 expectation of 5000 could be due to chance variation and could not be attributed to the effect of radiation with any confidence. The predicted effect from 1 rem would be 10 times smaller, namely 10 extra cases in 20 years, an increase even less significant. These statistical considerations show that the size of the irradiated and control populations necessary to demonstrate a significant increase in breast cancer from 1 rem would be greater than 10 million.

As might be expected, most of the human data showing increased cancer following radiation exposure apply to large doses of radiation. Occupational and environmental doses are generally low (in the range 0.01 to 5 rem per year) and often delivered In small increments over a period of many years. Largely because of the statistical problem illustrated above, there is little reliable data at such low doses (see Appendix A for a discussion of low-dose cancer studies), and to arrive at estimates of risk it is necessary to project downwards to these low doses from observed increases in cancer at high doses. The all-important shape of the dose-effect curve or curves, particularly for low

LET radiation. is known only at relatively high doses and assumptions about the shape must be made to estimate low-dose risks.

9. Human data on radiation-induced cancer.

On the basis of the wide range of doses received, the sexual distribution, and the wide age range of the exposed population, the best source of human data is the study of the Japanese A-bomb survivors now available for 33 years of follow-up (through 1978). About 82,000 persons exposed to an average absorbed dose to the whole body of' about 14 rem have been followed in the Life Span Study, and the number of' excess cancer deaths is about 250 (11). Doses range from 0 to more than 400 rem. The next largest group comprises 14,500 persons who received x-ray treatments to the spine for a form of' arthritis with spinal doses ranging from 500 to nearly 3000 rem (12). These patients subsequently showed a significant increase (approximately 140 cases) In leukemia l and cancer of a variety of organs in or near the treated area. Other important medically treated groups include:

a. Infants with enlarged thymus glands who were treated by x-ray doses typically in the 200 to 600 rem range. Of the approximately 2850 children treated, 30 have developed thyroid cancer, with thyroid doses ranging from 5 to 1100 rem (13).

b. Young women subjected to hundreds of fluoroscopic x-ray examinations during their treatment for tuberculosis. Skin doses ranged from 50 to several thousand rems over a long period of time. After about 30 years of followup, excess breast cancer has been found (14).

c. 10,000 immigrant children into Israel who were treated by x-rays over their entire scalp for ringworm have shown an elevated cancer rata in the brain, thyroid gland and several facial structures (15).

d. Children dying with cancer during the first 10 years of life. Several studies have shown that the mothers of these children had a higher

than normal incidence of abdominal x-ray examinations during the pregnancy. On this basis a controversial claim has been made that fetal irradiation at doses of approximately 1 rem increases the risk of childhood cancer (see later discussion) (16).

Victims of fallout in the Marshall Islands during nuclear weapons testing have shown an increased rate of thyroid cancer after receiving about 1000 rems of low LET radiation from radioactive iodine in their thyroid glands (Ref. 17). On the other hand, no) excess thyroid cancer was found in about 1400 children in southern Utah who received about 50 rem to the thyroid gland from drinking contaminated milk after atmospheric atomic bomb testing in Nevada (Ref. 18). However, a disputed study published in 1979 claims to show excess leukemia in children raised in high fell-out areas in Utah during the 1950's, although no excess malignancies of other types has been found (Ref. 19) (see Appendix A).

In connection with occupational exposure it has long been known that U.S. radiolagists starting their practices in the 1920's and 1930's developed excess leukemia and other cancers. The doses received are not known but are estimated in the hundreds of rems. The most recent group of radiologists (starting practice in the 40's) do not show increased leukemia although a few other forms of cancer such as skin cancer and multiple myeloma are elevated (Ref. 20). Strangely, multiple myeloma was not elevated among the radiologists who entered the profession two decades earlier and who probably received considerably greater radiation doses. However, a group of 6500 x-ray technologists trained in the U.S. Army in World War II and Followed for 30 years have not shown an increased cancer rate; again, doses are unknown (Ref. 21).

Occupational exposure to radon gas which emit high LET alpha particles has produced excess lung cancer in uranium miners in several countries. However, the relationship between lung cancer and radiation exposure is clouded by the

fact that most of the lung cancer cases are among smokers, who constitute a majority of the miners. The excess mortality from lung cancer is significant for doses down to about 200 rem. and the relationship to dose appears linear. The current absolute risk estimate is about 3 cases per year of lung cancer per million persons exposed to 1 rem (2).

Bone cancer has been produced by radioisotopes absorbed internally into bone, primarily by alpha emitters such as radium. After World War II several thousand German patients received injections of radium-224 (3.6 day half life) for treatment of tuberculosis and arthritis. About 60 cases of bone cancer in 2000 patients have been identified (2). In addition, about 2000 persons who absorbed radium-226 and radium-228 (much longer-lived isotopes with 1600 year and 5.8 year half lives) during their occupation as dial painters have been studied in the U.S.A. (2). About 80 cases of bone cancer have been identified, but only one case is known to have received a bone dose less than 20,000 rem (Q = 20). A risk of about 1 case of bone cancer per million persons per rem of bone surface dose has been deduced from these studies to date, assuming a linear (proportional) relation between dose and effect (2).

10. Animal data.

Information on the relationship between cancer induction and radiation dose, particularly at low levels, can be derived from animal experiments. In order to derive precise estimates of the number of cancers induced at low dose levels, large numbers of animals must be irradiated. Some of the best data are those of Robert Ullrich and his colleagues at Oak Ridge National Laboratory (1976-1979) for mice exposed to increasing doses of gamma rays or neutrons (22,23). The gamma ray data show a variety of responses for low doses ranging from a significantly reduced incidence for a few tumor types, to a threshold relationship, and to a curved relationship, concave upward, or a linear response for other tumors (see Figure 3).

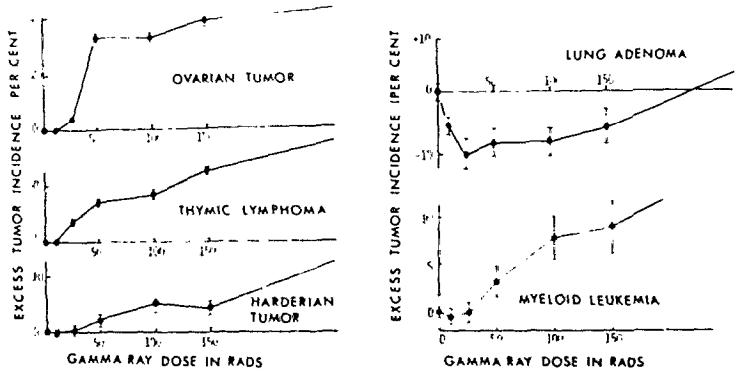

Figure 3. Dose response curves for five selected neoplasma in irradiated mice. Ovarian tumor, thymic lymphoma, Harderian tumor and lung adenoma were induced in female RFM mice and myeloid leukemia was induced In male RFMf/Un mice by whole body irradiation at 45 rads/minute with gamma rays from cesium-137. The curves are plotted from the data of R. L. Ullrich and his colleagues (22,23) after subtraction of incidence In unexposed control animals, Standard errors are shown as vertical error bars at each point: where not shown the error is contained within the sire of the point. (Reproduced by courtesy of E. W. Webster and the American Journal of Roentgenology, (25).

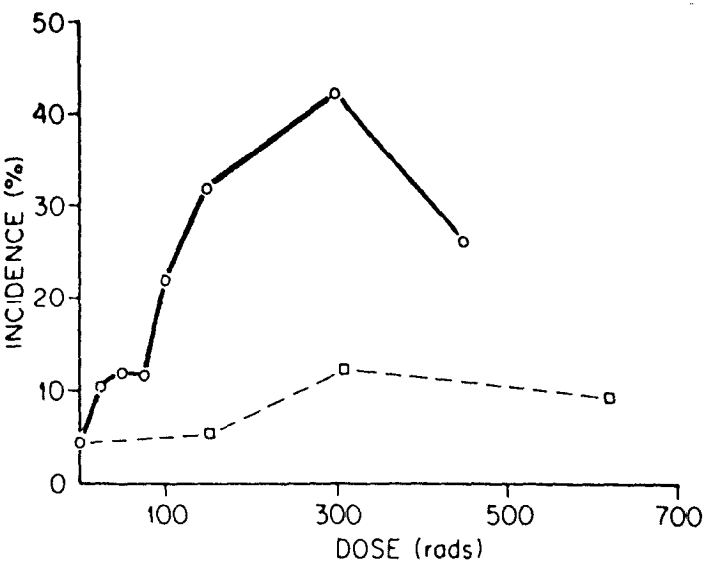

Figure 4. Myeloid leukemia in male mice following exposure to low LET radiation. Open circles: single dose. Open squares: small daily doses. Abridged from A. C. Upton in The Effects of Populations of Exposure to Low Levels of Ionizing Radiation (BEIR I Report). National Academy of Sciences, Washington, DC 1972 (42).

Leukemia induction by x-rays in mice (24) is much lower for low dose-rates than for high dose-rates as shown in Figure 4; i.e., there is a large degree of repair. A concave upward response (e.g., quadratic or linear-quadratic) has been found for bone cancer induced In a variety of mammals by strontium-90, a radioisotope produced in atomic bomb fallout (and in nuclear reactors) which emits low LET beta rays with a 30-year half-life (2). An example of a linear response is the induction of mammary cancer in female rats by x or gamma rays which appears linear down to quite low doses (15 rem). Because of the differences between animal and human cancers and the great difference in lifespan, the animal results suggest only general principles and cannot be used directly for risk estimation, although there is evidence that the percentage increase in cancer (i.e., the relative risk) from a given dose of radiation la similar for small rodents and for man (25).

11. Risk estimates for human cancer induction.

These have been reviewed by the BEIR Committee of the Rational Academy of Sciences. which published updated estimates in 1980 (2). Risk estimates may be specified in two ways:

a. the increment of risk for a given level of absorbed done; for example, the excess number of cancer cases In 1 million persons exposed to 1 rem (the “absolute” risk).

b. the percentage increase over the normal risk of cancer development (the "relative" risk).

The risk may be estimated for cancer induced in a given organ (e.g.. thyroid, lung), or for the total number of cancers of all kinds following exposure to the whole body.

Since there is no adequate knowledge for low doses, as explained earlier, the estimates depend on the shape of the dose/effect relation which is assumed.

When the estimate is based on high dose effects (e.g.. from therapeutic irradiation of parts of the body), it has in the past been usual to assume that the low-dose effects are proportional to dose. It is usual also to qualify such estimates by saying that they are upper limits of risk for low LET radiation (i.e., probably over-estimates).

The 1980 Report of the BEIR Committee (2) considered 3 methods by which the agreed risks of high x-ray and gamma ray doses (low LET) can be projected downwards into estimates of low-level radiation risks (see Fig. 51. These projection models are (1) the linear (proportional) method noted above, (2) the "linear-quadratic" method (shown as the curving line with a straight section at low doses in Figures 2 and 5, and (3) the "quadratic" or "dose-squared" method which employs a curving relationship down to the lowest doses, with zero slope at zero dose. There is evidence for all of these relationships, but based primarily on evidence from experiments with animals and living cells the BEIR Committee preferred the "linear-quadratic" method. This method, which predicts affects at low doses somewhat less than those derived from the linear approach, is supported by an analysis of the leukemia incidence In Nagasaki and Hiroshima employing a recent re-evaluation of the radiation dose distribution in those cities (26). The linear approach, on the other hand, is generally believed to afford a realistic estimate for high LET radiation (neutrons and alpha rays).

Table 7 gives for the three methods, estimates of the number of excess cancer deaths in a million people in the general population after receiving a single dose of 10 rem of low-LET radiation to the whole body. The preferred estimate by the BEIR Committee is derived using the linear-quadratic dose/effect relationship and future projection using the absolute risk model (2).

