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Risk evaluation and protection against ionizing radiation

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Abstract. The advent of nuclear reactors ushered in an era of increasing number of sources of ionizing radiations. However, the potential of ionizing radiations to cause harmful effects was recognized soon after the discovery of x-rays and radioactivity *i.e.* long before the building of nuclear reactors. Therefore, protection against ionizing radiations has been of paramount concern and has guided the development of atomic energy and related fields. The advances in technology in general resulted in an increase in accidents causing injury and death. It was realised that even medicines, food additives and a host of other substances of daily use had injurious side effects. Smoking was found to be extremely harmful. From these emerged the concepts of quantitative and relative risks. This article discusses briefly the concept of risk *vis-a-vis* ionizing radiations and approaches to protection against them.

Keywords. Risk evaluation; ionizing radiation; radio therapy; cancer induction; somatic effects; diagnostic radiology; radiation protection.

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1. Introduction

X-rays and radioactive emanations, both were in common use soon after their discoveries. That these radiations might be harmful at high doses was also quickly realized. Voluntary individual and coordinated efforts towards protection were taken up early. Scientific bodies in countries like Germany (1913), UK (1915) and the USA (1916) were reviewing the requirement of their safe use. In 1925 the International Congress of Radiology was formed and under its patronage were born the International Commission on Radiation Units and Measurements (ICRU) in 1925 and the International Commission on Radiological Protection (ICRP) in 1928.

The ICRP issued its first recommendations in 1928 itself and the latest major revision came in 1977 (ICRP 1977a). While the presence of radiations and radioactivity has tremendously proliferated during the 90 years since their discovery considerable information and knowledge has also become available concerning their interactions with and the effects on the living systems. More is probably known about the consequences of exposure to ionizing radiations than any other environmental agent or occupational hazard. Paradoxically greater knowledge about the consequences of radiation exposure has led to its becoming an issue of serious concern to society (Pochin 1980; Goldman 1982). Partly this is due to the growing awareness of risks encountered in day-to-day experience: *viz* that medicines have side effects, food colours may be harmful, industrial effluents are hazardous, pesticides leave damaging residues and a host of similar other findings, besides the usual accidents, house collapses and fires, etc. Detailed studies of occupational mortality risks have been carried out with a view to have the right perspective of risks in different occupations (ICRP 1977b; Cohen 1981).

2. Evolution of the concept of risk

For a long time many scientists believed in a threshold level of radiation exposure below which no harm would occur and which was, therefore, thought to be a safe level. But data have been accumulating over the years which show that any exposure, however small, may not be considered so low that there was no risk due to it. It, therefore, became essential to make a quantitative assessment of the amount of risk or the degree of safety ensured at the prescribed level of dose limits. But this immediately leads to questions such as "what is the amount of risk which is acceptable", "how safe is safe enough?" (Fischhoff *et al* 1979; Watson 1981; Darby and Keeney 1981). Answers to these questions involve not only technical but economic as well as social aspects. Equally important are individual and collective public responses to risk. Perception of risk is most likely to have variants such as (i) economic constraints (ii) bias (iii) strong personal beliefs and (iv) the format of presentation. It is, therefore, very important to place risks in proper perspective. This can be done by placing them in a comparative order with other hazards or different sources of the same hazard. Ionizing radiations, for example, are encountered as natural background, in the environment, medicinally, occupationally and so on. This approach has led to the realisation that radon gas emanating from construction materials and accumulating in closed or poorly ventilated buildings is a major source of public exposures. The acceptability of risk is associated with the benefits derived from the source of the risk. It also follows that the acceptable risk would be the risk associated with the best of the available alternatives.

