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Authors
Patterson, H. Wade.
Thomas, Ralph H.

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H. Wade Patterson and Ralph H. Thomas

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H. Wade Patterson and Ralph H. Thomas

Lawrence Berkeley Laboratory
University of California
Berkeley, California

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"A likely impossibility is always preferable to
an unconvincing possibility"
Aristotle—from the "Poetics"

1. INTRODUCTION

We have seen evidence in the past several years of a growing concern on the part of the general public over the possible risks to which they may be subjected as a result of Man's increasing uses of ionizing radiations.

The specific benefits derived from the uses of ionizing radiations in medicine and industry may be a matter of particular debate, but it seems generally to be accepted that benefits do in fact accrue. Public concern is centered on what risk, if any, is involved in such activities. In the words of the International Commission on Radiological Protection (ICRP), "If the quantitative relationship between dose and the risk of an effect were known, societies or individuals could judge the degree of risk that would be acceptable, taking into account the particular circumstances requiring a radiation exposure. Ideally, such a judgment would involve a balancing of the benefits or necessities of the practice against the risks of the given exposure, which could also be related to that of other risks in the particular society." (1)

With respect to physical and chemical components in the natural environment other than radiation, it would seem that Man has, through evolutionary processes, been adapted to function adequately over a rather broad range of exposure. Examples of this are carbon dioxide concentration in air, temperature, and barometric pressure. Observing this, we might be tempted to posit that Man's response to radiation exposure would be similar. However, as scientists we must stress that we do not know the effect of small exposures to radiation on human beings. We do not know whether such exposures are deleterious, of no consequence, or beneficial.

It is perhaps true that more is known of Man's response to ionizing radiations than to any other self-inflicted pollutant of his environment. This is largely due to the experience of radiation injury resulting from early uses of x-rays and radioactive substances, particularly radium. From these early experiences and from studies on certain other groups of individuals subjected to high radiation exposures as a result of radiotherapy, nuclear weapons attack, or radiation accidents, a limited amount of information has been pieced together. Such information is almost entirely about the effects of large exposures and high dose-rates. If we are to make any progress in the difficult task of understanding the possible deleterious effects on the health of the population due to small exposures to ionizing radiation at low dose rates it is clear that much greater efforts at interdisciplinary studies are needed. Radiation physicists can measure human exposures to ionizing radiations, physicians can advise on the appropriate indices of health, and statisticians can show us how to analyze available data in the most fruitful manner. It also seems clear that any conclusions we may reach as to the probable risks to human beings of low doses of

radiation will almost certainly have been reached by statistical inference. Heretofore much of the analysis of radiation-risk data has been performed by non-professional statisticians, and we believe that much benefit would derive from a re-evaluation of the existing data by professional statisticians.

Although much of what we say here will be familiar to specialists in the fields of study involved, we do try to draw together what seems to us the relevant threads of the argument involved in setting up an epidemological study of this nature.

In this paper we first briefly review the source of the studies that have been made of radiation-induced injury for rather large acute exposures. These studies enable one to make some first-order approximations on the level of risk involved.

Next we summarize Man's natural radiation environment and show that the extreme variations in whole body exposures vary from about 100 mrem/y to an upper limit of a few rem/y. Man-made radiation levels are, with one exception, small compared even with the fluctuations in these natural levels due to geography and personal habits. The one exception will be shown to be due to medical radiology.

2. SIZE OF POPULATION NEEDED FOR AN EPIDEMIOLOGICAL STUDY OF RADIATION-INDUCED DISEASE

It seems to us that a most important preparatory step in designing a study to identify the risks of radiation exposure inducing disease is to determine the size of the group needed.

The following simple arguments indicate the size of the population needed to identify the magnitude of risk.

The total number of cases of the disease, N_0, observed in a population, p, over a period of y years is given by

$$N_0 = f\,p\,y \tag{1}$$

where f is the probability of contracting the disease per year.

Assume that this disease may also be induced by low levels of radiation exposure and further assume that at low doses the dose-effect relationship is linear. At equilibrium an annual dose rate of D rem/y will then produce an additional number of cases of the disease due to radiation, N_R, given by

$$N_R = r\,D\,p\,y \tag{2}$$

where r is the risk per year per rad.

The total number of cases of the disease actually observed, N_T, is then

$$N_T = (f + r\,D)\,p\,y \tag{3}$$

and we ask the question, when can we be sure that the difference, Δ, $\Delta = N_T - N_0$ is greater than zero?

$$\Delta = r\,D\,p\,y \pm \epsilon \tag{4}$$

where the error ϵ is given by

$$\epsilon^2 = p\,y\,(f + r\,D) + f\,p\,y. \tag{5}$$

To be sure of the magnitude of Δ we must demand that

$$\epsilon \leq r\,D\,p\,y.$$

Typically, r D p y will be small and this constraint may be difficult to meet. However, let us arbitrarily write

$$\epsilon \approx \frac{r\,D\,p\,y}{2},$$

from which it follows that

$$(7) \qquad p\,y \approx \frac{4}{r\,D}\left(1 + \frac{2f}{rD}\right).$$

This equation enables us to calculate the number of man-years (py) required to form the basis of a study to reveal radiation-induced disease.

As an example, the probability of death in the United States due to malignancies is about 1.5×10^{-3} per year,[2] and one may readily calculate the number of man-years (py) from Eq. (7) for several dose rates and degrees of radiation-induced risk. Table 1 summarizes such a calculation.

Table 1. Number of man-rem years needed for an epidemiological study of radiation-induced cancer.*

Dose rate (rem/y)	Radiation risk (deaths/y/rad)	Man-years
0.1	10^{-1}	5.2×10^{2}
0.1	10^{-2}	1.6×10^{4}
0.1	10^{-3}	1.2×10^{6}
0.1	10^{-4}	1.2×10^{8}
0.1	10^{-5}	1.2×10^{10}
1.0	10^{-1}	4.1×10^{1}
1.0	10^{-2}	5.2×10^{2}
1.0	10^{-3}	1.6×10^{4}
1.0	10^{-4}	1.2×10^{6}
1.0	10^{-5}	1.2×10^{8}

*Taking "normal" risk of death due to malignancies as 1.5×10^{-3} per year.

As Sailor has already discussed in this symposium[3] and we shall show later, it is possible to find differences in radiation exposure rates of substantial populstions of up to a few hundred mrem/y. In comparing the death rates due to cancer in groups where radiation exposures have changed with time, studies must extend over periods long compared with the latency of the disease. It would seem mandatory therefore to carry out such investigations over periods of something like 10-30 years, and there are those who would suggest even larger periods. If one takes the risk of cancer induction due to radiation as 10^{-4} per rad per year [a conservative upper limit if the interpretation of the pertinent data presented by the International Commission on Radiological Protection (ICRP) is accepted[4]], Table 1 indicates that populations in excess of 10 million people whose radiation exposures differed by 0.1 rem/y must be studied for extended periods.

There is no chance of finding such large populations within the United States whose environments are so similar and stable over such extended periods—differing only with respect to their radiation exposures. However, much smaller populations are needed to test the hypotheses that the risk of death from radiation—induced disease is much higher than suggested by ICRP.

Gofman et al.[5] have suggested that the increase in cancer mortality rates is as high as 2×10^{-2} per rem/y. (This is in fact roughly equivalent to assuming that all cancer mortality is due to radiation exposure, since the "natural" mortality cancer rate is 1.5×10^{-3} deaths per year and the average annual dose rate is about 0.13 rem/y).[3] One might think this to be an upper limit since chemical carcinogenesis might be suspected to contribute to the death toll.