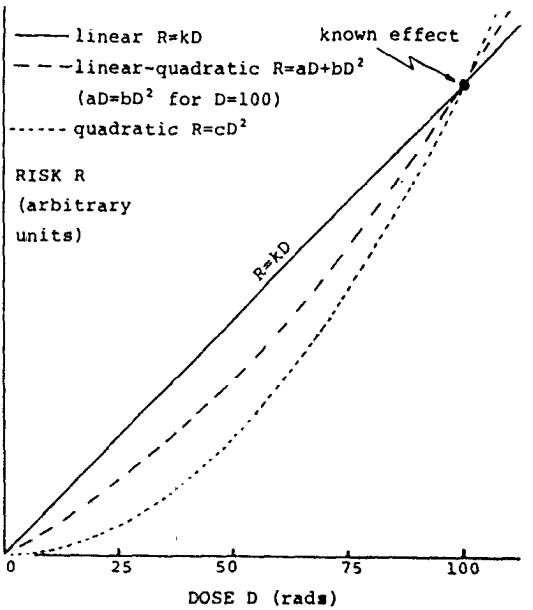

Figure 5. Some dose response relationships. These are frequently employed for predicting low dose "stochastic" effects of low LET radiation (e.g. cancer induction). In these examples it is assumed that a precisely known risk is established at 100 rads (rems) and downward projections to lower doses may follow linear, linear-quadratic or quadratic relationships shown in the diagram. (Courtesy of E. W. Webster and the American Journal of Roentgenology, (25).)

TABLE 7

Lifetime risks of cancer deaths per million persons following a 10 rem whole body dose of x-rays or gamma rays.

Dose response model	Excess cancer deaths			
	Leukemia, Bone cancer	Other Cancer	Total Cancer	% of normal expected cancer
Quadratic	30	65	95	0.058
Linear-quadratic	230	536	766	0.47
Linear	475	1196	1671	1.0

Thus the prediction of the preferred method is that there will be about one increased cancer death during the remaining lifespan of 10,000 persons each of whom received 1 rem (1000 millirem) of radiation to the whole body, or about two extra cancer cases. These estimates are consistent with (and primarily based on) the increased cancer found in the Japanese A-bomb survivors. For comparison, the number of cancer deaths normally expected in 10,000 persons is now about 2100, and the number of cancer cases expected is about 3600.

Although the linear-quadratic (curvilinear) dose response relation was preferred by the BEIR Committee, there is considerable support both old and new for the linear relationship in thyroid cancer and In breast cancer:

a. in the report of Modan that late thyroid cancer was induced in Israeli children by doses of about 6 rem of x-rays (15), and in the most recent analysis of thyroid cancer in persons irradiated in Infancy for enlarged thymus glands (13).

b. in the most recent analyses of increased breast cancer in the Japanese A-bomb survivors where the trend strongly favors a proportional relation to dose but does not rule out a curved relation (27).

Three developments since the BEIR Report was prepared are likely to cause revisions in the above risk estimate. The first is the increasing evidence that

certain radiation-induced cancers such as those of the lung and the breast may increase with the aging of an exposed population according to the “constant relative risk" model. This application of this model to all cancers except leukemia and bone cancer could lead to a risk increase by factors of 1.5 to 4 depending on the age grouping of the population as illustrated in the BEIR Report (2). The second source of change will be the availability of further follow-up data on cancer in the Japanese A-bomb survivors (specifically the extension from 1978 to 1985). The third source will be the re-evaluation of radiation doses received by the A-bomb survivors (28) which is expected to be finalized in 1986. These latter sources may affect both the risk estimates and judgments on the preferred dose/effect relationships.

GENETICS AND RADIATION

12. Genetic effects.

Genetic effects occur from permanent changes in the genes (gene mutations) or in the chromosomes carried by the germ cells in the reproductive organs (ovaries and testes) of the body. Gene mutations, which are responsible for a large fraction of birth defects, are almost entirely inherited from our forebears, both ancient and modern. Genetic effects are the cause of many disabilities and illnesses including sac types of neurologic degeneration. hemophilia, mental retardation, mongolism, and sickle-cell anemia. Radiation exposure is one of many agents that can cause gene mutation or chromosome damage, and is one of the minor contributors.

There is little direct evidence of radiation induced genetic effects in humans, and most of the data used to predict human genetic effects are based on mouse experiments. Figure 6 illustrates a visible mutation in the form of a

coat color change in a mouse born of an irradiated parent. The frequency of radiation-induced gene mutation is believed to be proportional to radiation dose delivered at low dose rates. It has been shown that some repair of the immediate damage will occur, since radiation delivered in fractions an hour or more apart produces fewer mutations than when delivered in a single dose (29). Chromosomal defects from low LET irradiation are generally not a linear function of radiation dose (30). However. chromosomal defects have been observed as a result of human irradiation with doses as low as a few rads (31).

The probability of inducing a mutation in the average gene of the mouse subjected to 1 rem of radiation dose at a low dose rate is considered to be about 4 per 100 million (2). The sensitivity of the genes to radiation is often evaluated by estimating the dose necessary to double the normal "spontaneous" mutation rate. This is known as "the doubling dose." Since the normal mutation rate in the mouse Is about 400 per 100 million per gene per generation, the doubling dose at low dose rates will be about 100 rem. Only a small fraction of these mutations art "dominant", i.e., will product a change in the characteristics of the offspring even though the gene is transmitted by only one parent.

13. Factors affecting genetic risk.

Several factors have been found to affect the probability of radiation-induced mutations in the mouse, particularly the female mouse; these include dose-rate, fractionation of the dose as noted above, and time interval between irradiation and conception. For irradiation with x or gamma rays, the mutation rate falls as the dose rate is reduced from about 100 rem/minute for both male and female mice. But whereas for the male it stabilizes below about 1 rem/minute at one-third of the rate for acute exposure (32), it continues to fall in the female until at 0.009 rem/minute it is not significantly different

Figure 6. A change in coat color of a mouse produced by a mutation Induced by the irradiation of one parent. (Reproduced through the courtesy of Dr. W. L. Russell, Oak Ridge National Laboratory.)

from the spontaneous mutation rate. In other words, at the lowest dose rate the radiation produces a very small if not zero mutation effect in the female (33). A dose rate of 9 millirem/minute (540 millirem/hour) is very high compared with most occupational exposure rates. As might be expected, a reduction in mutagenic effect also occurs when x-ray doses are delivered in several fractions with an intervening time delay, as indicated by the data in Table 8 for female mice. The effect has been attributed (by William Russell, who first noted this effect) to partial or almost complete repair of the primary genetic damage, as noted earlier (29). In addition. as shown In Table 9, lapse of a period of 7 weeks or more between the radiation exposure and conception in the female mouse also reduces the number of genetic changes in the offspring to zero, this effect occurring both for x-ray and neutron exposure (29).

Thus for radiation gradually delivered, as In occupational or environmental exposure, it is likely that the genetic risk to the female population is much smaller than that of the male population.

TABLE 8

Effect of fractionation of x-ray exposure
on mutation frequency in female mice

X-ray dose	Mutations per million offspring/rem
400 rem	3.6
50 rem	1.19
50 rem x 8*	1.39

*fraction at 75 minute intervals (29)

TABLE 9

Effect of mating delay on expression of mutations following irradiation of female mice

Radiation type	Dose (rem)	Time between radiation and conception	No. of offspring	No. of mutations
Neutrons	630*	less than 7 weeks	89,301	59
		more than 7 weeks	120,483	0
X-rays	50	less than 7 weeks	127,391	10
		more than 7 weeks	54,621	0

Ref. 29

*QF = 10

The BEIR Committee (2) concluded that the radiation-induced mutation rate in mice for low dose or low dose rate radiation relative to the spontaneous rate was in the range 0.5% to 1.0% for 1 rem of dose to the gonads. The estimated doubling dose was, therefore, in the range 100 to 200 rem. For man a greater range of uncertainty was specified based on the mouse estimate, namely 50 to 250 rem. It is of Interest that a recent analysis (34) of first generation offspring of the Japanese A-bomb survivors, while showing no significant increase in genetic damage, suggested a doubling dose of 156 rem for radiation delivered rapidly as in Japan. This estimate was interpreted by the authors as being equivalent to a doubling dose of 468 rem for radiation delivered at low dose rates.

14. Non-disjunction.

Non-disjunction, i.e., failure of the chromosomes to segregate properly, an important cause of spontaneous abortion and of serious abnormalities such a Down's syndrome (mongolism) in liveborn children. There is some evidence in animal and insect populations that the frequency of non-disjunction in the offspring increases following preconception irradiation. Laboratory studies on irradiated cells suggest that the doubling dose for disjunction is about 500 rads. The BEIR Report (2) reviews the conflicting results of several studies 1 humans of the incidence of Down's syndrome Following preconception irradiation of the mother, and suggests that a cause-and-effect relationship is uncertain. One important observation is the failure to observe an Increase in Down's syndrome among the children of the Japanese A-bomb survivors. Nevertheless, it is prudent to believe that there is a small risk of radiation-induced non-disjunction effects from preconception irradiation.

15. Risk estimates.

The 1980 BEIR Report (2) as shown in Table 10, concludes that 5 to 75 additional serious genetic disorders per million liveborn offspring will result from 1 rem received by the population over the generational tint of 30 years. If this dose were maintained constant for many successive generations, allowing "recessive" mutations to be expressed and for some elimination of mutations from the population, this number will increase gradually in each generation and reach an equilibrium after many generations estimated in the range of 60 to 1100 serious genetic disorders per million liveborn children. These numbers are to be compared with approximately 100,000 children per million (about 10% of all children) who normally show some serious genetic defect. For one rem given on one occasion the genetic risk to subsequent offspring appears to be somewhat less than the cancer risk in the irradiated person, the difference depending on the assumptions made about the dose/effect curve for induction of cancer.

TABLE 10

Estimated genetic effects of an average exposure to a human population of 1 rem per 30-year generation (BEIR III 1980)[2]

Serious genetic disorders per million live-born offspring		
Without radiation	With 1 rem/generation	
Current Incidence	First Generation	Equilibrium
107,000	5-75 extra	60-1100 extra

EFFECTS OF FETAL IRRADIATION

Probably the effect of radiation which causes most concern is the possibility of damage to the developing fetus. This is because there is some evidence that the embryo or fetus is more sensitive to radiation than the adult. The two principal effects are:

i. malformation and growth defects, particularly in organs which were developing at the time of irradiation;

ii. cancer developing during childhood

16. Developmental defects.

X-rays were one of the first agents observed to produce such defects. During the 1920's pregnant women who had received therapeutic irradiation of the abdomen were found to be at unusually high risk for a malformed child. Doses given were typically in the range of a few hundred rems. Later studies showed a wide range of abnormalities at these high doses, some of which are listed in Table 11. This was followed by experimental work on animals which showed parallel effects.

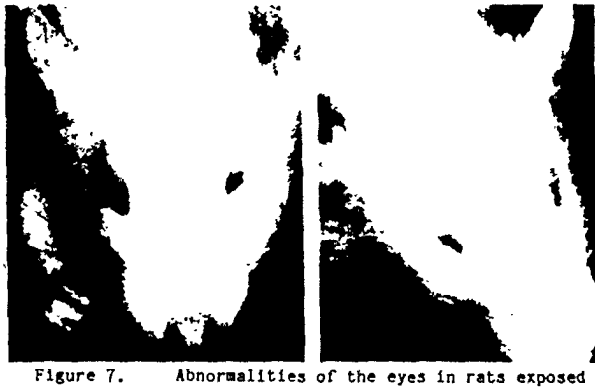

Figure 7. Abnormalities of the eyes in rats exposed during gestation to 150 rems of x-rays. The rats in the same litter were exposed at 9 days gestation. The rat on the left has a normal right eye and an abnormally small left eye, while the rat on the right has neither eye. (Reproduced from Ref. 35.)

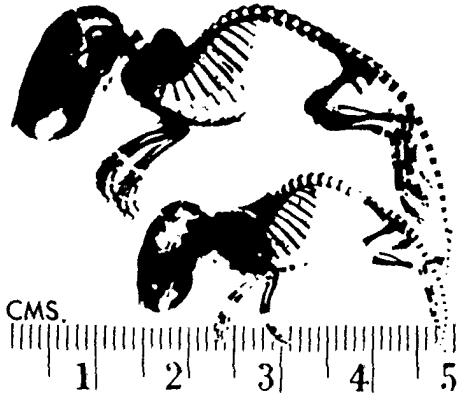

Figure 8. Stunting of growth in a mouse exposed during gestation to 150 rems of x-rays. Radiographs of the skeletons of two mice from the same colony are shown. The control mouse (not irradiated) is at the top. The mouse below. exposed at 14 days gestation, has an abnormally small skeleton throughout the body but no specific malformations or missing bones. (Reproduced from Ref. 35.)