There are various approaches to determining the best alternative, but four methods frequently employed are cost-benefit analysis, revealed preferences, expressed preferences and natural standards (ICRP 1982; Starr *et al* 1976). The most used index of risk is the fatality rate both for natural disasters as well as human activities. It is observed that risks of the order of 10^{-3} fatalities per person per year are non-existent and are obviously in the category of unacceptable. Risks of the order of 10^{-6} per year, on the other hand, do not cause any anxiety to the average person and are hence acceptable. For risk levels between these limits, society spends money to reduce them, or pays compensation or derives commensurate benefits to put up with the inconvenience. Starr (1969, 1981) who studied these rates arrived at a societally acceptable and unacceptable benefit risk pattern of involuntary exposures shown in figure 1. He also analysed the

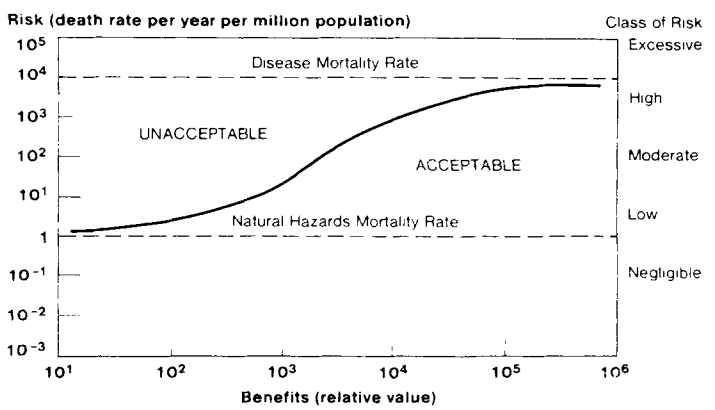

Figure 1. Benefit-risk pattern of involuntary exposure (Source: Starr 1981).

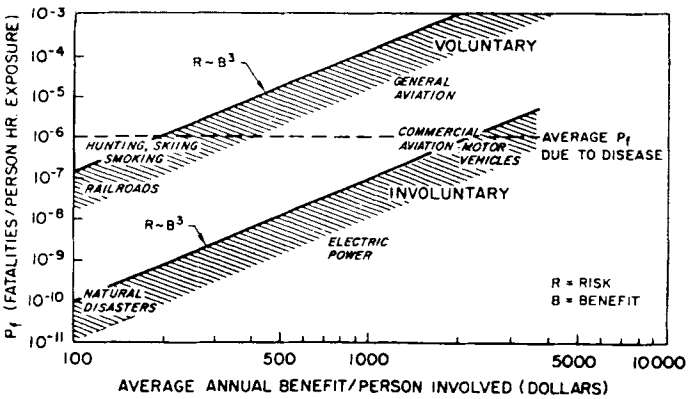

Figure 2. A comparison of risk and benefit to US society from various sources (Source: Starr 1970)

voluntary and involuntary aspects of risk, depicted in figure 2, and derived what might be called the "laws of acceptable risk" *viz*

(i) the acceptability of risk is roughly proportional to the third power of the benefits;

(ii) there seems to be a willingness on the part of the public to accept risks from voluntary activities, roughly a thousand times greater than it would tolerate from involuntary activities;

(iii) there is an inverse relationship between the acceptable level of risk and the number of persons exposed to that risk.

3. Ionizing radiations

3.1 *Nature and measurement of ionizing radiations*

The electromagnetic or particulate radiation interacts with matter causing physical disruption of the neutral atom into a knocked-out electron and the residual electron-deficient atom, the two constituting an ion pair. The process is called ionization. X-rays, gamma rays and beta particles produce these ion pairs in sparse paths and are called low LET (linear energy transfer) radiations. By contrast alpha particles, protons and heavy ions produce ion pairs in closely spaced paths and are called high LET radiations. The measurement of ion pairs produced in air is the first step in the measurement of radiation. The roentgen (R) is that amount of radiation which produces 1 esu of charge, positive or negative, in 1 c.c. of dry air at NTP. In the new international system of units (SI) radiation exposure in air is measured in units of coulomb/kg. The production of ion pairs causes absorption of energy leading to absorbed radiation dose. The deposition of 100 ergs/g of matter constituted 1 rad of absorbed dose. The SI system has replaced rad by Gray, symbol Gy, and equals 1 J/kg: 1 rad = 0·01 Gy.