At levels of risk as high as 10^{-2} per rad, studies with relatively small numbers of people (several hundred) should be capable of revealing significant differences between populations whose radiation exposures differ by a few rads (integrated dose).

One of the populations most frequently exposed to ionizing radiation is atomic energy workers. The USAEC makes annual reports of the exposures for such workers. Using data for 1960, Eisenbud[6] estimated a per capita dose of 0.6 rem to a population of 82 000 workers.

Table 2 summaries similar data for 1969.

Table 2. Estimated whole body doses to employees of AEC contractors, AEC licensees, and agreement state licensees for 1969.

Annual dose rem	Number of employees		
	AEC contractors	AEC licensees	state licensees
0 - 1	98 625	59 496	23 082
1 - 2	2 554	1 489	786
2 - 3	1 313	583	321
3 - 4	335	191	107
4 - 5	86	109	69
5 - 6	4	64	56
6 - 7	0	48	39
7 - 8	0	36	24
8 - 9	0	14	6
9 - 10	0	13	6
10 - 11	1	3	4
11 - 12	0	4	0
12+	0	22	19
Total	102 918	62 072	24 519

If we assume, with Eisenbud, that all members receive the mean dose of the dose grouping (probably an overestimate) we can conclude that within the atomic industry the accumulated dose for 1969 was about 110 000 man-rems (at an average per capita dose of 0.58 rem). Failure to find any significant increase in cancer risk in this population should

therefore be able to set the risk of cancer induction below about 10^{-3} per year per rad.

3. RADIATION AND RISK STUDIES—A BRIEF REVIEW

What has been established "beyond reasonable doubt" thus far?

Fortunately Man's experience of radiation-induced injury is nowadays quite infrequent. Nevertheless in the past 70 years a number of persons have been exposed to rather large doses of radiation, and the data obtained from epidemiological and cytogenic studies of them provide some measure of the incidence of radiation-induced diseases. In the main these persons fall into three main groups:

a) Medical patients undergoing radiotherapy—for example, ankylosing spondylitis patients treated by x-ray irradiation of the spine, radium-therapy and thorium-therapy patients, patients treated for hyperthyroidism, women treated for cervical cancer, or children irradiated for enlarged thymus and tinea capitis. A group of children exposed in utero for diagnostic purposes for the mother have also been studied.

b) Victims of nuclear warfare or testing, e.g., those exposed at Hiroshima, Nagasaki, and the Marshall Islands.[7]

c) Occupationally exposed persons, e.g., radium-dial painters, radiologists, and uranium miners.

From these three main groups the ankylosing patients, the Hiroshima and Nagasaki victims, and the radium-dial painters have been most extensively studied.

3.1. Hiroshima and Nagasaki Victims

Perhaps the most thorough and extensive study of the incidence of disease in human populations exposed to ionizing radiations has been performed (and is still in progress) for the victims of the nuclear weapons attacks on Hiroshima and Nagasaki in 1945.[8-10]

Within about 2 years from the exposure a significant increase in the incidence of leukemia was observed in the exposed population. Early studies showed the increased frequency of leukemia to be inversely related to distance from the hypocenter. This fact led Lewis[11] to suggest that the incidence of leukemia was linearly related to dose. However, subsequent analyses of the dosimetry have revealed some uncertainties that make such a conclusion uncertain. In his analysis Lewis utilized dose distance curves known by their originators to have substantial errors, but the best available at that time.[12]

Auxier et al.,[13] in a recent paper on dosimetry, have suggested the probable error in the air dose to be ±30% at Hiroshima and ±10% at Nagasaki. Problems of local shielding, spectral distribution, and relative proportions of neutron and γ dose make the assignment of individual doses a much more difficult problem. Moloney and Kastenbaum[14] made this distinction when they showed that for persons exposed at the same distance, the incidence of leukemia was higher in those who suffered radiation sickness in the few weeks immediately following the exposure. Milton and Shohoji[15] have reviewed the dose estimates due to Auxier et al. and those made by Hashizume et al.,[16] based on measurements of residual induced activity and thermoluminescence in irradiated material, and concluded that "it is not possible at present to give a quantitative evaluation of either the accuracy or precision of the final (individual dose) estimates."

Inability to assign doses to individuals required that morbidity and mortality data be lumped on the basis of distance. When this is done, even with a distance interval as small as 50 meters, the uncertainty in dose is as large as 30%. And, if the data are lumped in large intervals, as is done in ICRP Publication 8,[17] the dose uncertainty approaches two orders of magnitude. These considerations lead one to conclude that the Hiroshima-Nagasaki data are of insufficient accuracy to test any dose-exposure hypotheses. Lewis's analysis of several exposed groups summarized in Table 3, assuming a linear dose-effect relationship, suggested the incidence of leukemia to be 1 to 2 cases per million person-years at risk per rem.

Recent studies suggest that different types of cancer do not have the same dose incidence relationship.[19] These authors conclude: "It has been reconfirmed that in both sexes risk of leukemia mortality increases markedly with increase of dose. Also, in both sexes for all sites excluding leukemia, a slight trend is noted for the risk to increase with increase in dose. This increment is attributable chiefly to the increase of gastric cancer and lung cancer. Some, for example uterine cancer, show hardly any effect of exposure."

Studies made during autopsy indicated a slight tendency for higher mortality due to gastric cancer in females and lung cancer in females and lung cancer in both males and females, but the authors note that these trends were not statistically significant. No significant relationship was noted between radiation exposure and mortality due to cancer of the liver and biliary ducts and cancer of the uterus (in women).

Studies of the incidence of cancer, however, showed that thyroid cancer, breast cancer, lung cancer, and leukemia all showed increased incidence with increasing exposure. "However, in Nagasaki, while

Table 3. Summary of Lewis's estimates of the probability of radiation-induced leukemia per individual per rad per year. Source: Lewis 1957.[11]

Source of estimate	Type of radiation	Region irradiated	Types of leukemia produced	Probability of leukemia of specified type per individual per rad (or rem) to region irradiated per year		
				Estimated range		
				Lower limit	Upper limit	"Best" estimate
Atom bomb survivors	γ Rays plus neutrons	Whole body	All	0.7×10^{-6}	3×10^{-6}	2×10^{-6}
Ankylosing spondylitis Patients	x Rays	Spine	Granulocytic (only?)	0.6×10^{-6}	2×10^{-6}	1×10^{-6}
Thymic enlargement patients	x Rays	Chest	Lymphocytic (only?)	0.4×10^{-6}	6×10^{-6}	1×10^{-6}
Radiologists	x Rays, radium, etc.	Partial to whole body	All(?)	0.4×10^{-6}	11×10^{-6}	1×10^{-6}
Spontaneous incidence of leukemia (Brooklyn, N. Y.)	All natural background sources	Whole body	All(?)		10×10^{-6}	2×10^{-6}

[After Upton[18]].

incidence (for leukemia) increased with dose as in Hiroshima for the group exposed to 100 rad or more, no increase was noted under 100 rad." This latter conclusion by Maki et al.[19] indicates the difficulties (and possible overestimates) in deriving estimates of cancer incidence in humans at chronic low doses and dose rates from these data on acute high doses.

3.2. Ankylosing Spondylitis Patients

Studies of the subsequent incidence of disease in patients treated with x rays for ankylosing spondylitis have revealed an elevation in the incidence of leukemia and other cancers (see Table 4).