Figures 7 and 8 show typical and graphic examples: abnormally small eyes (microphthalmia) and congenital absence of eyes (anopthalmia) in rats after irradiation with 150 rems of x-rays during gestation; and second, stunted development in a mouse but no evident malformations, after fetal Irradiation with 150 rems (35).

TABLE 11

Some Human Abnormalties Reported After
Large Radiation Exposure In Utero

Growth retardation	Microcephaly (small head)
Blindness	Microphthalmi (small eyes)
Mongolism	Hydrocephaly (abnormal fluid in brain)
Skull malformations	Chorloretinitis (eye inflammation)
Skeletal abnormalities	Strabismus (eye deviations)
Mental retardation	Coordination defects
Ear abnormalities	Genital deformities

These effects are strongly dependent on three factors: development stage, radiation dose and radiation dose-rate.

a) Stage

There are three significant periods in pregnancy: first, a rather short preimplantation stage, then an extended period of major organogenesis. and then the fetal stage where differentiation is complete and growth mainly occurs. The times for these periods art shown in Table 12 for the important experimental animals, mouse and rat, and also for man.

TABLE 12

Time Scale of Embryo/Fetal Stages

	Times in Days After Conception		
	Preimplantation	Organogenesis	Fetal
Mouse	0-4.5	7.5-12.5	13-20
Rat	0-5.5	8.5-13.5	14-32
Human	o-9	14-50	51-280

Figure 9 illustrates schematically the times and degrees of sensitivity to various adverse effects following a fetal dose of 100 rems delivered at different times during gestation (361. In the preimplantation stage, which lasts about 9 days in man, the major effect is embryonic death. There is no chance of malformation being produced by irradiation at this stage. The organogenesis stage runs from the 3rd to the 7th week (14-50 days) and is characterized by a sudden large increase in radiosensitivity. A great many malformations and growth deficiencies may be produced during this period, continuing with lower likelihood into the fetal stage. The particular type of malformation depends upon the organ system being differentiated at the time of the irradiation. The peak sensitivity for man is in the 3rd and 4th weeks. During the later fetal stages an irreversible loss of cells occurs. The oocytes are particularly sensitive and the death of some of them leads to a reduction in fertility of the offspring.

Table 13 summarizes these various effects associated with the stage of development.

b) Dose.

The probability of malformation and growth retardation falls steeply as the dose is reduced. In animal studies malformations have not been produced

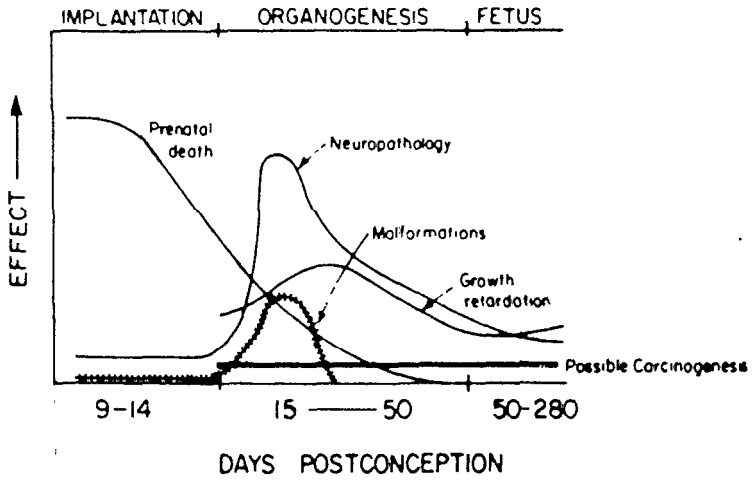

Figure 9. Adverse effects of radiation on the embryo/fetus related to the time period during gestation when the irradiation occurred. Large doses (e.g., 30 to 150 rem) early in pregnancy in the pre-implantation period may cause embryonic death. The major effects particularly those related to the development of the central nervous system, occur following irradiation in the period of organogenesis. Malformations have been observed at doses greater than about 10 rems delivered in the early stages of this critical period. but not at later periods of fetal development when growth retardation is the principal effect. (Reproduced by courtesy of authors and publishers from Medical Effects of Ionizing Radiation by F. A. Mettler and R. D. Moseley. published by Grune and Stratton, 1985.)

following x-ray doses below about 15 rem (37). An apparent small increase in skeletal malformations in mice with doses as low as 5 rem was confounded by a seasonal (summer vs. winter) dependence of the results (38). One of the lowest dose experiments was conducted by L. Russell on mice in 1957 (39). Mice were irradiated at the most sensitive period with 25 rems. Ho gross malformations were noted but there was variability in the number of ribs.

In Hiroshima there was a significant increase in microcephaly (small head) in children who were exposed to atomic-bomb radiation during the first half of the gestation period with average fatal dose originally estimated as 5.7 rem from gamma rays and neutrons (2), but perhaps about 12 rem according to more recent estimates (28). In Nagasaki no significant increase in microcephaly occurred with fetal doses below about 50 rem.

TABLE 13

Effects of 100 Rem of X-ray Dose on Early Mammalian Development (35)

Effect	Stage at Irradiation		
	Preimplantation	Organogenesis	Fetal
Early embryo death	++	+	
Neonatal/postnatal death	0	++	0
Gross malformations	0	++	0
Reduced birthweight	0	++	+
Central nervous system defects	0	++	+
Reduced fertility	0	++	++
Growth retardation	0	++	+

\+ effect observed with low frequency for particular development age.
++ effect observed for most of stage with frequency greater than 25% for some development ages.

There is a recent and controversial report (40) that the frequency of mental retardation appears to increase linearly with dose in children exposed during the A-bombing of Hiroshima between weeks 8 and 15 of gestation; the estimated risk is about 1 in 250 par rem. Generally, however, malformation and growth retardation are the consequence of relatively high doses of x or gamma radiation, with very small probabilities of occurring after low doses.

There have been several studies of malformation incidence in infants who received diagnostic x-radiation in utero, and no convincing positive results have emerged. Typical is the 1968 study of Mokkentved in Denmark of 152 exposed children and 141 matched controls not irradiated (41). No significant excess of malformations was seen (15 in irradiated children versus 13 in controls) in this typical statistically limited study.

Although many observers have concluded from the above findings that there was an effective threshold dose for gross malformations, ranging from 25 rem in the first report of the BEIR Committee of the National Academy of Sciences (42) to 10 rem (43), a small risk at lower doses cannot be ruled out.

In its most recent review (44) the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has proposed an upper limit of combined radiation risk for several fetal affects (mortality, induction of malformation, mental retardation and childhood cancer) of 3 chances per 1000 children for each rem of fetal dose. This estimate was obtained by weighting the separate risks according to the fraction of gestational time over which they occur. The estimate for each of these 4 affects separately would be somewhat lower. The normal total risk for these conditions in the absence of radiation was estimated as 60 chances per 1000 children (6%).

c) Dose-rate.

Reduction of dose-rate generally reduces or eliminates the likelihood of malformation or growth retardation. Table 14 show how some important fetal

effects of a large x-ray dose (150 rem) are greatly diminished when delivered over periods of several hours instead of minutes (44). The data of Table 14 are based on 52 irradiated pregnant rata and the examination of 251 surviving fetuses.

TABLE 14

Percent malformation rate in rats irradiated with 150 rems of x-rays at four different doze rates (45)

Irradiation time	1.5 min	4.5 min	2 hours	5 hours
Small head (microcephaly)	9.1	41.1	20.3	0
Loss of brain (anencephaly)	30.3	13.7	3.1	0
Congenital kidney absence	21.2	5.5	2.6	0
Cleft palate	51.5	37.5	17.8	11.5
Small mouth (microstomia)	31.8	16.4	7.8	0
Loss of bowel (evisceration)	16.7	6.8	4.7	0
Loss of ear	42.4	20.5	21.9	0
Malformation of limbs	43.9	16.4	3.1	1.3

17. cancer from fetal irradiation.

It is often said that the risk of childhood cancer following fetal exposure (particularly leukemia) is several times greater than the risk of cancer induction in adults from the same radiation dose. This assertion, while it represents a prudent working hypothesis, remains at present unproven.

From 1958 to 1976 Dr. Alice Stewart and her associates published successive results of the so-called Oxford Survey of childhood cancer cases in England and Wales (16). This was a "case/control study." Children who died From a malignant disease were compared with matched living control children with respect to factors which may have been related to their cancers. It was noted that the proportion of the dead children who had received x-ray exposure during gestation was greater than the proportion of living control children who were x-rayed, although not all of the dead children had been x-rayed in utero. From this it was concluded that x-ray examination during pregnancy was associated with childhood cancer and that the radiation exposure increased the chance of childhood cancer by about 50%. Table 15 shows the details of the most extensive study (16) and the conclusion that 1 rem of in utero x-ray exposure to 1 million children might cause about 500 extra cancer deaths in those children before age 10. This projects on the average 1 extra cancer death in 2000 exposed children. Since the normal childhood mortality From cancer is about 1 per 1000 (or 2 per 2000), this would be an increase of 50%.

The unresolved problem with this study. and several other similar studies reporting similar results, is that association does not prove causation (46). Since diagnostic x-ray examinations (mainly pelvimetry in these studies) were given to a selected group of pregnant women. it may be that the apparent cancer risk was predicated by that selection. In Great Britain at the time of the Oxford Survey only about 10% of children ware x-rayed in utero.

It was subsequently noted (1974) by Mole in England (46) that about 55% of twin births ware x-rayed in the Oxford Survey, and despite the much smaller degree of selection the excess cancer risk persisted. This is an argument against the criticism that the risk is apparent rather than real because of a selection process.

TABLE 15

Childhood malignancy following prenatal x-radiation (16)

No. of childhood cancer cases, 0 to 10 years old		7694	
No. x-rayed in utero		1141	(14.9%)
No. of matched controls without cancer		7694	
No. x-rayed in utero		774	(10.11)
Case/control ratio of x-ray examinations		1.97	
Conclusions:	Relative cancer risk	1.5	
	Excess deaths per million per rem	572	
	Range	300 to 800	

However. there Is direct evidence against the thesis of Dr. Stewart. In England in the late 1950's Court-Brown, an eminent epidemiologist, carried cut a prospective study on 39000 children of women who had received abdominal x-ray examinations in pregnancy and compared the number of leukemia deaths to those expected on a national basis (48). The study is summarized in Table 16. There was no excess leukemia in the irradiated children whereas there should have been five excess deaths over the expected number according to the above conclusions of the Stewart study.

TABLE 16

Childhood Leukemia Following Prenatal X-radiation (48)

No. of women receiving diagnostic abdominal x-rays	39,000
No. of leukemia deaths in children	9
No. of leukemia deaths expected (U.K. statistics)	10.5
Excess leukemia	zero
Probability of observing 9 instead of 15.5 as predicted by Stewart et al	0.04

Another population which should shed light on cancer incidence in children irradiated in utero is the 1290 children in gestation at the time of the A-bomb detonations in Japan. These children were followed up to age 10 by Jablon and Kato (49). Table 17 shows the results. Only one cancer case was found and this was the expected number in 1290 children without radiation. According to the Stewart study at least 5 excess cases should have been found with a beat estimate of 10, and therefore the studies are In clear disagreement.

TABLE 17

Childhood Cancer Mortality Following Radiation Exposure
In Utero During Japanese A-Bombing (49)

No. of children exposed during gestation	1292
No. exposed to less then 500 rem air dose	1250
Estimated mean fetal dose (rem)	14
Observed cancer deaths, during first 10 years	1
lo. of expected cancer deaths	0.75
Excess predicted by Stewart and Kneale	
Range:	5.2-14
Best estimate:	10

RADIATION PROTECTION DOSE LIMITS

18. Maximum permissible doses.

There are four sets of maximum permissible doses (MPD) depending on the segment of the population involved:

a. Occupationally exposed persons

b. Members of the public near radiation installations

c. The whole population (average dose)

d. The developing fetus

These standards for maximum dose have been set both nationally and internationally by expert committees. In the U.S.A., the primary group is the National Council on Radiation Protection and Measurements (NCRP), which operates independently under a Congressional charter and whose recommendations are usually adopted by federal and local government agencies. New members of the Council are nominated on the basis of their expertise by professional societies, existing members and others, and elected by the Council. International standards are published by the International Commission on Radiation Protection (ICRP).

The early history of the occupational standards, summarized in Table 18, was one of steady reduction as more information on the effects of radiation was obtained and improved technology became available. The occupational MPD has been unchanged since 1957.