The specification of absorbed dose is a physical event. The differences in the LET of different radiations causes differences in microscopically distributed energy in matter. To account for these differences, which ultimately lead to differences in biological effects an LET-dependent multiplier factor Q, called the quality factor, is used to obtain a new quantity called the dose equivalent (DE). The factor Q has values ranging from 1 to

20. The DE is measured in units of rem or its SI equivalent Sievert, symbol Sv; 1 rem = 0·01 Sv (ICRU 1980; Jain and Soman 1978).

3.2 *Radiation effects on the living system*

Ionizing radiations are known to affect living cells by killing, interfering with their normal functioning like delay in cell division, inducing chromosomal aberrations and gene mutations, etc. However, the living system is also capable of self-repair of at least some types of insults to it. Therefore, damages to the living organism are intervened by the repair mechanisms particularly at low doses of low LET radiations. The high cellular sensitivity to radiation (radiosensitivity) is governed by certain characteristics of the cell system like (i) primitiveness (ii) proliferative activity (iii) differentiation (iv) number of divisions required to attain specialisation/maturity etc. Lymphocyte is an exception to these general observations. Table 1 lists cellular sensitivity in the decreasing order. However, potential radiation-induced cancer risk is not necessarily correlated with rapid cell division in tissues highlighting perhaps the complexity of cancer etiology.

3.3 *Acute effects of radiation*

Information on the effects of radiation has come either from laboratory experiments on animals or persons exposed accidentally to high doses of radiation. *In vitro* studies of single cell preparations show that cell survival and reproductive capacity can be diminished by relatively high radiation doses. Uniform irradiation of the human body leads to changes in the blood count picture, the extent of which depends on the dose. The lymphocyte being the most sensitive, its numbers decrease rapidly and remain so for long periods. Hemoglobin being the least sensitive is affected least and decreases slowly. Other components of the blood have a response which is between these two extremes and is shown in figure 3 for neutrophils and figure 4 for platelets (Paretzke *et al* 1982). Table 2 gives the acute effects of whole body irradiation at different doses. Large total body doses impair most severely regenerative cells of the bone marrow, proliferative cells of the intestinal epithelium and the endothelial cells of the microvascular system. As a consequence a number of effects evolve in which phagocytic function is impaired, electrolyte balance is not maintained and gastrointestinal integrity is affected. The result is loss of defenses to infection and spread of internal infections.

Table 1. Cell sensitivity to radiations in the decreasing order.

1.	Lymphyocytes
2.	White blood cells and red bone marrow
3.	Intestinal crypt cells
4.	Cells of the reproductive organs (gonads)
5.	Liver cells
6.	Cells of glands
7.	Cells of the connective tissue
8.	Kidney cells
9.	Muscle cells
10.	Nerve cells

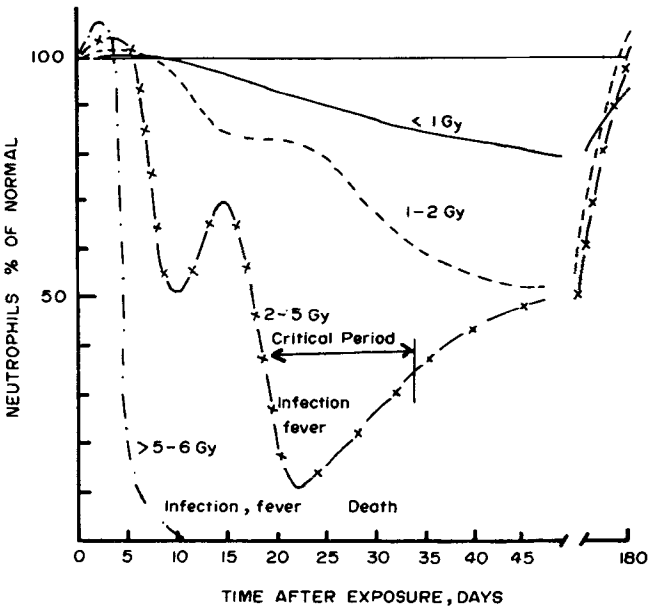

Figure 3. Acute effect of irradiation on neutrophils (Source: Paretzke *et al* 1982).