Table 4. Change in rate of induced malignant disease with duration of time since exposure in irradiated ankylosing spondylitics (data from Court Brown and Doll, 1965).[20]

Years after irradiation	Cases per 10 000 man-years at risk	
	Leukemia + aplastic anemia	Cancers at heavily irradiated sites
0-2	2.5	3.0
3-5	6.0	0.7
6-8	5.2	3.6
9-11	3.6	13
12-14	4.0	17
15-27	0.4	20
Total of expected cases in 10 000 persons in 27 years calculated from the rates given	67	369

Court Brown and Doll[21] first suggested a correlation between the incidence of leukemia in these patients and radiation exposure. Furthermore, in the dose range studied, the data were consistent with a linear relationship. Court Brown and Doll, however, excluded those cases in which extraspinal irradiation was given. Brues[22] has noted that this exclusion resulted in a severe bias in the analysis because the cases excluded were predominantly in the high-dose range. The complete Court Brown and Doll data thus indicate not only a curvilinear relationship, but perhaps also a threshold for leukemia induction in the range 50 to 100 R[22] (see Fig. 1).

Nevertheless, this study clearly demonstrates an almost 10-fold increase in leukemia among irradiated patients and an almost 30-fold increase in the related disease aplastic anemia, whereas cancer of other heavily irradiated sites was increased by a factor of only 1.6. In absolute numbers, 67 cases of leukemia and aplastic anemia were found, 61 cases more than expected as compared with 73 cases of all other cancer beyond the expected. However, there should be some caution in necessarily attributing this increase in cancer (other than leukemia) found in this study to irradiation. The largest contributor to the excess deaths from cancer of patients in the study was contributed by lung cancer, now well known to be caused by smoking and unfortunately the smoking habits of these patients are not known, and it is therefore possible that differences in cigarette smoking may be responsible for part or all of the difference in lung cancer rates between patients and controls. Furthermore, it is not known whether lung cancer may or may not be increased among patients with rheumatoid spondylitis irrespective of radiation. Lung disease is known to occur as part of the primary disease.[23] Still another reason for caution in attributing all these additional cancers to

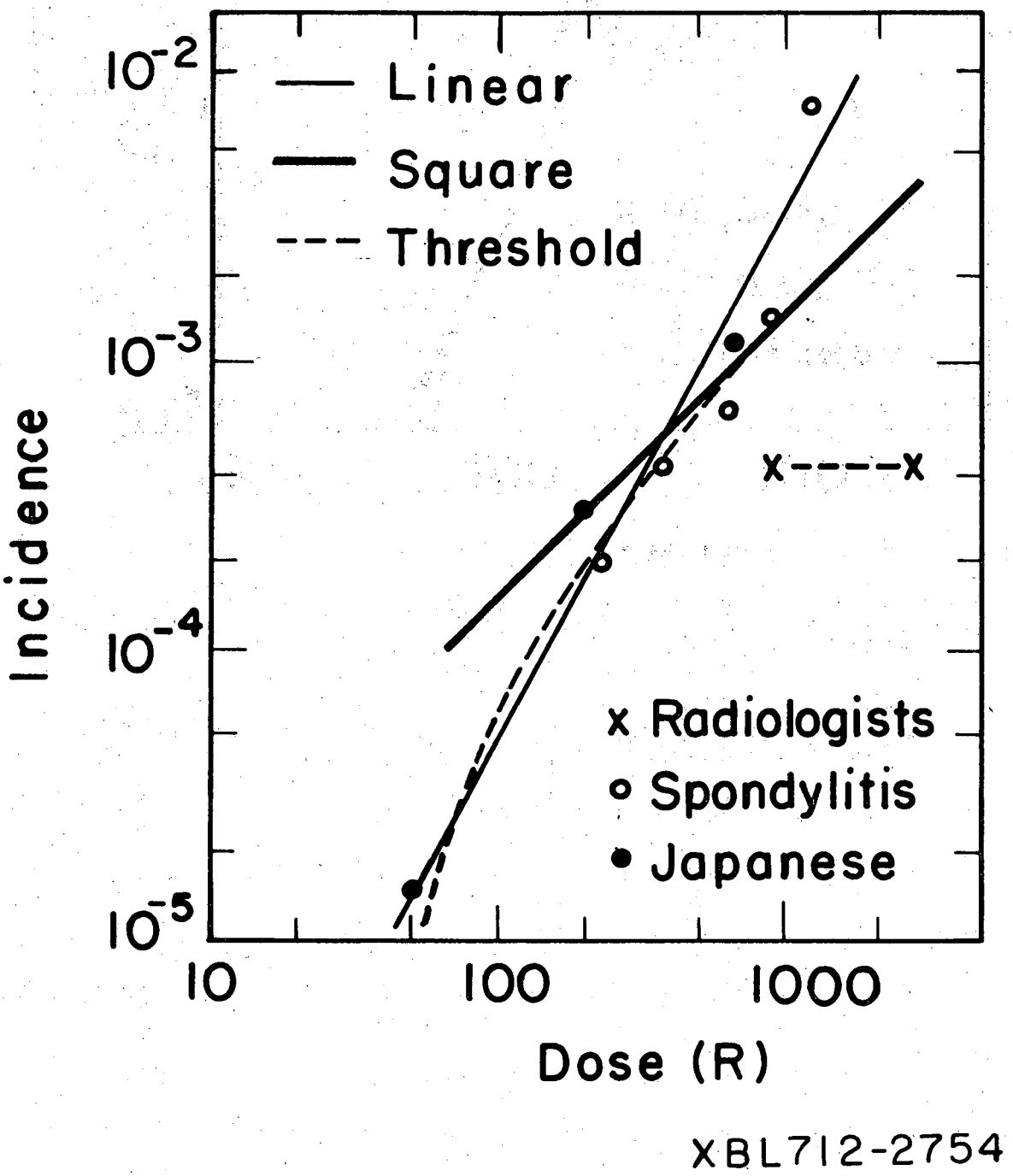

Fig. 1. The dose-response relationships for radiation leukemia in radiologists, irradiated spondylitic patients, and Japanese A-bomb survivors. (From Brues, 1959.)

radiation is due to the absence of the typical latent period, peaking, and decline in incidence associated with radiation-induced cancers.

3.3. Radium-Dial Painters

The fate of radium-dial painters who ingested toxic quantities of radium and radium daughters as a direct result of their occupation has been studied over the past 40 years. These painters absorbed radium through the mouth as a result of their practice of tipping their paint brushes with their lips. Radium and its daughters are depositied in bone and in time, if absorbed in sufficient quantities, can lead to skeletal damage, osteosarcoma, and other injury.[24] One of the most extensive and complete analyses of radium and mesothorium toxicity in human beings derives from the MIT group that has followed 604 cases of radium exposure over the past 40 years.[25-28] These data have been interpreted as showing both a curvilinear dose-effect response relationship and a practical threshold. The time for appearance of bone cancer is inversely related to the quantity of radium absorbed in bone. Thus at the point at which the latent period exceeds probable life span a practical threshold exists, and the MIT data put this at a few tenths of a microgram of radium deposited in bone. Statistical analysis of the data in which some incidence of bone cancer is observed (those cases in which the absorbed dose to the bone exceeds 1200 rads) indicates extreme improbability that the dose-response relationship is linear.