TABLE 18
50-year history of the occupational Maximum Permissible Dose

1934	200 mrem/day	(ICRP)
1936	100 mrem/day	(NCRP)
1948	300 mrem/week	(NCRP)
1957	100 mrem/week	(NCRP) (5 rem/year)*

*current value

The threefold reduction in 1957 was occasioned by concern over the genetic effects of radiation. Since then this particular concern has lessened and the long-term induction of cancer by radiation has emerged as the primary concern.

The NCRP recommended In 1957 that persons near radiation installations but not occupationally exposed (including minors) should not receive more than 10%

of the occupational MPD, i.e., 500 mrem/year whole body (50). Also, following a recommendation of the U.S. National Academy of Sciences in 1956, a new limit for the population as a whole was introduced by the NCRP on genetic grounds: concern for the public health problem of increased inherited disease induced by the general irradiation of a whole population. This limit was 5000 mrem/30 years or 170 mrem/year (average population dose), in addition to background radiation (50). Subsequently the U.S. Nuclear Regulatory Commission (51) added the shorter term requirements that the dose shall be limited to 2 mrem in any one hour and 100 mrem in any 7-day period in unrestricted areas assuming full occupancy of those areas.

At its meeting in 1985 the International Commission on Radiological Protection (ICRP) (52) modified the application of the limit of 500 mrem/year for members of the general public in the vicinity of radiation facilities. This limit was considered to be acceptable for occasional exposures received for some years, but for annual exposures over a lifetime the recommended limit was an average of 100 mrem per year. It is likely that this latter limit for long-continued exposure to specific members of the public will also be adopted by the MCRP in the near future. 100 mrem/year is roughly the average dose from background radiation in the USA, so that this individual limit over a long period would represent an additional background dose. The risk associated with 100 mrem/year is calculated to build up to 1 extra cancer death in 100,000 persons per year after sustaining many years of such exposure. It should be emphasized that no studies of human populations to this time have actually shown an increase in cancer in populations living in regions of the world with higher than normal background.

The maximum permissible dose does not represent a boundary between safety and harm. It is prudently assumed (but not proven) that any radiation exposure,

including background radiation, carries some degree of risk that harm will ensue, i.e., there is no threshold below which there is no risk. The probability of the harmful result is believed to increase with the dose level received, each new increment of dose increasing the overall level of risk. The MPD concept rests upon a judgement of acceptable risk. Depending on the importance of the objective which necessitates the radiation exposure, a greater level of risk can be tolerated. Thus, for life-saving purposes an emergency radiation exposure of 25 rems can be accepted on a voluntary basis. The increased risk of subsequent cancer even with this dose would still be small. According to current estimates the lifetime risk of developing cancer in the l average person 20 to 35 years old after a 25 rem dose would increase from 37% to 37.5%.

19. Radiation and other risks: l comparison.

Many of the effects that are produced by large doses of radiation. such as several hundred rems, will develop in a relatively short time and will affect every person exposed to a greater or lesser degree depending on their sensitivity; for example. skin reddening by radiation. The severity of the effect will also increase at higher doses. In contrast, the three effects that are possible at lower doses, namely cancer, genetic effects, and congenital defects following fatal irradiation, are expected to appear in a very small fraction of the persons exposed. The effects either appear or do not appear and there is no gradation of the severity of the effect. Like shooting at a target, it is a hit or a miss. Since radiation exposure rarely produces any of these three effects, particularly with low doses, it almost always "misses the target". These radiation effects should, therefore, be thought of as presenting a very small risk and the size of those risks, for example 1 in 10,000 following 1 rem to the whole body, has been discussed earlier.

In trying to comprehend the size of these radiation risks, it is helpful to compare them with other risks that people fear, voluntarily or involuntarily. during their everyday life. statistics are available on the occurrence of a variety of fatal illnesses produced by environmental pollutants, cancer-producing social habits and food additives, and fatal accidents in different kinds of human activity. If we assume, as is often done with radiation, that the risk is proportional to the amount of exposure to the agent or activity, then we can estimate the amount of each agent end activity which provides a risk equal to that from a certain amount of radiation. For example, from our earlier discussion we can deduce that a radiation dose to the whole body of 10 millirems carries a lifetime risk of 1 in a million that a fetal cancer could eventually result. 10 millirems is approximately the radiation dose averaged over the body from two chest x-ray examinations. It is also the increased radiation background dose incurred in moving to Denver from New York for two months or from flying coast-to-coast on two round trips. The same risk of death has been estimated (53) for travel using several types of conveyance listed in Table 19. The same risk has also been estimated (53) for consumption of various carcin-genic materials listed in Table 20.

TABLE 19

Risks of One in a Million (10 millirem equivalent) of Fatal Accidents During Travel

10 miles on a bicycle
6 minutes in a canoe
300 miles in a car
1000 miles in a commercial plane

TABLE 20

Risks of One in a Million (10 millirem equivalent) of Fatal Cancer from Consumption

Smoking 1.4 cigarettes
Drinking Miami water for 1 year (chloroform)
Drinking 30 12 oz. cans of diet soda (saccharin)
Eating 100 charcoal broiled steaks (benzopyrene)

A more direct ccmparison can be made between the cancer risk from an average smoking habit and receiving 10 rems (10.000 millirems) of whole body radiation over a period of time. On the basis that 21.6% of the population dies from cancer (1983) and 30% of cancer is smoking related (according to Doll and Peto (10), the death of 65 persons in every 1000 is attributable to smoking. In 1000 persons receiving 10 rems of radiation dose, only 1 person would be expected to develop a fatal cancer (2). By this measure, the effect of smoking In our society is 65 times more damaging than a population exposure of 10 rems, which is about the amount most people now receive in a lifetime. The average radiation dose to the whole body from all sources including background radiation is only about 0.2 rem (200 millirems) per year.

Radiation risks can also be assessed directly relative to other occupational risks. With an occupational exposure of 0.5 rem per year, continued for 50 years, the application of the mortality risk reviewed earlier indicates that radiation-induced cancer deaths would eventually build up to 50 per million per year. This figure is compared in Table 21 with the accidental death rate in other U.S. occupational groups for the year 1982 (54).

TABLE 21

Accidental Death Rate in Various U.S. Occupations for year 1982 (54)

Occupation	Deaths/million/year
Trade	50
Manufacturing	50
Service	60
Government	100
Transportation/Utilities	260
Construction	400
Agriculture	520
Mining/Quarrying	550
Radiation (estimated)	50

Death from cancer as a result of radiation exposure differs from death by accident In one important way: accidental death is immediate and frequently results in a large loss of lifespan, perhaps 40 or 50 years. But death from radiation-induced cancer follows radiation exposure by a time lag of at least 10 years for moat cancers and on the average about 20 years. Therefore, a fairer comparison between the risks would be baaed on life-span shortening.

The mean whole body dose to the U.S. population from medical x-ray exposure has been estimated as 75 millirems per year (55). and the mean occupational whole body exposure for persons receiving measurable doses as 230 millirems per year (56). The loss of life expectancy from these exposures can be estimated (55) as 6 days and 18 days, respectively. In comparison with these small losses, Table 22 shows the estimated losses due to a variety of causes.

TABLE 22

Loss of Life Expectancy in Days Due to Various Causes (55)

Medical x-ray exposure	6
Occupations involving x-ray exposure	18
.....	
Cigarette smoking - male	2250
Being 30% overweight	1300
Cigar smoking	330
Motor vehicle accidents	207
Alcohol consumption	130
All occupational accidents	74
Falls	39

On the basis of both the relative mortality in the population and relative loss of life expectancy from radiation exposure compared with other common hazards, it is obvious that radiation exposure constitutes one of' the minor risks in our society.

APPENDIX A.

Reviews of Controversial Studies of Increased Cancer In Populations Exposed to Low Levels of X-rays or Gamma Rays

There have been several controversial studies during the last decade which claim to show increased cancer rates in occupationally, medically, or militarily exposed groups of persons receiving doses of x-rays or gamma rays of the order of 1 rem, and that the cancer risk from radiation is very much greater than has been estimated by national and international committees. In each case the allegations are unproven, although In a few studies they cannot be completely discounted. Four of the following studies (numbers 1,2,4 and 6) were discussed in the 1980 BEIR report (2) with similar conclusions.

A-l The Mancuso Reports.

In 1977 and 1978 Mancuso, Stewart and Kneale published two analyses (57,58) of the deaths between 1944 and 1972 of 3500 male employees of the Hanford Atomic Plant in Richland, Washington as related to radiation doses recorded on film badges. Mortality from various types of cancer was compared for occupationally exposed workers (mean dose 2.1 rems) and non-occupationally exposed workers (mean dose 1.6 rems). Excess risk was claimed for cancer of the bone marrow, lung cancer, pancreatic cancer, and lymphatic cancer -- specifically, 125% per ram for bone marrow cancer, 16% for lung cancer, 14% for pancreatic cancer, and 40% for lymphatic cancer. These estimates were revised in the second paper to 28%, 7% and 6% for the first three cancers. Increases of 40% and 28% per rem signify doubling doses of 2.5 rems and 3.6 rems for lymphatic and bone marrow cancers, which suggest that radiation from normal background and medical l exposure alone will produce more of these cancers in the general population than actually occur normally.

Among the multiple published criticisms of these studies are the failure to adjust the data for confounding factors such as exposure to medical radiation, solvents, and smoking, and for the association between total dose, at death, and normal cancer risk. A crltlcal analysis of the same data by four nationally recognized epidemiologists (59) failed to find an association of radiation dose with myeloid leukemia, lymphoma and lung cancer, but did find an association with multiple myeloma and pancreatic cancer. However, the doubling doses of 1.5 rems and 5 rems estimated by Mancuso, et al. for these two cancers again suggest an impossibly large role for background and medical exposures in cancer induction. Moreover, these estimated risks are 20 to 100 times higher than those derived from the much larger studies of the Japanese A-bomb survivors and patients treated for ankylosing spondylitis. The confounding factors mentioned above may well account for these results. The Mancuso study has a statistical power about 600 times smaller than the Japanese study: as a consequence, negative results are almost as likely as positive results in finding excess cancer (59).

A-2 The Portsmouth Naval Shipyard Study.

In 1978 Najarian and Colton (60) published a preliminary study of relative cancer deaths between 1959 and 1977 In two groups of dockyard workers who were reported as exposed or unexposed to radiation. This classification was established by interviews with next of kin without reference to the radiation exposure records in the possession of the U.S. Navy. The study claimed that the exposed workers, who had received an average dose of less than 10 rem, had developed leukemia at a rate 5.5 times greater than the normal rate (namely 6 cases observed versus 1.1 case expected), which would indicate an absolute risk more than 100 times greater than that authoritatively estimated. Subsequently, the National Institute of Occupational Safety end Health (NIOSH) performed a

more comprehensive study of the cancer rates (61), classifying the workers as exposed or unexposed according to the dose information released by the U.S. Navy. The new study comprised 4566 deaths between 1952 and 1977 among the workers, whereas the first study comprised an 11% sample of 525 deaths. As shown in Table 23, the NIOSH study found no increase in either leukemia or cancer among the exposed workers. There was also no trend of increasing mortality from malignant disease with dose up to a maximum of 90 rem. Although the population of shipyard workers was relatively small, the NIOSH study had the power to detect a doubling of the leukemia rate.

TABLE 23

Leukemia and Other Cancer Mortality
Among Portsmouth Naval Shipyard Employees 1952-1977 (61)

	Total deaths	Observed	Expected	Ratio O/E
Exposed workers	833			
Leukemia		7	8.3	0.84
All cancer		201	218.5	0.92
Unexposed workers	3733			
Leukemia		31	29.1	1.06
All cancer		726	723.6	1.00

A-3 Cancer in Military Personnel at the Smoky Nuclear Test.

The mortality and cancer frequency among participants in the military exercises during and after the nuclear weapon test "Smoky" in 1957 has been reported for the period 1957-1979 by the Centers for Disease Control (CDC), Atlanta (62). Table 24 summarizes the mortality results from cancer. Of the 3217 participants, 3072 have been traced (95.5%) and their health status ascertained. 64 deaths from cancer were found compared with 64.3 expected based on age and sex specific rates for the USA. The only significantly increased malignant disease ($p<0.02$)* was leukemia, where 8 deaths (10 cases) were found versus 3.1 deaths (4 cases) expected. The mean cumulative 1957 dose from gamma radiation based on film badge readings was 0.456 rem. Only 20 persons received more than 4 rem (maximum 10.5 rem) and none of these has developed cancer. The mean cumulative exposure of the leukemia cases was 1.033 rem. The mean latent period for leukemia was 14.2 years.