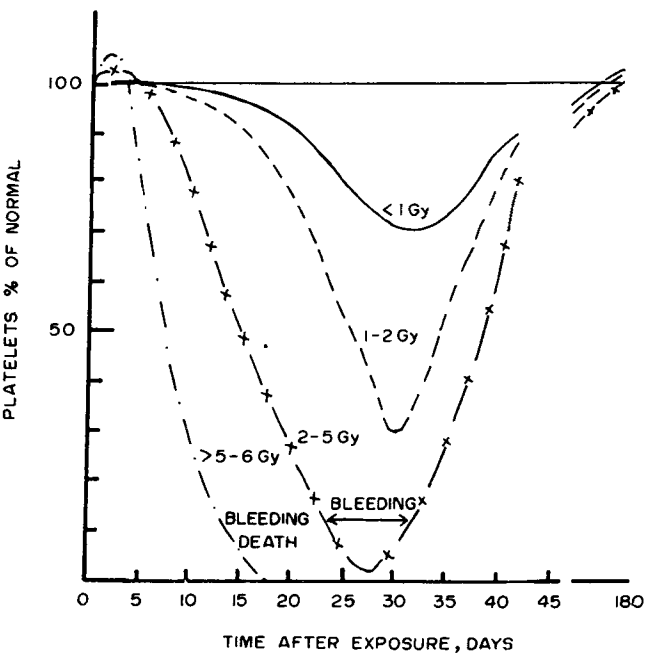

Figure 4. Acute effect of irradiation on platelets (Source as in figure 3).

Table 2. Effects of acute whole-body radiation doses (based on Glasstone and Sesonske 1963).

Acute dose, rems	Probable clinical effect
0 to 50†	No observable effects.
50† to 100	Slight blood changes but no other observable effects.
100 to 200	Vomiting in 5 to 50% of cases within 3 hours, with fatigue and loss of appetite. Moderate blood changes. Recovery will occur in all cases within a few weeks.
200 to 600	For doses of 300 rems and more, all exposed individuals will exhibit vomiting within 2 hours or less. Severe blood changes accompanied by hemorrhage and infection. Loss of hair after 2 weeks for doses over 300 rems. Recovery in 20 to 100% within 1 month to a year.
600 to 1000	Vomiting within 1 hour, severe blood changes, hemorrhage, infection, and loss of hair. From 80 to 100% of exposed individuals will succumb within 2 months; those who survive will be convalescent over a long period

† This number is sometimes given as 25. However, there is a recent tendency to believe that observable effects of radiation do not begin until about 50 rem.

Three different radiation syndromes, following acute exposure, have been noted. The first of these, occurring at 2 to 4 Gy, is the bone marrow syndrome in which due to heavy killing of blood cells, particularly stem cells of the bone marrow, there is very high susceptibility to infection and loss of nourishment. At somewhat higher doses, 4–6 Gy, the gastro-intestinal tract is severely affected leading to internal hemorrhage and infections. The central nervous system syndrome follows at very high doses of 10 Gy or more.

3.4 *Low level radiation effects and the latent period*

Low levels of radiation exposure do not cause any immediately observable effect. Protraction of dose over a long period of time causes much less severe effects than if the same dose were delivered acutely. Several animal studies have shown these differences in radiation effects. To cite one example a recent study on the effects of strontium 90, a low LET beta particle emitting radionuclide that concentrates in the skeleton shows that bone cancer is induced only when the radiation dose rates exceeded about 1 rad per day for several years *i.e.* after several hundred rems of dose (Raabe *et al* 1981). The American National Council on Radiation Protection and Measurements collected tumourigenesis data on some 12 different studies on laboratory animals covering doses upto 200 or 300 rem (NCRP 1980). They all show that low-dose rate radiation is about two to ten times less carcinogenic for the same doses compared with radiation delivered at high rates.