Other studies of radium-painters, of patients treated therapeutically with radium, and of animals have shown essential agreement with the conclusions of the MIT group.[29-36] Finkel et al.,[37] in a study of 293 patients treated with radium, found no person with a radium body burden below 1.2 μCi who had developed a malignant tumor ascribable to

radium deposition.

Recently Goss[38] has expressed some reservation about the analyses of the data in both these two studies. In the MIT studies it is suggested that the data do not exclude the possibility that the dose response model is linear and with no threshold. In the ANL studies Goss suggests that the higher-than-expected incidence of tumors of the central nervous system might be significant in an evaluation of risk.

It would seem that here are studies that would benefit from an independent analysis by one or more groups of statisticians.

3.4. Incidence of Lung Cancer in Uranium Miners

As early as 1500 the high incidence of lung disease amongst miners in the cobalt mines of Saxony and the pitchblende mines of Bohemia was recognized.[39] One component of this disease—colloquially referred to as "Berg Krankheit"—was finally identified, at the beginning of the twentieth century, as lung carcinoma. Sikl[40] suggested in 1950 that the one common factor to these mines that seemed primarily responsible for the high incidence of lung cancer was the radiation exposure from the radioactive daughters of uranium, particularly radon and polonium. Several studies of the incidence of lung cancer showed the death rate from lung cancer in these mines to be about 30 times as great as normally expected.[39]

Studies of the relationship between the incidence of lung cancer and radiation exposure for uranium miners in the United States have recently been reported.[41, 42] The lowest-exposure group studied in 1968 by a National Academy of Sciences Subcommittee[42] had cumulative exposures roughly corresponding to lung doses from radon and its daughter products up to 250 rads. After careful study the sub-

committee favored the hypothesis that radiation exposure had probably at least contributed to the higher incidence of lung cancer found in this group of workers than in the general population. However, they were careful to point out that a curvilinear relationship between dose and probability of cancer induction would be expected for lung cancer, which depends on localized tissue damage for its inception. Wagoner et al.[43] did in fact find a curvilinear relation between working level-months (a rough measure of radiation exposure) and annual incidence of respiratory cancer. Even after correction for the influence of age distribution in the working population, smoking habits, and number of years since onset of cancer, the relationship is still curvilinear.

3.5. Incidence of Leukemia in U. S. Radiologists

Some additional data may be gleaned from a study of the incidence of leukemia in the early U. S. radiologists, who—it is estimated—received doses as high as 2000 rads over a period of many years.[44] Although this cumulative dose resulting from chronic exposure was far in excess of a lethal single dose in man, it resulted in an incidence of leukemia far lower than for either the nuclear bomb victims or the ankylosing spondylitis patients (see Fig. 1). This fact suggests that some substantial dose-rate effect may be important.

The difficulties in establishing a measure of the risk of radiation-induced disease are evident from this brief review.

In its studies of external radiation effects on humans, ICRP has concentrated on two familiar sets of data: (i) those from a study of victims of the nuclear weapons attacks on Hiroshima and Nagasaki and (ii) those from the study of ankylosing spondylitis patients exposed to high levels of radiation for therapeutic reasons. Neither of these

studies provide evidence of an effect with whole body irradiation of less than 100 rads. In order to provide guide lines for the control of radiation exposure, however, ICRP have estimated the risk of the incidence of leukemia and other cancers on the basis of a linear dose effect–no threshold model. This model was not, however, advanced as a scientific hypothesis. Nevertheless, "...there must already be many health physicists who believe as a fact that radiation risks are linearly related to dose and independent of dose rate, although this simplification is little more than a convenient simplification from which to derive basic radiation standards." (45)

In discussing its most recent re-examination of the available data, ICRP concluded (46) "In essence this re-examination involved as detailed a sub-division as possible of the category of 'other fatal neoplasms' and the recognition that tissue dose was far from uniform in each of the three chief irradiated human populations–medical radiologists, ankylosing spondylitics and survivors of the atomic bomb explosions in Japan. It had also to be recognized that the time which has elapsed since exposure is still much too short for it to be possible to assess the full tumor incidence in the spondylitics and the Japanese: the following table shows that evidence collected during the first 15 years or so after exposure could be regarded as covering only the beginning of the period in which neoplasms other than leukemia might be expected to appear. If so, relatively small differences in the latent period of neoplasms arising in different tissues could lead to quite erroneous ideas about relative tissue susceptibility.

"The data in the table [Table 4] may also suggest that malignant disease other than leukemia will be 5–6 times more frequent than

leukemia plus aplastic anemia when the yield is assessed after 27 years of observation. However, in this context the rates cited for 15-27 years after irradiation are quantitatively the most important and it should be stressed that these have a considerable statistical uncertainty."

4. NATURAL BACKGROUND RADIATION

4.1 Terrestrial Radioactivity

Those radionuclides which have survived in measurable quantities in the earth's crust are of course those with half-lives comparable with the age of the earth ($\sim 5 \times 10^9$ years). Three radioactive decay chains account for much of the natural radioactivity to which man is exposed — the familiar uranium series (derived from ^{238}U), thorium series (^{233}Th), and the actinium series (^{235}Ac). Of the other naturally occurring radionuclides ^{40}K contributes most significantly to the natural background. In addition to these radionuclides of terrestrial origin one must include in this discussion of naturally occurring radioactivity those radionuclides produced by the interaction of cosmic radiation with the earth's atmosphere; of these, the most significant are ^{3}H and ^{14}C. Many extensive studies of terrestrial radioactivity have been made around the world, and the interested reader is referred to excellent summaries prepared by Claus,[47], Eisenbud,[48], Adams and Lowder,[49] and the United Nations.[50]

Table 5 shows the typical concentration of ^{40}K, thorium, and uranium in igneous and sedimentary rocks.

Table 5. Potassium-40, thorium, and uranium in igneous and sedimentary rocks (in ppm).

	Igneous Rocks		Sedimentary Rocks		
	Basaltic	Granitic	Shales	Sandstones	Carbonates
Potassium-40*					
Average	0.8	3.0	2.7	1.1	0.3
Range	0.2-2.0	2.0-6.0	1.6-4.2	0.7-3.8	0.0-2.0
Thorium					
Average	4.0	12.0	12.0	1.7	1.7
Range	0.5-10.0	1.0-25.0	8.0-18.0	0.7-2.0	0.1-7.0
Uranium					
Average	1.0	3.0	3.7	0.5	2.2
Range	0.2-4.0	1.0-7.0	1.5-5.5	0.2-0.6	0.1-9.0

* Chemical potassium contains 0.0119 per cent potassium-40.

These variations in concentration of radionuclides in rock naturally lead to changes in external radiation levels, and Table 6 shows estimates of external exposure levels for four regions around the world. We see that natural background levels due to this source may range by more than a factor of 10, principally depending upon the concentration of thorium, uranium, and potassium in the surrounding rocks.

Table 6. Mean dose of irradiation to gonads and bones from natural external sources in normal and more active regions.

Region	Population in millions	Aggregate mean dose[a] (mrem/y)
1. Normal regions	2500	75
2. Granitic regions in France	7	190
3. Monazite region, Kerala in India	0.1	830
4. Monazite region, Brazil	0.05	315

[a] Using a shielding factor of 0.63 for γ rays and a dose rate of 28 mrem/y due to cosmic rays.

Although there is large variation in external radiation levels from place to place, at a particular location there is little variation with time. Because the contribution to Man's external exposure is dominated by the component due to terrestrial radioactivity, it follows that the secular perturbations in the other sources of his external exposure, e.g., cosmic radiation, do not have a great influence in the variation of exposure with time.