The excess of 6 leukemia cases in 3072 persons with a mean dose of 0.456 rem represents an absolute risk of 4280 cases per million per rem over a follow-up period of 22 years. This is about 100 times the risk predicted by authoritative committees based on much larger studies. This result, assuming no dose rate factor, suggests that accumulated background radiation alone should produce a radiation-induced leukemia rate several times greater than the total leukemia incidence actually observed in the general population. The CDC has refrained from concluding that the excess leukemia is induced by the stated radiation levels, pointing to other factors which may account for the observation: subsequent exposure to chemical carcinogens, and the possibility of higher doses including unmeasured internal contamination. However, the unusually long mean latent period for the observed leukemia cases casts doubt on a radiation exposure etiology.

*The p-value is the probability that this result could have occurred by chance.

TABLE 24

Deaths from Malignant Disease Among Smoky Participants (62)

Neoplasm	Observed	Expected
All malignant	64	64.3
Digestive	15	15.6
Respiratory	21	22.2
Lymphomas	3	4.2
Leukemia	8	3.1
Other malignancies	17	19.2
No, of persons contacted	3072	
Estimated mean dose, rem	0.456	

A more comprehensive study (63) has subsequently been performed by the Medical Follow-Up Agency of the National Research Council stimulated by these findings for the Smoky shot. This larger study was commissioned to determine whether participants at nuclear weapons tests other than Smoky also showed an excess of leukemia or other cancer. Included in the new study were those military personnel present at the other shots in the Plumbob series in 1957 of which Smoky was a part, and those present at 4 other test series in Nevada or the Pacific Ocean; namely the Upshot-Knothole series (Nevada 1953). the Greenhouse series (Pacific 1951). the Castle series (Pacific 1954). and the Redwing series (Pacific 1956). The total number of personnel in the new study is 46,186, about 13 times the size of the revised Smoky cohort of 3554. The study was conducted in a similar fashion to the Smoky study by the CDC (62):

Table 25

Leukemia and the Smoky Shot:

Comparisons between Smoky and 5 Nuclear Test Series (63)

I. Dose distribution: percent of participants

		Doses in rem					
	Mean	<0.1	0.1-0.3	>0.3-1.0	>1.0-3.0	>3-5	5+
Smoky	0.571	37.5	23.7	24.7	11.3	1.7	1.1
Plumbob-Smoky	0.519	46.0	19.8	21.2	8.6	2.8	1.6
All 5 series	0.934	33.9	12.0	24.8	20.5	7.1	1.7

II. Observed (O) and expected (E) leukemia mortality

		Doses in rem						
		Unkn.	<0.1	0.1-0.3	>0.3-1.0	>1.0-3.0	3.0+	Total
Smoky	O	0	1	3	4	1	1	10
	E	0.13	1.63	0.87	0.85	0.38	0.11	3.97
Plumbob-Smoky	O	5	3	1	4	0	0	13
	E	4.71	4.61	2.06	2.20	0.69	0.33	14.60
All 5 series	O	16	16	5	10	5	4	56
	E	15.91	15.62	4.93	9.30	7.37	3.24	56.37

III. Time distribution of leukemia deaths

		Follow-up Years after exposure			Total
		1-10	11-20	21+	
Smoky	O	1	5	4	10
	E	1.12	1.69	1.14	3.97
Plumbob-Smoky	O	2	6	5	13
	E	3.77	6.24	4.60	14.60
All 5 series	O	10	22	24	56
	E	14.02	20.72	21.64	56.37

deaths observed in the sub-groups and the total group from various causes was compared with those expected. The number of deaths expected was calculated from U.S. vital statistics for each 5-year age group and for each year of follow-up after exposure.

The results of this much larger study arc summarized in Table 25 for the Smoky shot, for the Plumbob series minus Smoky, and for all 5 series. Section I of this table shows the dose distribution and average dose for those with known doses as recorded by film badges. The doses in Smoky and Plumbob-Smoky are similar but are considerably lower than in the 5 series together, where the Fraction of persons receiving more than 1 rem is doubled. Section II shows that there was no excess leukemia in Plumbob-Smoky or in the 5 series together. despite the higher doses in the latter. Section III shows the unusual distribution of leukemia with time after exposure for the Smoky personnel. The excess occurs only after the tenth year, whereas typically in other studies about one-third of the excess would have occurred in the first 10 years. The observation of three of the excess six cases more than 21 years after exposure is very unusual. An important conclusion of the new study (63) is that the excess leukemia seen in the Smoky shot participants is likely to be a statistical quirk. A random occurrence of such an excess is probable in one test out of the considerable number included in the 5 series where no other excess was observed. A similar conclusion holds for the single significant increase in cancer (of the prostate) seen only in the Redwing series.

A-4 Leukemia in Early Entrants to Hiroshima and Nagasaki.

Prof. J. Rotblat (64) has suggested that the many thousands of persons who entered the cities in the first few days after the bombing, for rescue work or in starch of relatives, developed leukemia with an absolute risk of about 160

deaths per million per rad during the period 1950-1967. This estimate is 4 to 9 times higher than the generally accepted range of leukemia risk. The 1980 BEIR Report (2) questions the accuracy of the dose estimates and the quality of the epidemiology on which the Rotblat estimate is based. Data on cancer mortality, including leukemia, among the early entrants during the period 1950-1978 (Nagasaki) and 1954-1978 (Hiroshima) recently published by the Radiation Effects Research Foundation in Hiroshima (65) are shown in Table 26 and indicate no significant increase in malignant disease.

TABLE 26

Observed versus Expected Deaths from Leukemia and Other Cancer Among the Early Entrants into Hiroshima and Nagasaki through 1978 (65)

	Deaths Observed	Deaths Expected
Hiroshima, 1954-78		
Leukemia	4	4.36
Other cancer	231	259
Nagasaki, 1950-78		
Leukemia	2	0.93
Other cancer	45	48

a based on all-Japan death rates

A-5 Childhood Leukemia Associated with Nuclear bomb Testing.

Lyon et al. reported in 1979 (19) on a study of leukemia and other cancer in children under 15 years of age during three time periods and in two areas in Utah: those counties receiving high fallout during the period of atmospheric nuclear weapons testing in Nevada (1951-58) and those counties receiving low fallout during that period. Mortality during this "high exposure" period was compared with that of children who reached age

15 In the years before 1951 and those who were born after 1958, both "low exposure" periods. It was concluded that the high exposure cohort in the high fallout counties experienced a leukemia rate approximately twice that Of the earlier and later low exposure cohorts, whereas an increase in leukemia was not seen in the low fallout counties. The claimed increase suggests that the absolute leukemia risk is 5 to 10 times greater than the generally accepted risk.

The above claim has drawn adverse criticism From Land, Bader, Enstrom, and Yalow (66-70), pointing out the statistical weaknesses and inconsistency of the study which cast doubt on the validity of the conclusion. The data shown in table 27 are based on small numbers of leukemia deaths and suggest that the apparent "increase" in the high exposure cohort is probably due to the unusually low leukemia rate seen in the low exposure cohorts in the high fallout counties (66,67). The leukemia rates in the high and low fallout areas were essentially equal for the 1951-58 cohorts. Moreover, as is shown in Table 28, the statistical distribution of the leukemia deaths in the high fallout counties following the start of testing in 1951 is peculiar, with cases clustered in the second and third year Following the 1957 year of high-yield tests while the aggregate number of oases is unusually low and identical in the 8-year periods preceding 1959 and following 1960. It is also striking that cancer other than leukemia apparently declined by a Factor of two for the high exposure cohort relative to the earlier low exposure cohort. Thus the total cancer mortality (leukemia plus other cancer) is constant for the low exposure and high exposure cohorts. Finally, a recent evaluation of the radiation levels due to Fallout in Utah during the 1951-58 period of weapons testing (71) concludes that the radiation dose level in the presumed "low fallout counties" of N. Utah was higher than in the “high fallout counties" of S. Utah, which would appear to refute the claimed significance of the Lyon paper conclusion.

TABLE 27

Mortality from Leukemia and Other Cancer in Utah 1944-1975 Including Period of Fallout from Nuclear Weapons (19)

Exposure Cohort*	Leukemia rate**	Other cancer rate**
High fallout counties:		
Low exposure 1944-50	2.1 ± 1.56***	6.36 ± 2.72
High exposure 1951-58	4.39 ± 1.59	3.07 ± 1.36
Low exposure 1959-75	1.96 ± 1.23	3.05 ± 1.61
Low fallout counties:		
Low exposure 1944-50	3.84 ± 1.13	4.52 ± 1.26
High exposure 1951-58	4.21 ± 0.69	4.33 ± 0.68
Low exposure 1959-75	3.28 ± 0.64	3.09 ± 0.62

* Children below age 15 during periods of high and low exposure

** Age and sex adjusted rates per 100,000 population

*** Two standard errors

TABLE 28

Yield from Nuclear Weapons Tests and Leukemia Deaths In High Fallout Counties, Utah, 1951-1969*

Year	1951	52	53	54	55	56	57	58	59	60
Yield, kilotons	112	104	252	--	167	?	343	38	--	--
Leukemia deaths	0	3	2	0	2	2	0	0	7	6

Year	1961	62	63	64	65	66	67	68	69
Yield, kilotons	--	101	<20	<20	<80	<60	20-200	<60	20-200
Leukemia deaths	2	1	1	0	1	1	2	1	1

*(70) Table 1

A-6 Leukemia Risk in Sensitive Subgroups.

In a 1977 paper Bross and Natarajan (72) claimed that 0.5 to 5 rem x-ray doses to the fetus damaged a sensitive subgroup of 1% and that the risk of leukemia in this group was enhanced by a factor of 50. Bross characterized this subgroup by a history of infections and allergies. This claim was based on an analysis of the Tri-State study of childhood leukemia by Graham et al. (73) which concluded that children dying with leukemia were more likely to have been exposed to x-rays in utero than children who did not develop leukemia. Oppenheim (74) has reviewed the Bross study and noted two serious criticisms which appear to invalidate the study.

a) The majority or the exposed fetuses received doses from scattered x-rays far below the 0.5 rem received by regions outside the material abdomen due to primary beam diagnostic exposure. Since the fatal x-ray doses in these cases were millirems rather than rems but the relative leukemia risk was similar for scattered and primary radiation to the fetus, it is likely that the childhood leukemia risk was not radiation-induced.

b) The incidence of leukemia in a control population of children unexposed in utero was biased by excluding children who had received post-natal irradiation. This procedure eliminated a fraction of the controls with a history of infections and allergies and increased the association between in utero irradiation, sensitivity to these "indicator" diseases, and childhood leukemia.

In a later paper Bross et al (75) claimed that diagnostic x-ray skin doses between 0.1 and 10 rems increased the leukemia risk by a 10-fold factor beyond the generally accepted leukemogenic radiation risk, and that the excess risk was associated with x-ray induced heart disease. These

conclusions were drawn from an earlier Tri-State study (76) on the frequency of prior x-ray examination in persons who had developed adult leukemia. This earlier study failed to find an association between leukemia and prior diagnostic x-ray dose and leukemia in females. The apparent association between leukemia and prior x-ray dose in male subjects may be an artifact due to an excess of infections during the 5-year pre-leukemic phase as noted by Stewart (77). In this regard Boice and Land in a critique of the Bross study published simultaneously in the same journal (78) noted that one-half of the x-rays preceding the onset of leukemia occurred in the 5 years prior to diagnosis. They also note that the earlier Tri-State study was compromised by potential biases In the discovery of prior x-ray exposures of the leukemia cases and of the control (non-leukemia) cases, which was not conducted on a "blind" basis. In addition, the reanalysis of the Tri-State study was limited to an unexplained 60% of the leukemia cases. Moreover, the data do not show an increasing association between heart disease and leukemia as the radiation dose increases, which makes it unlikely that radiation is an etiological factor in the development of heart disease; indeed, some heart disease could be a normal complication of the leukemic state.

It is concluded that the two studies of gross et al., in light of the above serious criticisms, do not justify the claims that leukemia risks from low level radiation are much greater than those generally accepted.