There is considerable incidence of natural cancer which in general increases with age. Each incremental dose of radiation causes an increase in this rate but with a time lag following irradiation in which no medical evidence is seen. This time lag is referred to as the latent period as explained in figure 5 (NAS 1980). For most solid tumors this is of the order of 10–15 years and for leukemia it is about 3–5 years. The excess rate continues for a further period of about 25 years for leukemia and lifetime for solid cancers. This is known as the period of expression (NAS 1980). Thus the principal, perhaps the sole,

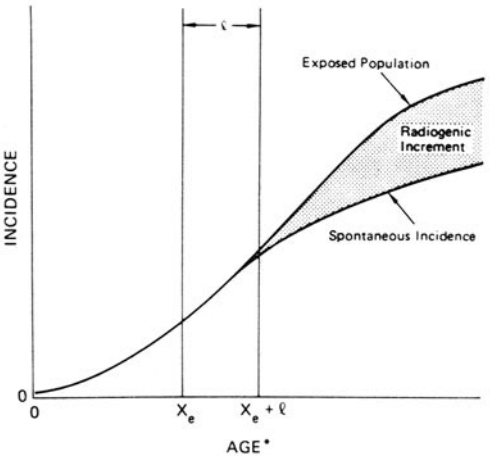

Figure 5. Radiation cancer incidence spontaneous and following exposure and latent period (l) (Source NAS 1980).

radiation effect, at low-dose ranges, in the exposed population is the somatic risk of increase in probability of cancer induction. It is important that the evaluation of this increase in probability of cancer induction *i.e.* risk be based on human experience and not on animal studies.

4. Somatic effects: Cancer induction

The most important somatic effect of concern for radiation protection purposes is the induction of cancer in different tissues. Table 3 gives the sensitivity to natural and radiation-induced cancer of various sites. To determine this a considerable number of epidemiological surveys have been made of human populations who have been exposed to known absorbed doses and have been followed with adequate ascertainment of cancer incidence or mortality, for long periods that are needed to estimate the frequency of cancer induced by radiation (UNSCEAR 1977). Reliable information in this connection has come from three main types of exposures (i) radiological procedures, therapeutic (for benign conditions) and diagnostic, (ii) Japanese survivors of atom bomb attack and people of Marshall Islands affected by fallout from weapon testing, (iii) occupational-exposure in uranium and other hard rock miners and in radium dial painters.

4.1 *Exposure in radiotherapy and diagnostic radiology*

Ankylosing spondylitis has been treated with x-rays as well as intravenous injection of radium 224. The former led to enhanced mortality from leukemia related to bone marrow dose and the latter to an increased incidence of bone sarcoma (Court Brown and Doll 1965; Spiess and Mays 1973). Certain other organs such as stomach, lung lymphoid tissues and intestine exposed within the radiation fields also showed increased mortality due to cancer related to the mean absorbed dose in these organs (Smith and Doll 1978). Similarly other radiotherapeutic treatments have yielded

Table 3. Organ sensitivity to radiation-induced cancer

Site of Cancer	Natural incidence	Radiation-induced
Female breast	Very high	High
Leukemia	Moderate	Very high
Thyroid	Low	Very high
Lung	Very high	Moderate
Stomach and colon	High	Moderate

estimates of cancer induction in breast, thyroid, brain, skin, etc (Shore *et al* 1977; Baral *et al* 1977; Hempelmann *et al* 1975; Modan *et al* 1974; Shore *et al* 1976). Treatment of benign diseases of the uterus by irradiation has provided risk estimates of pelvic organs (Smith and Doll 1976). Patients treated for thyroid cancer by radioiodine have given indications of leukemia incidence (Pochin 1969).

Diagnostic exposures being much lower, only in three situations the data has yielded information on cancer induction: firstly breast cancer increase in female patients who underwent repeated fluoroscopy during lung collapse therapy for tuberculosis (Boice *et al* 1979), secondly incidence of leukemia and liver cancer in patients who were given thorotrast as a contrast medium (UNSCEAR 1977); and thirdly increase in leukemia and other malignancies when the foetus got exposed during pelvic or abdominal radiographic examination of the mother (Stewart and Kneale 1970).