Considerable variation in radiation exposure from buildings due to the use of differing construction materials is to be expected, however. Studies of the incidence of cancer and leukemia in areas of high terrestrial radioactivity or in areas which utilize building materials of high radioactivity have been suggested as possible sources of information in radiation-induced disease.

Table 7 lists some areas of high terrestrial radioactivity, while Table 8 lists areas with high radiation levels in dwelling houses due to the use of special construction materials.

Table 7. Some details of areas of high terrestrial radioactivity.

Area	Population	Demographic information available	Natural radiation received (multiply by 0.63 to get gonad dose)	Possible control populations
Part of Kerala State and adjoining area in Madras State	Approx 80,000	Some information on births and deaths: could probably be developed relatively easily	Approx 1300 mR/y (plus about 200 mrad beta rays)	Similar ethnic group further along coast
Monazite area in Brazil (States of Espirito Santo and Rio de Janeiro)	Approx. 50,000	Specially prepared statistics would be required	Average 500 mrad/y	?
Mineralized volcanic intrusives in Brazil (States of Minas, Geraes and Goiaz) — 6 km^2 in a dozen scattered places	Pastureland, scattered farms, 1 village with 350 inhabitants	Very little	Average 1600 mrad/year Peak value 12,000 mrad/y	?
Primitive granitic, schistous and sandstone areas of France with slight elevation of natural radiation said to cover about 1/6th of French population (7 million)		Specially prepared statistics would be required	180-350 mrem/y	Remainder of France estimated at 45-90 mrem/y

There are also some areas of high natural radiation in the Belgian Congo, but these are said to be uninhabited.

Table 8. Some details of areas with high natural radiation in houses made of special materials.

Area	Population	Demographic information available	Natural radiation received (multiply by 0.63 to get gonad dose)	Possible control populations
Sweden — houses made of light-weight concrete containing alum shale	Relatively small	Special statistics being obtained	158-202 mrad/y (cosmic radiation excluded)	Wooden houses 48-75 mrad/y (cosmic radiation excluded)
United Kingdom (Aberdeen) — houses and buildings made of granite	Population of Aberdeen approx. 186,000	Leukemia statistics being studied	Results from a few buildings indicate 102 mrad/y	Approx. 78 mrad/y in other cities with brick buildings, e.g., Dundee — population 178,000
Austria — granite houses	?	Special statistics necessary.	Granite houses 85-128 mrad/y Brick or concrete houses 75-86 mrad/y	Wooden houses 54-64 mrad/y

One interesting example of how Man may (unwittingly) change his radiation environment due to his use of a naturally radioactive substance has been reported by Jaworowski et al.[51] These authors studied the concentration of ^{226}Ra occurring in snow around a coal-burning power station in Warsaw. Table 9 shows their data presented as a function of distance from the generating plant. Similar data from U. S. coal-burning factories and stations could be developed.

Table 9. Concentration of ^{226}Ra in show around a power station in Warsaw.[a]

Distance from power plant (km)	pCi/kg ± S[b]
0.6	0.98 ± 0.12
1	0.63 ± 0.07
2	0.45 ± 0.07
4	0.076 ± 0.019
30	0.073 ± 0.033
45	0.019 ± 0.011

[a] From Jaworowski et al

[b] Statistical counting error at 0.95 confidence level.

4.2 Natural Radioactivity in the Diet

The natural radioactivity of soil necessarily leads to a transfer of radioactive material to human tissues through ingestion. Much of the α - activity ingested can be directly absorbed to decay products of the uranium and thorium radioactive series , in particular ^{226}Ra

and ^{228}Ra, and ^{210}Pb (and their decay products).

Table 10 gives estimates of the total human intake of ^{226}Ra and the contribution to the total from different foodstuffs for three different countries. We see that within the continental United States the average ingestion rate is about 2 pCi/day with some suggestion that the quantity ingested by young people is somewhat higher.

It is important to know what quantity of ^{226}Ra becomes permanently incorporated in human tissues (principally bone in this case). Table 11 shows the quantities of ^{226}Ra measured in human bone around the world. It seems that the total quantities of ^{226}Ra in the human skeleton correlate with the intake in the diet given in Table 10.

Table 10. Estimates of total intake of ^{226}Ra and of contributions from different foodstuff categories (from UNSCEAR Report, Ref. 50).

	United States						United Kingdom	India	
					Consumers' Union				
Category of foods	New York, N.Y.	Chicago, Ill.	San Francisco, Ca.	San Juan, P. R.	Five-city study	Teenager twenty-two city study	Country-wide study	Bombay	Kerala State Monazite area
Cereals and grain products.....	0.56	0.76	0.51				0.17	0.41	1.48
Meat, fish, eggs.....	0.38	0.37	0.28				0.38		
Milk and dairy products.....	0.14	0.12	0.13				0.14	0.04	0.19
Green vegetables, fruits and pulses.....	0.81	0.56	0.48				0.32	0.17	0.81
Root vegetables	0.40	0.22	0.26				0.10	0.02[a]	0.07[a]
Water	~0.02	~0.03	~0.01				0.07	0.06	0.29
Total pCi/day	~2.3	~2.1	~1.7	~0.7	~3 (2.2-4.3)	~5 (2.5-6.5)	~1.2	~0.7	~2.8
pCi ^{226}Ra/g Ca	2.2	2.0	1.6	1.3	1.9	2.5	1.1		

[a] Miscellaneous

Table 11. ^{226}Ra in human bone as reported after 1962 (from UNSCEAR Report, Ref. 50).

Location of area	pCi/g ash	pCi/g Ca	Total[a] in the skeleton(pCi)
NORMAL AREAS			
Central America			
United States			
Puerto Rico	0.006	0.017	17
Europe			
Federal Republic of Germany	0.013	0.040	36
United Kingdom	0.008-0.02		
North America			
United States			
Illinois	0.012[b]		32
New England	0.014		39
New York, N.Y.	0.012	0.032	32
Rochester, N. Y.	0.010;0.017		28, 48
San Francisco, Calif.	0.0096	0.026	27
HIGH LEVEL AREAS			
Asia			
India			
State of Kerala	0.096		~ 270
(monazite area)	(0.03-0.14)		
North America			
United States			
Illinois	0.037[c]		~100
Illinois	0.028[c]		78

[a] Skeleton of 7000 g fresh weight yielding 2800 g ash was assumed.

[b] In people consuming water with "normal" levels of ^{226}Ra.

[c] In people consuming water with elevated ^{228}Ra concentration.

4.3 Cosmic Rays

The principal variation is the dose rate from cosmic radiation is with altitude. Table 12 shows that the dose rate roughly doubles with an increase in altitude of 5000 feet.

Table 12. Cosmic-ray intensities at various altitudes (from S. A. Lough[52]).

Altitude, feet	Cosmic-ray intensity (μR/hr)
Sea level	4.0
1000	4.7
2000	5.4
3000	6.2
4000	7.1
5000	8.1
6000	9.1
8000	11.7
10000	14.6
12000	18.0
14000	21.0

Cosmic radiation contributes only about a third of the total external natural radiation levels and so such a change is not large. Furthermore the relatively small population that lives about 10 000 feet in the United States militates against carrying out a useful epidemiological study. Nevertheless it has been suggested that such studies might be made of populations who live at high altitudes, for

example, in La Paz in Bolivia. Table 13 gives details of high-cosmic ray intensity areas.

Table 13. Details of some high-altitude areas.