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SECTION 2

COMMON QUESTIONS ON RADIATION

A. Physical Facts

A.1 After being x-rayed for a diagnostic examination, how much radiation stays in my body?

Ans. No x-rays remain in your body. The x-rays are gone as soon as the x-ray machine shuts off -- in the same way that the light from a light bulb vanishes when the switch is turned off. X-rays must not be confused with radioactivity, where the radiation slowly decreases with time.

A.2 What is the difference between neutron and gamma radiation? Do low doses have different effects?

Ans. A neutron is a basic particle of matter but a gamma ray is a small packet of energy similar in some ways to a radiowave. The biological effects are similar and due to the ionization and chemical changes produced. The differences between the effects are in degree, not in kind. The same amount of energy deposited by neutrons and gamma rays while passing through the body will have a greater effect in the case of neutrons.

A.3 What are the main sources of radiation to which the public is exposed?

Ans. We assume in our answer that the question refers to ionizing radiation and thus excludes radio waves and light. The largest source for the average person is background radiation which accounts for approximately 43% of the exposure of the average person in the U.S.A. Background radiation consists of comic rays that reach us from the sun and outer space, radiation from radioactive materials which are always present naturally in the earth and buildings around us and radiation from radioactive materials naturally present in the body, particularly from a radioactive form of potassium, which is present in all potassium. Close behind is the radiation from diagnostic x-ray examinations, which accounts for about 40% of the radiation to which the average person is exposed. Another 7% is contributed by medical procedures involving the use of radioactive materials to diagnose illness such as heart disease and cancer. All other sources of radiation, such as radioactivity In consumer products, radioactive fall-out from past nuclear explosions, nuclear power. and occupational exposure, contribute only 10% to the exposure of an average person.

A.4 Is there only one kind of radiation which exposes the public?

Ans. So far as ionizing radiation is concerned, exposure is mainly from three types of radiation which have essentially the same effect for each unit of dose; these are x-rays and gamma rays, which are the same radiation (differing only in their origin) and beta rays which are electrons emitted by radioactive materials. There is also some exposure, particularly to the lungs, from a normally occurring radioactive gas called radon which emanates from the ground and is breathed by all of us.

The radon gas emits alpha particles, relatively massive and slow-moving compared to electrons, which have little penetrating power but will affect the surface of the lungs when inhaled. Radon levels vary with the type of soil and building material, and are higher near granite rock areas. The public is also exposed to neutrons which are produced in the atmosphere by cosmic rays, but the neutron dose is only a small fraction of the background dose.

A.5 What is the difference between low level and high level radiation?

Ans. The difference lies in the amount of energy deposited In the body by the radiation. This is signified by the radiation dose received as measured in rems. Low-level radiation generally means doses less than about 20 rems received by parts or all of the body either in a short time or spread over many years, as in the case of occupational exposure or background radiation. An example of the use of high level radiation in man is the treatment of cancer. In a course of radiation therapy, a small volume of tissue receives a dose of thousands of rems. The terms low level and high level are also applied to radioactive waste. Low level waste is mainly generated in medicine and industry and contains relatively small amounts of radioactivity which can be handled by people with simple precautions such as the use of rubber gloves, handling tools, containment vessels and light shielding in some instances. It can be disposed of by storage for decay, incineration. shallow land burial or, if liquid and not toxic, via the sewerage system. High level waste is generated mainly by nuclear reactors (for example, spent reactor fuel) and may contain radioactivity greater by factors of millions (even billions) than low

level waste. It is usually handled by remote control behind massive radiation shields. It can be disposed of by long-term storage, including deep burial in sealed containers in suitable geologic formations.

A.6 How far should I sit from my color television set to avoid harmful x-rays?

Ans. The answer depends on the level of radiation which you would consider harmful. The great majority of persons would not consider background radiation or moving to a part of the country with higher background to be harmful. All television sets must meet a federal standard of acceptable x-ray emission, 0.5 mrem per hour at 5 cm from the surface of the set. If you watched the set at a distance of 5 feet or more for 40 hours a week, the radiation exposure would be less than .03 mrem per week. which is 1-2% of normal background level in the U.S.A.

A.7 I read that besides natural background, most of the radiation exposure to people tunes from medical x-ray examinations. Do all medical x-ray examinations give about the same dose?

Ans. The dose from diagnostic x-ray examinations varies greatly and depends mainly on the thickness of the body section traversed by the x-ray beam and the number of films in the study. The very common chest x-ray examination requires a relatively small skin dose of about 30 millirems (0.03 rem) due to the ease with which x-rays penetrate the lung. Extensive studies in the pelvis can lead to doses of several rems. If there is a medical need for an x-ray examination, it should be carried out since the modest risk from the radiation dose is usually far outweighed by

the benefit of the information gained.

A.8 How can I tell how much radiation I have received even if I didn't wear a film badge?

Ans. A rough estimate of exposure can be made if the physical factors controlling the exposure are known: time, distance, primary beam intensity. field size, source activity and type. It is possible to deduce the dose received, if relatively high, by assaying the frequency of chromosome aberrations In circulating lymphocytes. This technique is not useful in estimating an unknown dose in the average radiation worker because of the residual effects of previous exposures. It is also expensive.

A.9 How accurate are the exposures reported by the use of film badges and how much exposure can one receive before it is readable on the badge?

Ans. The use of film badges provides a simple method of measuring personnel exposure for radiation workers. The accuracy of the reading Is typically 20%. the error increasing as the dose level falls. Because of its limited area the film badge provides only a sample of exposure of an individual. The minimum dose readable varies with the radiation energy but is generally of the order of 10 mrem.

A.10 I read articles in the newspapers about the dangers of radioactive waste generated by nuclear power reactors. In my hospital, I often see waste containers with labels indicating that radioactive waste is present. How

safe is it to work in their vicinity?

Ans. Nuclear power reactors generate two types of radioactive waste. One type is called high-level waste and consists of used reactor fuel elements which are very intense sources of radiation. This spent fuel is generally stored on the reactor site or shipped to a few specially designed storage areas, The other type, low-level waste is generated by nuclear reactor operations and also by hospital operations. It is composed of contaminated gloves and clothing, glassware, tools, paper towels, etc. This waste is collected and isolated in a restricted area under the supervision of a Radiation Safety Officer. The radiation level near low-level waste containers is generally low by definition, and presents no danger to people working in the vicinity. Some of the radiations, e.g., beta rays from radioactive hydrogen. carbon-14, sulfur-35 and calcium-45, emit beta rays which are completely absorbed by the waste containers. However, if the contents of the waste receptacle were to be strewn around the laboratory or storage area, there would be a contamination problem which should be brought to the attention of the Radiation Safety Officer.

B. Radiation Hazards: General

B.1 Isn't it true that exposure to even a small amount of radiation is dangerous?

Ans. The risks associated with low doses (of the order of l-10 rems). are not precisely known. Based upon the increased number of cancer deaths observed at doses of the order of 100 rems a lifetime risk per rem of about 2 in 10,000 for fatal induced cancers has been estimated. This, when compared to the spontaneous level of 2000 fatal cancers per 10,000 population, is a very small increased risk. In fact, it is likely that this is an overestimate of the radiation risk. The word "dangerous" to describe low-level radiation greatly exaggerates this miniscule risk

B.2 I have gotten the impression that little is known about the effects of radiation, other than that it is very dangerous. Is this true?

Ans. This is untrue. We probably know more about radiation than any other cancer-producing agent (carcinogen), physical or chemical. Experience goes back about 80 years and the information is probably better documented than that for any other carcinogen. Radiation is, in fact, a relatively weak carcinogen and mutagen.

8.3 It is sometimes suggested that if a person is exposed to radiation he/she will develop cancer or at the least is likely to. Is this true?

Ans. This is untrue. Radiation is not very effective in causing cancer

and even in large populations exposed to high doses of radiation the increase over the normal cancer rate is small. For instance, follow-up studies (through 1978) of 82,000 exposed Japanese A-bomb survivors have produced an estimated 250 radiation-induced cancers.

B-4 Can the radiation exposure associated with a diagnostic radiologic procedure be more dangerous than the associated illness?

Ans. This is almost always untrue. For individual patients undergoing radiologic procedures related to illness, the benefits of the procedure far outweigh the risks.

B.5 I have had several x-ray exams over the past couple of years. Will this sterilize me?

Ans. No. The radiation dose required to sterilize (500-600 rems) is at least a factor of 100 larger than the gonadal radiation exposure from "several" x-ray exams, even if the exams art directly of the gonadal area. If the x-ray exams are directed at other regions, the radiation exposure to the gonads would be even less.

B.6 Is it really true that low doses of radiation art more damaging than higher doses of radiation per unit of dose?

Ans. Not so. There is very little evidence for this assertion. There is considerable evidence that the effect of radiation is either constant per unit of dose or, particularly with x-rays and gamma rays, that the effect per unit dose is smaller at low doses. Studies which claim greater

effects at low doses art discussed in detail in the 1980 BEIR Report of the U.S. National Academy of Sciences and in Appendix A of this primer, and are discounted. For very large doses such as several hundred rems which kill a substantial fraction of cells in an organ, there is a reduced incidence of late cancer and therefore a smaller risk per rem.

B.7 What happens to you when you work with x-rays?

Ans. With the very small amounts of radiation received by persons working with x-rays and with the small amounts delivered to patients in typical diagnostic x-ray or nuclear medicine examinations, we expect nothing to happen to patients or personnel. Studies of persons occupationally exposed to x-rays or gamma rays for the past 30 years have not shown an increase in cancer.

B.8 What is considered to be the minimum lethal dose of radiation for humans?

Ans. The mean lethal dose (L.D. 50/30). or the dose that would be lethal to 50% of the human population within 30 days after irradiation, is approximately 350 rems of x-rays or gamma rays given in a single exposure to the whole body. The minimum lethal dose which may cause death in a few percent of exposed persons is probably about 250 rems. The mean lethal dose will be considerably higher if only part of the body is irradiated or if the irradiation is spread out over a longer tine period, such as 1 week. The administration of special medical care, including infection control and bone marrow transplants, will also increase the LD 50/30.

B.9 Just how much radiation will cause injury to my body?

Ans. Obvious injury such as skin burn, loss of heir and cataracts may be produced after doses of several hundred rems have been received in a short time such as one day. At low doses such as a few rems the only possible injury is long-delayed cancer, but the possibility is very small. Depending on the assumptions made, the risk has been estimated by expert committees as about 2 in 10,000 persons over a lifetime after receiving 1 rem of radiation dose to the entire body.

B.10 How much danger, if any, are x-ray technicians in, if they work with radiation for their entire lifetime?

Ans. The "danger" to which an x-ray technician is exposed, resulting from contact with radiation during a working lifetime, is very small. If the typical radiation exposure of about 0.5 rem per year is received, no observable effects are expected.

B.11 Is it true that there is no dose below which there is no damage to cells?

Ans. This is not clearly known but may be true. Even very small amounts of ionizing radiation can produce damage at the sub-microscopic level in some of the exposed cells. Whether this damage affects the properties of the cell, such as its ability to divide or its transformation to a malignant form, depends on many factors including where the damage occurs, the extent of the damage, and the health of the cell. It is clear, however, that the malignant transformation of a cell is extremely unlikely at low doses.

B.12 What art the delayed effects on people of low and moderate doses of x-rays?

Ans. In the great majority of persons there is no evident effect, immediate or delayed. However, there is a very small chance that cancer may develop after a delay of several or many years, and that genetic effects may appear in future generations.

B.13 Thirty years ago I was assigned for two years to nursing care of women receiving radium treatment. I often used to sit with the patients. Ever since then I have had accute infections and periodic spells of nausea and stomach craps. I believe this is the result of my radiation exposure. Do you agree?

Ans. There is no basis for believing that persistent problems of infection, nausea and abdominal pain are the result of low-level radiation exposure. Even if a nurse at that time spent as much as 10 hours per week about 3 feet away from a radium patient, she would receive no more than 20 rem per year. While this is more than the maximum permissible occupational exposure by today's standards, it would have no immediate medical effect on the nurse. It is true that a much higher dose such as 100 rem received in a period of a few days could conceivably produce early nausea and vomiting in some people during the ensuing week but the dose you speak of would have been too small and spread over too long a period to produce this effect. In any case it would not be a chronic continuing effect. Doses of 20 rem per year for 2 years would also have no observable effect on the white blood cells (or other blood fractions) and would not account for a series of acute infections.