4.2 *Japanese survivors and Marshall islanders*

The exposed surviving populations from the cities of Hiroshima and Nagasaki (victims of atom bomb attacks) have shown enhanced frequencies of leukemia and at least nine other forms of malignancy namely the lung, female breast, thyroid and salivary glands, lymphoid tissues, bladder and urinary organs, aesophagus, stomach and large intestine (Beebe *et al* 1977). Although dose estimates are being revised (Lowe 1982) which may change the earlier derived risk factors, the fact remains that these studies have provided extensive human experience. Fallout from a weapon test on Bikini in 1954 resulted in ingestion of radio-iodine by some Marshall islanders. These have provided risk estimates for cancer of the thyroid (Conrad *et al* 1975).

4.3 *Occupation exposures*

Estimates of lung cancer induction by alpha radiation from radon daughter products to the bronchial epithelium among uranium and other hard rock miners have given relatively consistent risk estimates for the lung (UNSCEAR 1977). Uncertainties in radiobiological effectiveness of this radiation and the carcinogenic effect of smoking complicate the general applicability of these estimates. Definite evidence and correlation of bone cancer have been obtained from radium dial painters and from people who were administered radium for its supposedly therapeutic effect (Rowland *et al* 1971). Radiologists who were exposed to substantial occupational doses during the early part of this century, in spite of uncertainty of dose received, provide assessments

of excess over expected different fatal malignancies. From this relative frequency of leukemia as compared with all other malignancies, for more or less uniform irradiation of the whole body, can be obtained (Matanoski *et al* 1975).

5. Somatic risk estimation

5.1 *Dose-response models*

We have seen above that the cancer incidence data available is at high doses and high dose rates. The need, however, is to estimate the risk of cancer induction at very low doses and dose rates. Therefore, it becomes necessary to construct dose response models to fit the available data and extrapolate it to low doses. It is this modelling and extrapolation which has been a matter of controversy. In recent years, a general hypothesis for estimation of excess cancer risk in irradiated human populations, based on theoretical considerations, on extensive laboratory animal studies, and on limited epidemiological surveys has been developed which takes the complex quadratic form (NAS 1980):

$$I(D) = (\alpha_0 + \alpha_1 D + \alpha_2 D^2) \exp(-\beta_1 D - \beta_2 D^2),$$

where I is the cancer incidence in the exposed population at dose D(cGy).

This multicomponent dose-response curve contains initial upward curving linear (α_1) and quadratic (α_2) functions of dose, which represent the process of cancer-induction by radiation, and a modifying exponential function of dose which represents the competing effect of cell-killing at high doses (β_1 and β_2). The constant α_0 accounts for the natural incidence of cancer in the population. Attempts to fit the available data have demonstrated that for different radiation-induced cancers only certain of the parameter values of these constants can be theoretically determined. Therefore, simpler models for the dose-response relationship in the low-level dose range are advocated as shown in figure 6, namely,

(i) linear $I(D) = \alpha_0 + \alpha_1 D$,
(ii) quadratic $I(D) = \alpha_0 + \alpha_2 D^2$,
(iii) linear quadratic $I(D) = \alpha_0 + \alpha_1 D + \alpha_2 D^2$.

5.2 *Risk projection models*

The US National Academy of Sciences (1980), BEIR-III report and the United Nations Scientific Committee on Effects of Atomic Radiation (1977) report use two models: the absolute risk model and the relative risk model. The absolute risk is the expression of excess cancer risk due to radiation exposure as the arithmetic difference between the risk among those exposed and that occurring in the absence of exposure (figure 7). This model considers the latent period and the period of expression. The absolute lifetime risk co-efficient is expressed as the total number of excess cancer cases in the exposed population per unit collective dose. The relative risk is the expression of cancer risk due to exposure as the ratio of the risk among the exposed population to that occurring in the absence of exposures (figure 7). Therefore, the excess risk is a multiple of the spontaneous age-specific cancer rate in that cohort population. The greater the natural rate of cancer incidence in a population (*e.g.* ageing population) the greater will be the