Area	Population	Demographic information available	Natural radiation received (multiply by 0.63 to get gonad dose	Possible control populations
La Paz, Bolivia (altitude about 11 909 ft 3630 m); latitude 16° S	Approx 319 600	Some statistics available but not comprehensive	Approx. 3-fold increase in cosmic rays near equator at 3000-4000 m above sea level. Cosmic radiation tends to be about a third of total external natural radiation	This might present difficulties as lower oxygen tension at high altitude is a complicating factor

Other high towns in South America —

Quito, Ecuador - altitude 9350 feet (2850 m) lat. 0°; pop 212 873

Bogota, Colombia - altitude 8660 feet (2640 m) lat. 4° N; pop 325 658

Cerro de Pasco, Peru - altitude 13 973 feet (4259 m) lat. 10° S; pop 19 187

Himalayan area - altitude 12 087 feet (3684 m); latitude 30° N; population (Lhasa) about 20 000.

Populations and altitudes from the Columbia Lippincott Gazeteer of the World (1952).

4.4 Summary

Table 14 [53] summarizes the exposures to Man due to natural background radiation.

Table 14. Various estimates of exposure of man to natural background radiation (mrad/y) (from Morgan and Turner.[53])

Type of Exposure		Mean dose to gonads (mrad/y)	Mean dose to bone (mrad/y)	Mean dose to lungs (mrad/y)
Internal, from radionuclides	^{40}K	19, 20, 18, 18, 22	10, 11, 7	15
	^{226}Ra		3, 3.8, 6.7, 3	0.5
	^{228}Ra		3	
	^{210}Pb		2	
	^{14}C	1, 0.7, 1.5, 1.3, 1.8	1.6, 1.3	
Internal from $^{220, 222}Rn$	^{220}Rn in wooden house			26 av, 45 max, 2.3 min
	^{220}Rn in brick house			46 av, 210 max, 2.7 min
	^{220}Rn in concrete house			94 av, 290 max, 3.9 min
	^{222}Rn in wooden house			19 av, 39 max, 1.3 min
	^{222}Rn in brick house			58 av, 120 max, 5.7 min
	^{222}Rn in concrete house			64 av, 140 max, 4.0 min
External, from ^{238}U and ^{232}Th series and from ^{40}K	Normal regions	47[a], (range 28-82)[a], 39[a]	Approximately the same as for gonads	
	Granite regions in France	162		
	Monazite regions in India	802		
	Monazite regions in Brazil	287		
External, from cosmic radiation	Sea level, 0° geo. lat.	23, 24, 35, 28, 30, 33	Approximately the same as for gonads	
	Sea level, > 50° geo. lat.	26, 27, 41, 37		
	10,000 ft, 0° geo. lat.	56, 50, 89, 80		
	10,000 ft, > 50° geo. lat.	84, 66, 128, 120		
Total for normal regions near sea level[b]		100 av, 150 max, 70 min	100 av, 150 max, 70 min (180), (250), (140)	200 av, 570 max, 70 min (1100, (4500), (110)

[a] These values were reduced for both sexes by a shielding factor of 0.63.

[b] The values given in parentheses are in units of mrem/y using an RBE of 10 for alphas.

5. MAN-MADE RADIATION

There are various sources of man-made radiation which contribute to population exposure. Nuclear reactors are relatively unimportant in terms of the radiation exposure they deliver to the population. This has been estimated by a number of authors to be less than 1 mrem/y average and no more than a few millirem per year to any individual. As reported at this Symposium, epidemological studies of populations living near nuclear reactors have shown no evidence of changes in infant mortality due to radiation exposure (the index of health suggested by some as the most sensitive indicator of radiation-induced disease[54]).

At the present time there is a dramatic increase in the number of nuclear power plants planned or under construction in the United States, as can be seen by inspecting Fig. 2. However, even with this large increase in the number of reactors it seems unlikely that the populations in their immediate vicinity will be suitable for epidemiological studies of radiation-induced disease because of the low exposures involved.

Fallout from nuclear weapons testing has, in the past, contributed significantly to population exposure. At present, it does not. Table 15 gives the dose commitments from nuclear explosions taking place between 1954 and 1965.

Table 15. Dose commitments from nuclear explosions (from UNSCEAR Report, Ref. 50).

Tissue	Source of radiation	Dose commitments (mrad) for period of testing 1954-1965
Gonads	External, short-lived	23
	^{137}Cs	25
	Internal, ^{137}Cs	15
	^{14}C[a]	13
	Total[b]	76
Cells lining bone surfaces	External, short lived	23
	^{137}Cs	25
	Internal, ^{90}Sr	156
	^{137}Cs	15
	^{14}C[a]	20
	^{89}Sr	0.3
	Total[b]	240
Bone marrow	External, short-lived	23
	^{137}Cs	25
	Internal, ^{90}Sr	78
	^{137}Cs	15
	^{14}C[a]	13
	^{89}Sr	0.15
	Total[b]	150

[a] As in the 1964 report, only the doses accumulated up to year 2000 are given for ^{14}C; at that time, the doses from the other nuclides will have essentially been delivered in full. The total dose commitment to the gonads due to ^{14}C from tests up to the end of 1965 is about 180 mrads.

[b] Totals have been rounded off to two significant figures.