B.14 What are the radiation doses required to produce either temporary or permanent sterility in men and In fertile young women?

Ans. In the male the dose to the testes in a single exposure required to cause permanent sterility is greater than 400 rem (lethal if applied to the entire body). Non-lethal but large doses, such as 100 rem, may produce an impairment of fertility dependent on the dose. Fertility falls gradually after exposure since sperm will continue to be formed temporarily through maturation of surviving spermatocytes and spermatids which are relatively radioresistant. The maximum reduction in fertility occurs about 10 weeks after the exposure and recovery may take as long as 2 or 3 years.

In fertile young women. the single dose to the ovaries to cause permanent sterility is in the 500 to 1000 rem range (again lethal if delivered to the whole body). Doses of the order 100 to 400 rem may cause an impairment of fertllity, since the ovary contains its entire supply of oocytes early in life and cannot replace oocytes lost due to radiation damage or other causes.

B.15 How much radiation is considered an acceptable level of exposure? How were these levels arrived at?

Ans. The acceptable level depends on who you are, where you are, your age, and a judgement on whether that level can readily be reduced. There are maximum permissible exposure levels which have been promulgated by the International Commission on Radiological Protection and in the USA by the National Council on Radiation Protection and Measurements. These

recommended levels have been endorsed by various federal agencies such as the Nuclear Regulatory Commission, by State governments and by numerous employers. There are dose limits for the whole body and for specific organs of the body. The limits are different for persons who are occupationally exposed and persons who are not occupationally exposed but are in the vicinity of radiation sources; for the whole body these limits are 5 rem per year and 0.5 rem per year, respectively. The lower limit additionally applies to persons younger than 18 and the fetus in pregnant women.

Very few occupationally exposed persons receive the 5 rem limit per year. The national average for occupationally exposed persons is less than 0.5 rem per year. All such persons carry a radiation monitor. It is a requirement of the Nuclear Regulatory Commission that radiation doses received occupationally must be reviewed periodically by radiation safety personnel and that efforts be made to maintain such doses at levels which are "as low as reasonably achievable."

These maximum levels were arrived at by considering the combined cancer and genetic risk from radiation and choosing a dose level such that radiation work would carry a risk comparable with the less hazardous occupations in our society. The general public limit has been set somewhat arbitrarily at 10 times less than the occupational limit with an average to the whole population not to exceed 170 millirem per year which is somewhat more than an additional background dose.

C. Genetic Effects

C.1 Non-disjunction -- the failure of chromosomes to segregate during meiosis in the gonads of both males and females -- can have tragic consequences. It is responsible. for example, for Down's syndrome and for a great deal of fetal wastage -- about half of spontaneous abortions. Why do many discussions of genetic effects of radiation ignore this important effect

Ans. The relationship to radiation; is controversial. Laboratory studies make it clear that radiation increases non-disjunction, and some suggest that the increase is greater in older mothers than in younger ones. However, although some epidemiological studies show an increased risk following relatively small doses of radiation, others have failed to do so. Part of the reason is simply that the spontaneous, rates are high, and also dependent on parental age so that it is difficult to see the additional cases due to radiation in the human populations studied. Nevertheless, the preponderance of evidence supports the proposition that reducing exposure of the gonads in young people and children to as low a level as feasible is good policy, even if it reduces only marginally the risk of non-disjunction.

C.2 The chromosomes are the principal target of radiation in all cells. Why do we consider somatic effects and genetic effects differently?

Ans. Chromosome aberrations in somatic cells, for example deletions and translocations, can activate oncogenes and produce cancer. Similar damage in germ cells (those involved in sexual reproduction) can lead to fetal

wastage, or to serious birth defects. Thus the consequences of chromosome damage are very different, even if the basic damage is the same, when it occur.3 in germ cells or somatic cells. Similarly, the consequences are very different when the chromosomes are damaged in the fetus, and lead, say, to the death of cells important for organ formation, than if they are damaged in the adult.

C-3 The doubling dose at high radiation dose rates for gene mutations in humans has been estimated from the atomic bomb data as about 156 rads. Gonad doses associated with diagnostic procedures are usually in the millirad range or less. Why should we be concerned with genetic risks of radiation from medical examinations;?

Ans. All the data we have from radiation biology and radiation chemistry points to' the same conclusion -- there is no threshhold dose 'for radiation-induccd mutations. thus even very low radiation levels could result in an increased number of mutations, although with a very low probability -- but these might have consequences over many generations.

C.4 Certainly there are good as well as bad mutations. Wouldn't our miserable species be improved by irradiating it and inducing some good mutations (less aggression, slower aging, for example)?

Ans. All mutations that are compatible with life are already present many times over in the human population of some 5 billion people. We can leave it to natural selection to increase their frequency. Or we can leave it to the genetic engineers. Mutations produced by radiation or occurring

"spontaneously" are overwhelmingly deleterious. We need to reduce. not increase, our load of mutations.

C-5 In the purely statistical sense of years of life lost, birth defects are far more important than cancer. It is estimated that 80% of birth defects are due to defective genes inherited from one or both parents. Why do committees charged with setting radiation standards pay attention, apparently, only to carcinogenic rather than to mutagenic effects of radiation?

Ans. In fact, standard-setting bodies such as the International Commission on Radiation Protection do consider both types of effect, but the risk of cancer induction is thought to be higher. However, in 1957 the maximum permissible radiation dose to persons occupationally exposed was reduced from 15 rems per year to 5 rems per year mainly because of concern over mutagenic affects. In the interim, concern over the carcinogenic effects has increased. By contrast, the full effects of a given mutation are in essence incalculable - they range from small effects spread over generations to lethal effects expressed during fetal development. Moreover, there is no agreed upon method to compare the impact of different genetic defects -- is a spontaneous abortion preferable to a retarded child? to a son with hemophilia? These are unanswerable questions.

C.6 How much radiation will cause genetic damage?

Ans. Theoretically one single photon or particle of radiation could cause genetic damage but the chance that it would is inconceivably small.

Remember that normal background radiation bombards us with about 1 million photons each minute but increased genetic damage has not been found in persons living in regions where twice or even more background radiation exists. In fact a significant increase in genetic damage has not been found In the children of the Japanese A-bomb survivors who received an average radiation dose of about 20 rem.

C.7 If radiation causes a genetic effect in the next generation, how many future generations will be affected?

Ans. Genetic effects can be transferred through many generations. Some mutations will die out because they are less likely to be inherited or confer a lower fitness on affected people.

C.8 I work as an x-ray technician. I plan to start a family in the next couple of years. What is the likelihood that I will have "damaged" children?

Ans. Occupational exposure is maintained at values less than the maximum permitted dose (MPD). Exposure to such low protracted doses would have a "negligible probability of genetic injury" according to MCRP Report 39. In fact, no significant genetic effect has been observed in studies involving the 74,000 children born to Japanese A-bomb survivors between 1948 and 1962, nor in a 1961 study extended to a 34-year period.

Animal experiments, mainly with large colonies of mice, have suggested two reasons why females who have been exposed to radiation over a long period of time are less likely to contribute genetic defects to

their offspring than are males receiving similar radiation exposures. first reason is that irradiated female mice produce a decreasing number offspring showing mutations as the radiation dose rate is reduced, and a dose rate of 500 millirems/hour the mutation frequency does not exceed that found with non-irradiated mice. The second reason is that offspring conceived after a 7-week delay following a single dose of radiation to mother also show no increase In mutation frequency.

D. Fetal Effects

D.1 How does radiation affect the unborn fetus?

Ans. The possible effects are dependent upon both the dose and the time of exposure during gestation. Large doses. over 25 rem. during the first 12 days of pregnancy can increase the spontaneous abortion rate, usually without the woman being aware of the pregnancy. Large doses to the embryo between the second and eighth week of gestation can increase the incidence of structural malformations. Thereafter, the effect, if any, may be growth retardation and possible mental retardation. Childhood leukemia and solid tumors have been imputed to low-level irradiation in utero, but there is considerable doubt whether radiation was the causative factor. Small doses of radiation delivered during gestation have a very low probability (such as one chance in a thousand per rem of fetal dose) of **Causing any** detectable effect,

D.2 When should a pregnant x-ray technologist stop working as a tech.? I have heard that she should not work during her first 5 months of pregnancy and then work 4 months until delivery.

Ans. The National Council on Radiation Protection and Measurements (NCRP) has established a guideline that the fetal dose from its mother's occupational exposure should not exceed 0.5 rem for the total gestation period. Host x-ray technologists receive less than 0.5 rem to the surface of the abdomen in a 9 month period and even less to a fetus. Hence if the technologist follows good radiation safety practices established for the

profession, she need have no fear that her unborn child will receive a dose exceeding the NCRP limit and there is no reason for the technologis to stop working as far as the radiation risk is concerned, unless her usual dose is at least 2 rem per year. which is most unlikely. There are some jobs, however, which should be avoided if possible, such as angiography and fluoroscopy. in order to keep the radiation dose as low as reasonably achievable.

D.3 How much radiation could a pregnant nurse receive without hurting the fetus at all?

Ans. A recent estimate of total radiation risk to the embryo/fetus by a United Nations committee is one chance in 1000 of any harmful effect from the allowed dose of 0.5 rem. In 999 babies out of 1000 there would be no harm. The risk, if any, from 0.5 rem would not be observable since it is far below the normal risk of 60 per 1000 that a child will show a birth defect.

D.4 What is the chance my baby could be mentally defective as a result of recent x-ray examination of my spine when I was six months pregnant?

Ans. Practically none. The dose to your unborn child from the x-ray exam of the spine was probably less than 1 rem. It is known that large fetal radiation doses delivered during the second half of a pregnancy Can produce mental retardation, but there is no evidence that this has occurred at doses below about 10 rems.

D-5 My wife recently had an x-ray examination of her lower back. Is it OK for her to become pregnant?

ANS. The chance of any genetic defect appearing in any future children is essentially nil. In spite of this, she could delay pregnancy for a few months to reduce any remote chance even further.

D.6 I had dental x-rays during my first trimester of my pregnancy and I was not given a lead apron to wear. What are the possible dangers to my baby?

Ans. Even without a lead apron, the dose to the fetus from a series of dental x-rays would be no more than one thousandth of a rem, much too small to have any observable effect on the baby.

D.7 I had an IVP during my first trimester before I knew I was pregnant. What are the dangers to my baby?

Ans. Probably none, especially if the examination was done during the first 12 days of pregnancy. The dose to the fetus from an IVP probably was less than 1 rem. Even if the dose were several rems and if the exposure occurred during the most sensitive stage of the pregnancy, the risk of radiation injury to the baby would be no more than 1 in 100 and no detectable harm would be expected.

D-8 I live very close to a commercial nuclear power plant. If I become pregnant would my fetus be in danger due to radiation exposure?

Ans. No. The amount of radiation permitted in the surrounding environment from the operation of a nuclear power plant is only about 5% of the natural background radiation and is much less than the variation background radiation in different areas or inside different buildings in the same area. Even during the accident at "Three Mile Island", the dose to a fetus in the vicinity of the plant was negligible.

D.9 My husband had, about 20 x-rays of his prostate including his testicles before I became pregnant. What are the chances that the x-rays could have damaged his sperm and that our baby will be malformed?

Ans. Your husband probably had several IVP studies in which case the dose to the testicles probably was less than 1 rem. The chance of a significant genetic disorder in a child of the first generation from a gonadal dose of 1 rem to the father is estimated to be between 5 and 75 in a million. This compares with the natural chance of one hundred thousand per million. Hence the risk of a serious genetic disorder occurring in your baby from non-radiation causes is probably several thousand times greater than the risk from the radiation.

D.10 During my first trimester I was exposed to x-rays indirectly while holding my son who received about 10 x-rays following an auto accident, I was not wearing a lead apron. Is there a possibility that my unborn baby could have been harmed at that time?

Ans. If the x-ray beam was properly collimated, it is very unlikely that your body intercepted the primary beam with the possible exception of your hands, In that case, your abdomen probably received less than 0.01 rem and the fetus probably received less than 0.002 rem. Therefore, the chance of a radiation-induced injury to your baby is on the order of one in a million, or essentially zero, In the unlikely event that your abdomen was struck by the primary beam for all ten x-ray exposures, as an upper limit the fetal dose might have been one-thousand times greater. Even then the risk of injury to your child from the radiation exposure would have been very small, and much less than the normal risks not due to radiation.