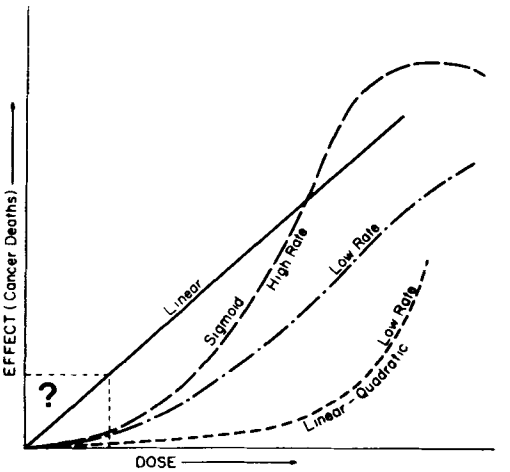

Figure 6. Radiation risk models (source Goldman 1982).

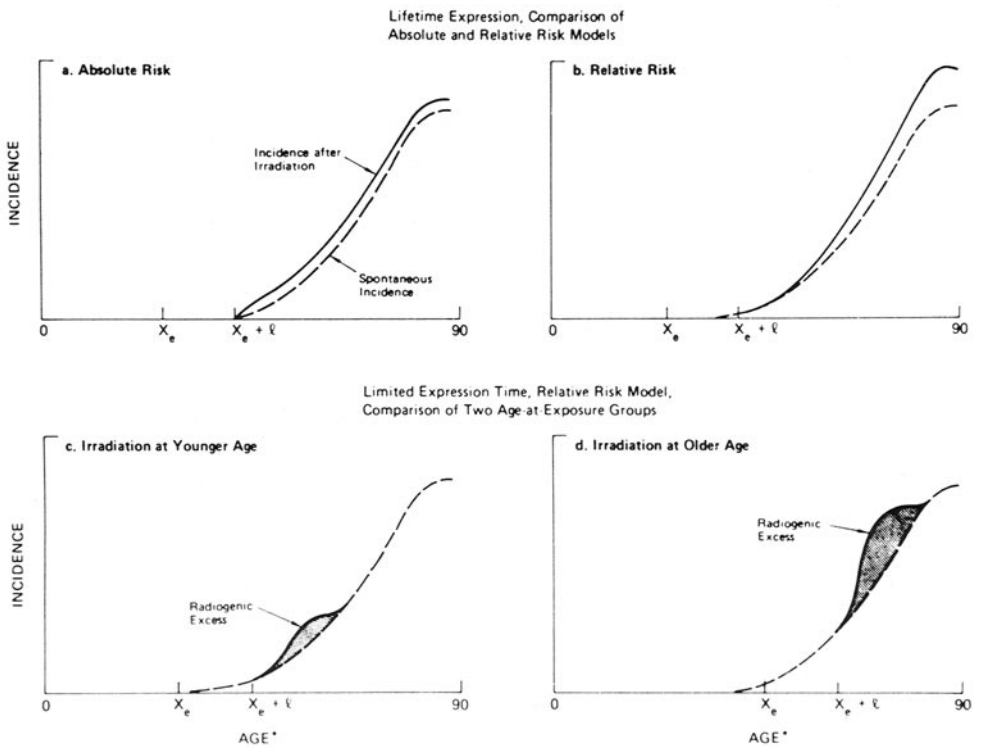

Figure 7. Models of absolute and relative risk and age dependence. Xe is age at exposure and l is the minimum latent period (Source NAS 1980).

Table 4. Risk estimates for individual organs—cancer risk. Fatalities per rem per 10^6 persons.