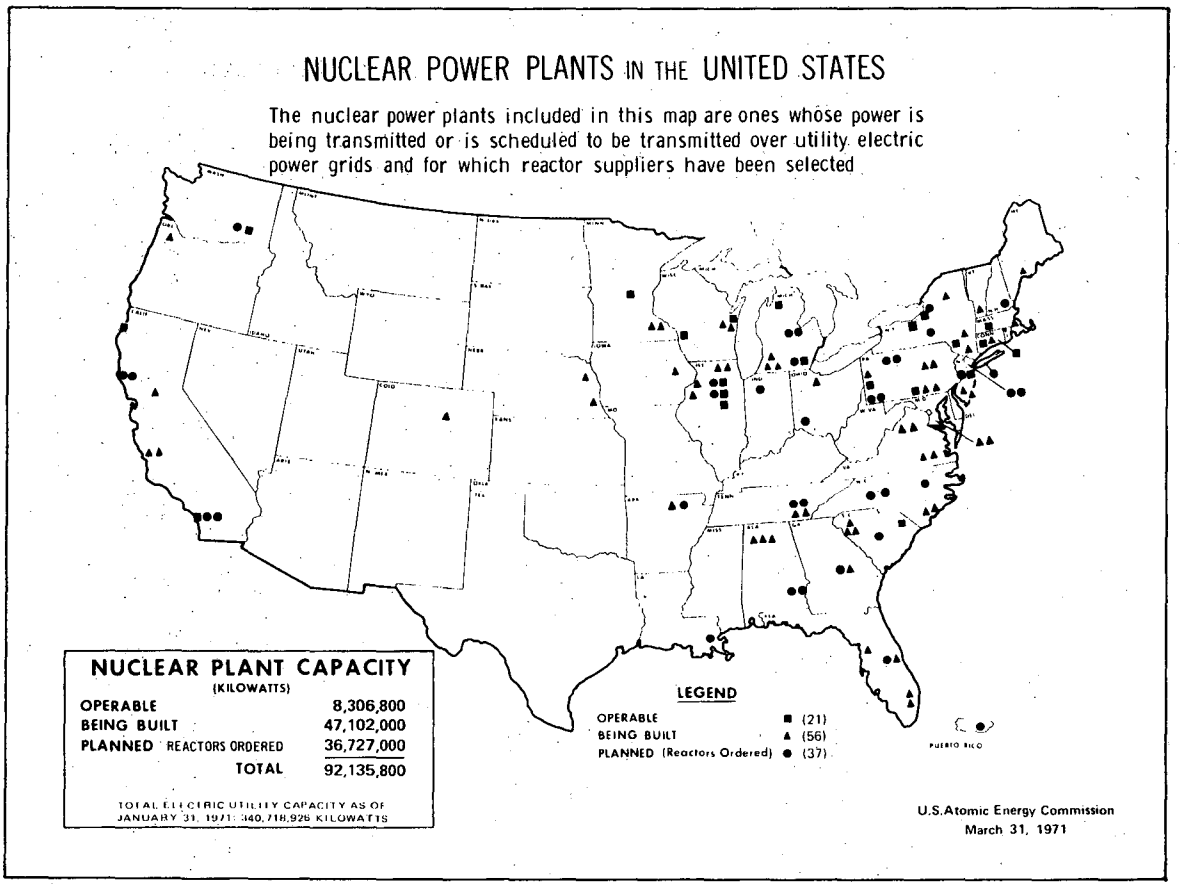

Fig. 2. Nuclear power plants in the United States (from Radiological Health Data and Reports, May 1971).

5.1 Radiation Exposures Resulting from the Medical Uses of Ionizing Radiation

Several authors, most recently the ICRP[55], have drawn attention to the increasing medical uses of radiation. The Adrian committee report identified medical radiology as the dominant component of man-made radiation in the United Kingdom. Table 16 summarizes typical estimates of the average genetic dose due to medical radiology in the late 1950's. Morgan[56] estimates that medical x-ray diagnosis accounts for over 90% of all radiation exposure from man-made sources. In 1963 the U. S. Public Health Service reported the genetically-significant dose from diagnostic radiology within the United States was 55 mrem/y. Morgan[56] has estimated that this has probably increased to 95 mrem/y on the basis of a recent USPHS survey.

It is possible to identify single procedures that contribute substantially to these exposures. Thus, for example, Penfil and Brown[57] estimate that nearly half of the genetically significant dose for U. S. males aged 15 - 29 years is due to x-ray examinations of the lower spine (see Fig. 3).

"Probably the most important criterion of the somatic damage incurred by a given population is the mean annual bone marrow dose per capita. Surveys have indicated that its magnitude is similar to the per-capita genetically significant dose." This may be seen in Table 17, which summarizes estimates of the gonadal and bone-marrow doses published recently by ICRP.

Table 16. Average genetic dose to each member of a population from diagnostic and therapeutic use of ionizing radiation (after K. Z. Morgan[53]).

Country	Diagnostic (mrem/y)	Therapeutic (mrem/y)	Radioisotopes (mrem/y)
United States	84	12	8
United States	137 ± 100	17	0.25-7
Australia	159	28	-
Hamburg, Germany	17.7	2.2	0.19
France	58.2	5.6	-
Leiden, Netherlands	6.8	4.1 - 13.1	-
United Kingdom	14.1	5	0.18
Denmark	27.5	1 - 1.5	-

Great attention has been given to the suggestion first made by Stewart in 1956[58] that prenatal exposure significantly increases the risk of cancer induction. MacMahon's[59] studies have supported the conclusion of Stewart et al. His data suggested an increase in cancer mortality by 40% among children who were irradiated in utero. Gibson et al.,[60] however, found no association between in utero irradiation alone and an increased risk of leukemia. This multivariant study of 13 000 000 children revealed an association between irradiation and an increased risk of leukemia only when other factors were involved.

Most recently Stewart and Kneale[61] have suggested that the leukemia incidence among such children is linearly related to the number of abdominal x-rays taken during pregnancy of the mother.

These studies have led some workers to suggest that infants and

Table 17. Gonad dose grouping, radiological examination of adults.

	Gonad dose (mrad)		Approximate percentage contribution to genetically significant dose	Mean bone marrow dose (mrad)	Approximate percentage contribution to per capita mean bone marrow dose
	Male	Female			
A. Low Gonad Dose Group					
Head (including cervical spine)	less than 10		less than 1	50	3
Dental (full mouth)	"		"	20	6
Arm (including forearm and hand)	"		"	<10	-
Bony thorax (ribs, sternum, clavical, shoulder)	"		"	100	-
Dorsal spine	"		"	200	-
Lower leg, foot	"		"	<10	-
Chest (heart, lung) including mass miniature radiography	"		4	40	35
B. Moderate Gonad Dose Group					
Stomach and upper gastro-intestinal tract	30	150	4	300	15
Cholecystography, cholangiography	5	150	1	100	-
Femur, lower two-thirds	400	50	4	50	-
C. High Gonad Dose Group					
Lumbar spine, lumbosacral	1000	400	18	200	7
Pelvis	700	250	9	100	2
Hip and femur (upper third)	1200	500	8	50	-
Urography	1200	700	12	500	10
Retrograde pyelography	1300	800	4	300	-
Urethrocystography	2000	1500	1	300	-
Lower gastro-intestinal tract	200	800	13	600	8
Abdomen	500	500	4	100	3
Obstetric abdomen		600	10	100	2
Pelvimetry	Foetal	1000			
		1200	7	800	4
	Foetal	4000		2000	
Hysterosalpingography		1200	< 1	300	-

*The gonad dose values given are composite figures from many measurements in many countries and are to be taken only as an indication of the order of magnitude of dose in the three groups. The mean bone marrow doses, included in this table for convenience are similarly composite figures. The mean bone marrow dose is the average dose to the active bone marrow [From ICRP Publication 16.[55]]