D.ll How much protection is givcn to my baby if I wore a lead apron while pregnant and receiving a chest x-ray?

Ans. If the x-ray beam were properly collimated so that it was confined to the area of the chest, the exposure to the fetus would be very small. probably less than 0.0001 rem. even if a protective lead apron was not worn over the abdomen. Wearing a lead apron in this case might reduce the dose by another factor of 2. If the primary beam was much larger than good practice would permit and the abdomen was Struck by the primary beam, the dose to the fetus would be higher, for example 0.01 rem. In this situation shielding the abdomen with a lead apron would reduce the dose to the fetus by a factor of about 20 to roughly 0.0005 rem. However. even without the apron the risk of radiation-induced injury to the fetus would be small, such as 1 chance in 100,000.

D-12 I work In a laboratory where certain chromatographic units contain radioactive sources. What are the chances that my child has been harmed while I was pregnant?

Ans. Most chromatographic units use radioactive sources which do not emit any penetrating X or gamma rays so that there is normally no detectable radiation in adjacent working areas. Hence as long as the units are not misused, there is essentially no radiation hazard to the mother or the baby she might be carrying.

D-13 I work In a laboratory using tritium, carbon-14 and phosphorus-32 in amounts up to 10 millicuries. What is the risk to my baby now that I am pregnant?

Ans. Essentially none. If proper precautions art taken to avoid or minimize any inhalation or ingestion of these radioactive materials (which do not emit any penetratlng gamma radiation). there is essentially no risk to the fetus.

D-14 I was in an accident and received several x-rays to my head, chest and abdomen not realizing that I was in my first month of pregnancy. What art the chances that my baby will be malformed?

Ans. The chances are that you received less than 1 rem to the uterus from the x-ray examinations, Even if this exposure occurred during the most critical period of fetal development (weeks 3 to 8 of pregnancy), the chance of injury to the baby is on the order of one in a thousand. The

"natural" risk of a congenital defect is about forty in one thousand. Therefore. at worst. there might be a 3% increase (i.e.. from 40 to 41) in the possibility of a congenital defect. If the exposure occurred during the first twelvc days of pregnancy, before organogenesis started, than there is essentially no risk of malformations in the baby.

D.15 A patient of mine received a barium enema examination and later discovered that she had been 3 weeks pregnant. How much dose do you estimate the embryo received, and how should I advise the patient?

Ans. The dose to the uterus from a typical barium enema examination is less than 1 rem. However. since the exposure occurred during the third week of pregnancy, after the beginning of organogenesis, there is a small increased risk of the radiation causing a structural malformation. The increased risk is believed to be on the order of one chance in a thousand. Abortion is not recommended. At a dose level of 1 rem to the embryo, one would have to abort hundreds of "normal" pregnancies to prevent the birth of one child with radiation induced abnormality. The patient should be advised of these estimated risks and she should be encouraged to continue with the pregnancy unless there are other extenuating circumstances that would dictate otherwise. In any case, a summary of the conversation with the patient and with her decision In the matter should be entered into the patient's record. It should be noted that both the American College of Radiology and the American College of Obstetrics and Gynecology have adopted a policy that rarely if ever is termination of pregnancy advisable because of the radiation risk arising from diagnostic x-ray examinations.

E. Induction of Cancer

E.1 Are my chances for developing cancer in later life as an x-ray technologist greater than those of a person who has nothing to do with radiation?

Ans. There are no studies at the present time which show that an x-ray technologist's chances for developing cancer later in life are greater than those of persons not occupationally exposed. For example, one study comparing 6000 U.S. x-ray technologists with 6000 laboratory technologists all trained during World War II showed no significant difference in cancer mortality between the two groups. Also a 1981 study on British radiologists who started practice after 1920 and who died before 1977 shows a significantly lower death rate from cancer compared with other professionally employed persons and no significant difference compared with other medical practitioners. If it is assumed that there is no threshold dose of radiation for cancer induction and that risk increases with the total dose received, then a very small increase in cancer incidence cannot be ruled out.

E.2 Can radiation from a routine x-ray or any radiologic exam cause cancer?

Ans. It is theoretically possible that the radiation dose received during a routine radiological examination could increase the risk of developing cancer. The theoretical risk incurred is certainly extremely small but not precisely known. To date all studies of populations who have received small doses of radiation similar to those received in routine radiologic

examinations have either failed to show an increase in cancer clearly attributable to the radiation. or are the subject of much controversy.

E-3 Over the past 15 years, starting when I was 16. I have had three upper G.I. series for suspected stomach ulcers. How long do you think it will be before I get stomach cancer?

Ans. The short answer is that you will not get stomach cancer as a result of the radiation. The x-ray dose to the stomach from a typical upper G.I. series is typlcally about 1.5 rams from a combination of flouroscopy and radiography, or 4.5 rems from the three examinations. Assuming the cancer risk is proportlonal to dose and using the risk estimates for stomach cancer derived by the National Academy of Sciences in 1980, the chance that stomach cancer due to the radiation will occur much later in life is estimated at about 1 in 5000. In other words 4999 out of every 5000 persons would not develop stomach cancer from the radiation. On the other hand the normal chance of getting stomach cancer is 1 in 100. This means that even if stomach cancer did develop it would almost certainly not be due to the x-rays.

E.4 My personal physician recently recommended that since I am 55 and there is breast cancer In my family, I should have a baseline mammogram with repeat examinations every two years. What is the risk that these exams will themselves produce cancer?

Ans. Today about 1 woman in 14 can expect to develop breast cancer at some the during her life. The likelihood is clearly greater in high risk families - say 1 in 10 at your age. There is considerable evidence that radiation to the breast carries a very low risk of subsequent breast cancer, which appears more than 10 years after the exposure. The dose to the breast tissue is usually between 0.2 and 1.0 rem per examination depending on the x-ray system used. If you started these examinations at age 55 and continued them for the next 15 years, once every 2 years, the estimated breast cancer risk is about 1 chance is 15000 with the lower dose and 1 chance in 3000 with the higher dose. Even with the higher dose. the risk that the series of x-rays would produce a breast cancer is about 300 times smaller from the x-rays than your normal risk. and 1500 times smaller if the low-dose system is used.

E.5 Do you think it was John Wayne's smoking (5 packs per day) or fallout from A-bomb tests while making a movie in Nevada which was responsible for his lung cancer?

Ans. The estimated death rate from lung cancer in white males in the age group 50-70 years who are heavy smokers (more than 2 packs per day), has been reported as about 2000 per million per year. The radiation dose to the lungs received by a person subjected to radiation in a fallout area subsequent to a nuclear weapons test during the 1950's Is a matter of speculation but would be unlikely to exceed 10 rem. This dose is estimated to produce a lung cancer risk after a 10 year lapse of time of

about 50 per million par year. It would, therefore, appear to be a virtual certainty (40 to 1 odds and probably greater) that John Wayne's lung cancer was the result of his long established smoking habit.

E.6 How long after exposure does It take for cancer to appear?

Ans. The best short answer is that in nearly everybody who has been exposed, cancer from the radiation will never appear. For example it is estimated that in 10.000 persons whose whole body has been exposed to penetrating x-rays or gamma rays with the low dose of 1 rem only 2 persons will eventually develop cancer. With the large dose of 100 rem which could only be received in a serious accident, only 2 persons in 100 exposed will develop cancer.

If a person does develop cancer from the exposure. it may occur 10 years later but more usually 20 or maybe 30 years later. The exception to this is leukemia which could occur as early as 3 years after exposure but not later than about 30 years after exposure.

E.7 What has been the Cancer experience of Japanese survivors of the atomic bombing of Hiroshima and Nagasaki?

Ans. The health of these persons has been followed from 1950 to 1978 and it is clear that the principal observed deleterious effect has been a small excess of cancer cases over the number normally expected during that period. Among the 80,000 survivors studied over this period 4750 cancer deaths have occurred instead of 4500 expected, an excess of 250. In other words 33 years after the bombing about 1 survivor out of every 300 has

died with a cancer attributed to the radiation dose, estimated on the average about 14 rems to the whole body. There has been no increase observed in other types of disease and no significant genetic effect on the health of the children of the survivors, including no increase in cancer among children who were exposed during the fetal stage.

E.8 Have there been any experiments with rats or other animals that have told us that certain radiation levels may be safe?

Ans. Yes. Some recent experiments with large groups of mice have shown no increase In the cancer rate after 10 rems or even greater doses. However, the possibility cannot be ruled out that there is a real but imperceptible increase in risk per rem at these low doses, much smaller that is evident from experiments at higher doses. For a few kinds of cancer there appears to be a decrease in the amount of cancer after moderate doses of radiation, e.g., 25 rems. Many experiments have been performed on a variety of animal species to determine whether bone cancer develops after intakes of radioactive elements which concentrate in the skeleton -- such as strontium-90, an important element in radioactive fallout from atomic weapons. In this special case the data is consistent with a threshold dose level of several hundred rems below which cancer does not occur: or at least occurs with a much lower level of risk than for other cancers after doses of this size.

E.9 Cancer induction may be related to suppression of the immune responsiveness of the body. What is the minimum radiation dose expected to have an effect on immune responsiveness and what dose will result in a 90% suppression?

Ans. Whole body radiation exposure in doses over 100 rem may be expected to have a significant effect on immune responsiveness. The dose necessary to cause a 90% suppression depends on species but is sublethal. For man the dose lies in the 200 to 400 rem range.

E.10 Is it true that the child in gestation is more sensitive to Cancer induction from radiation than an adult person?

Ans. There is conflicting evidence on this question. Some studies have shown an association between abdominal irradiation of the pregnant mother at low doses and subsequent cancer development in the child with an estimate that the fetus is 5 times more sensitive that the adult. Some human studies and several animal studies do not support this conclusion and suggest that the fetus and adult are at about equal risk for cancer development after a given dose.

E-11 I am 44 years old and about to have an x-ray exam of my esophagus because of repeated burping, and I am thinking of buying a new car. Do you think I should postpone buying the car until after the exam because I night get esophageal cancer from the x-rays?

Ans. Definitely no. In the first case the chance of induction of cancer by the low doses of x-rays given during the diagnostic examination is extremely small. The radiation dose to the esophagus if 4 x-ray films are taken is typically less than 1 rem, and if in addition fluoroscopy is used the dose will probably increase to no more than 2 rem. Such a dose is estimated to carry a risk of inducing cancer of the esophagus no greater

than 1 in a 100,000. a risk which is too small to measure. You should also realize that a cancer, if indeed it is induced at all, would not appear for at least 10 years after the x-ray exposure. So buy your new car now and don't give the possibility of cancer another thought.

E-12 A month ago after my 2-month old baby fell on his head, I took him to the local hospital. The baby was given a series of x-rays of the head which proved to be normal. Since then I have heard that x-rays may cause cancer in my child. I am very worried about this. Is it true? Should I have agreed to the x-rays?

Ans. Clearly you wanted to find out whether your child had suffered possible serious damage to the head, such as a skull or facial fracture The x-rays were taken to determine this and you should be reassured. It is true that there may be an extremely small risk of your child develop a cancer in the x-rayed part of the body many years into the future. The cancer risk from 4 or 5 x-ray films is far smaller than the normal risk that your child could develop cancer from other causes at same time during his life -- at least 10,000 times smaller. It is also about 1000 times less likely than the chance of your son being killed in an automobile accident at some future time. The x-rays produced helpful information you and your child with a risk far too small to worry about.

E.13 Does the radiation cancer risk increase as you receive more and more radiation exposures? Also is the cancer risk smaller when the exposure spread over several years?

Ans. Studies on groups of persons who have been exposed to moderate or high doses of x-rays or gamma rays show that the risk of developing cancer increases as the radiation dose increases. For this reason, limits of radiation dose have been set which keep the risk to an acceptable level. There is considerable controversy over whether the risk increases proportionally or more than proportionally to an increasing dose. Animal experiments have frequently shown that the smaller doses product less cancer per unit of dose than do higher doses. Some animal experiments have also shown that the delivery of a certain dose over a long period of time generally produces less cancer than the same dose delivered rapidly. such as in a few minutes or hours. This effect is usually attributed to partial repair (or healing) of the initial cellular damage before the later radiation is received. It stems likely, although also controversial at present, that the reduced effectiveness of slowly delivered radiation also applies to cancer induced in human populations.