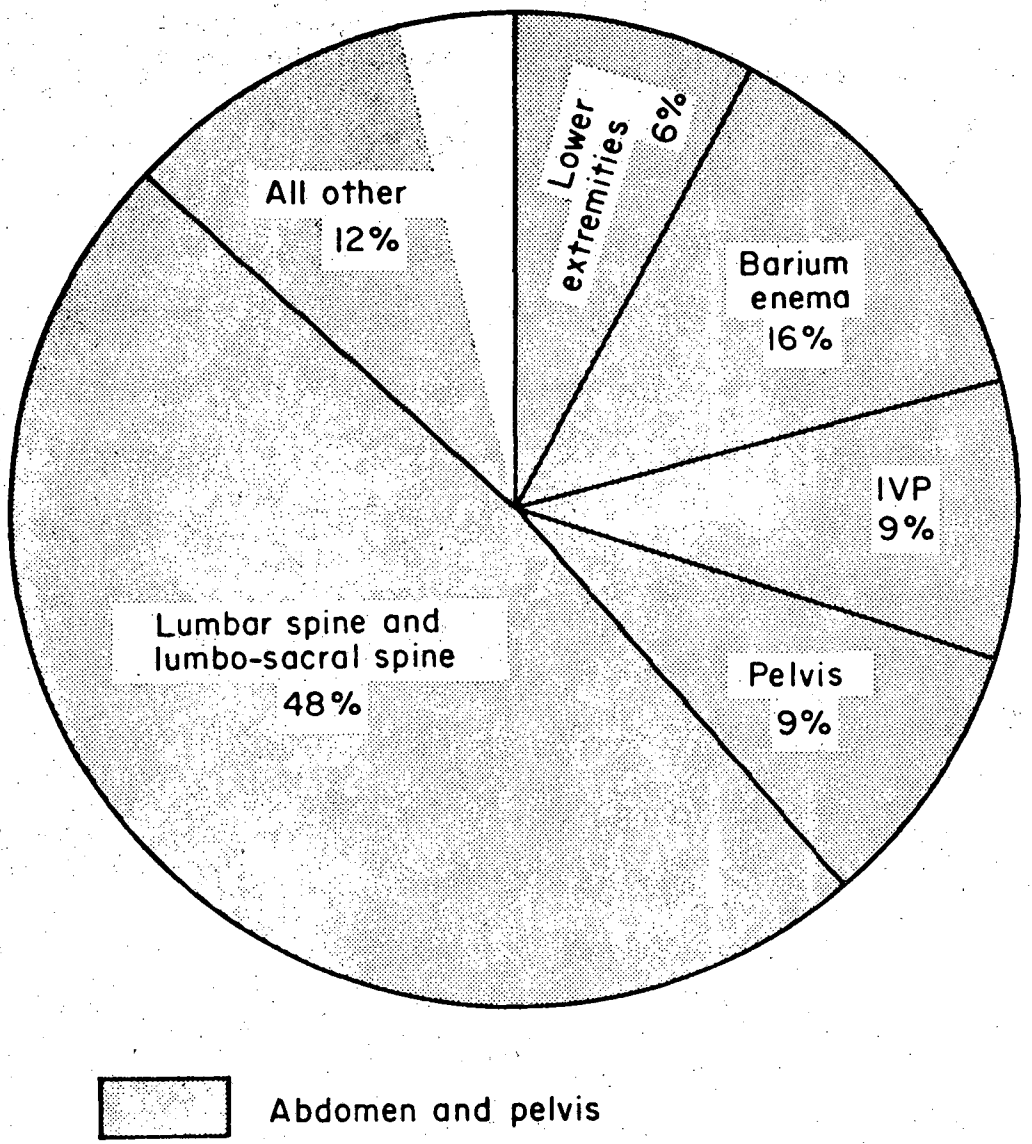

Fig. 3. Estimated percent distribution of genetically significant dose by type of medical roentgenological examination for males aged 15 - 29 years, United States, 1964, indicating that the major contributing examinations are those involving the abdomen and pelvis [From ICRP Publication 16[55].]

the developing embryo are some 100 to 1000 times more sensitive to radiation than the mature adult.[5, 62] Gofman et al[5] in a recent study suggest that in utero irradiation will result in a 50% increase in cancer mortality rate per rad.

It is surprising to us (perhaps because we are not statisticians) that there can be such disagreement as to the implications of these studies. It would be of great benefit to have an authoritative study of the mortality rates due to leukemia and cancers in young people over the past 50 years in the United States. If this were coupled with careful measurements of the medical radiation exposure to the individuals in the group studied it should be possible to make some definitive statements. If the risk of cancer induction is indeed as high as suggested by Gofman, Sternglass, and others we can expect to detect substantial increase in cancer mortality rates due to medical radiation exposures from studies of fairly small population groups.

6. CONCLUSIONS

In reaching our conclusions we should perhaps first indicate our general views as concerned scientists and citizens. Matters concerning the future welfare of mankind are of course, of grave concern to all of us. The fact of Man's pollution of his environment is not at debate; the impact of this pollution upon his health is not completely known. It seems to us that one of the first concerns of a symposium such as this should be to order its priorities. Given a limited amount of effort and talent that may be employed on identifying the significantly harmful components of pollution, it would indeed be tragic if this effort were

ineptly directed toward trivialities.

We, of course, hope to learn these priorities from symposia such as this, but, while reserving judgment, expect to learn that the risks due to "radiation pollution" do not rate high on the list of urgent priorities.

Nevertheless there are many valuable contributions that independent statistical studies may make to our understanding of the risks of low radiation doses.

At the present time our estimates of radiation risk basically all derive from high-dose, acute-exposure data. There does not seem to be general satisfaction with the analyses of the data. It would seem to us extremely worthwhile if much of these data were re-examined by fresh minds drawn from all the disciplines necessary for an exhaustive study. Such an authoritative independent study clearly stating what the high-dose data tell us about the dose-effect relationship would be invaluable in planning future studies of the induction of disease by low-radiation doses.

It does not seem reasonable to expect that we can establish from epidemiological studies that the risk of cancer induction by radiation is less than 10^{-4} per rad per year, since such a study would require a population containing 10 million man rem years at risk. While fairly large differences in radiation exposure from natural sources occur around the world, such differences are at most a few hundred mrem/y within the United States.

Of all man-made sources, medical x-rays are by far the greatest contributor to population exposure and little is known about the

individual exposure received by a member of the population. It seems imperative that any statistical study must take both population average exposure and individual exposures into account.

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*Work done under the auspices of the U. S. Atomic Energy Commission.

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APPENDIX: RADIATION CONCEPTS AND UNITS

The units and terminology used to quantify exposure to ionizing radiations is a source of confusion to more than laymen. We therefore append some brief definitions of the terms used in this paper, appealing to the knowledgeable reader to forgive us for stating the obvious.

The first attempts to quantify radiation fields began with x and γ radiation. Although the energy absorbed by irradiated material is important in determining the biological response of living organisms, in practice these energies are typically too small to measure directly. Energy absorption in air, however, produces ionization and provides a convenient method of measurement. Therefore the concept of <u>exposure</u> was developed,[1-3] which is a measure of the radiation based upon its ability to produce ionization. The special unit of exposure is the roentgen—one roentgen being that exposure that produces one electrostatic unit of charge of both positive and negative signs in one cubic centimeter of air at standard conditions of temperature and pressure.

It should be noted here that in this brief review of radiation units our discussion cannot be of great depth, our purpose being only to paint a broad canvas indicating points of special importance. The reader interested in more detail is referred to texts on radiation dosimetry—for example, that edited by Attix, Roesch, and Tochilin,[4-6] or the authoritative reports of ICRU.

Despite its great utility, dissatisfaction with the concept of exposure arose because of its exclusiveness—it is, for example, inappropriate for neutron irradiation—and the fact that exposure is not linearly

related to energy absorption in tissue. Both disadvantages are due to the basic difference in atomic composition of air and tissue. This difference is most striking for neutrons, since the production of recoil protons is the main mechanism for energy transfer to tissue, but even for photons the different chemical compositions of various tissues—fat, muscle, bone—compared with air become important at low energies.[7] A concept more widely applicable to radiation protection was needed. Since energy absorption seemed to be related to biological response, it was natural to define absorbed dose.

Absorbed dose due to any ionizing radiation is the energy imparted to matter by ionizing particles per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the "rad" and is equal to an energy absorption of 100 ergs/g.

Relative biological effectiveness is the ratio of the absorbed dose of reference radiation to the absorbed dose of a different radiation required to produce the same biological effect. An RBE may be specified for any kind of radiation or condition of exposure.

The RBE for radiation of type i is, then,

$$(RBE)_i = D_x/D_i,$$

where D_x, D_i are absorbed doses of 200 keV x rays and of radiation of type i to produce the same biological effect. Thus the biological effect of irradiation by n different types of radiation would be identical to that from $\sum_{i=1}^{n} (RBE)_i D_i$ rads of 200 keV x-rays. This concept was first known by the term RBE dose,[2] later becoming modified to dose equivalent;[3] its unit is the rem (Roentgen Equivalent Man).

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TECHNICAL INFORMATION DIVISION
LAWRENCE RADIATION LABORATORY
UNIVERSITY OF CALIFORNIA
BERKELEY, CALIFORNIA 94720