Organ	UNSCEAR 1977	BEIR III 1980	ICRP 26 1977
Blood (leukemia)	15–25	22	20
Breast	50	87	25
Lung	25–50	42	20
Thyroid	10	2·2 M 5·8 F	5
Bone	25	—	5
Stomach	10–15	15 M 17 F	10
Liver	10–15	8	10
Lower large intestine	10–15	—	10
Gonads			40

susceptibility of the individuals comprising that population to radiation-induced cancer. Since epidemiological studies on human population are not complete as yet (not followed upto death) there can be wide differences in risk estimation by the two methods at any one period of follow-up. When the study is complete so that no more cancers occur in the studied population, both models should lead to the same numerical estimates of lifetime cancers though the risk distribution may be different in the exposed population. The BEIR-III report emphasised that some experimental and human data, as well as theoretical considerations, suggest that for exposure to low doses of low LET radiation such as x-rays and gamma rays, the linear model probably overestimates but can be used to define the upper limits of risk of most radiation-induced cancers in man. Similarly the pure quadratic model may be used to define the lower limits of risk from low dose, low-LET radiation. For exposures to high-LET radiation (neutron, alpha particles, etc) linear risk estimates are less likely to overestimate the risk and may, in fact, underestimate. The International Commission on Radiological Protection has used the linear extrapolation to estimate risks. Table 4 gives the risk estimates for individual organs by different bodies.

6. Genetic effects

The induction of genetic mutations by radiations has been known now for more than five decades. A large amount of information is available on the frequency with which certain hereditarily transmitted abnormalities are caused in the mouse, as well as in various vegetable and other animal forms, by irradiation even at low doses (UNSCEAR 1977; Oftedal and Searle 1980). In man, however no corresponding information has been obtainable on the rate of induction of harmful genetic effects by radiation. But the frequency of chromosomal aberrations is known in human blood lymphocytes cultured after exposure of the blood 'in vitro' or after irradiation received *in vivo* in the course of medical or other procedures at known dose. But these cannot form a basis for genetic risk estimation. The latter estimates must at present be based largely upon mice data of

radiation exposure required to double the normal frequency, the doubling dose. This doubling dose for twelve different types of hereditary abnormality in male or female mice have ranged from 0·4 to 2·6 with a mean of 1·4 ($\pm$0·2, S.E)Gy (Searle 1977).

Genetic risk estimates have been obtained assuming that a comparable doubling dose, taken as 1 Gy for low-LET low-dose rate radiation, is a characteristic of genetic damage in mammals and may also apply to man (UNSCEAR 1977). Knowing the normal frequency of all genetic abnormalities that are maintained by mutation, a risk estimate of major genetic harm expressed in the first generation has been derived as $0{\cdot}9 \times 10^{-2}\,Gy^{-1}$ with $0{\cdot}35 \times 10^{-2}\,Gy^{-1}$ in the second, and a total of $3{\cdot}2 \times 10^{-2}\,Gy^{-1}$ in all generations (Oftedal and Searle 1980). UNSCEAR (1977) estimated $0{\cdot}65 \times 10^{-2}\,Gy^{-1}$ for first generation and a total of $1{\cdot}85 \times 10^{-2}\,Gy^{-1}$ for all generations. ICRP (1977a) has used a value of $10^{-2}\,Gy^{-1}$ for the first two generations and $2 \times 10^{-2}\,Gy^{-1}$ for all generations.

7. Risk limitation through system of dose limits in radiation protection

It is believed that like genetic mutations, for cancer induction also the initiating processes are chromosomal, since the abnormal reproductive behaviour of transformed cells must be transmitted in subsequent cell divisions (Pochin 1980; Rossi and Kellerer 1972). The dose-effect relationship is thus likely to have the same general form as for genetic effects. Cancer induction and genetic effects are therefore classed as stochastic effects; where there is no threshold and there is a probability of induction at all doses however low. Non-stochastic effects on the other hand occur if their threshold is exceeded and their severity depends on the amount of dose.

Non-stochastic effects are prevented by ensuring that no tissue receives annual doses which reach threshold values after a lifetime. ICRP (1977a) has recommended that no tissue shall receive more than 0·05 Sv annually under conditions of occupational exposure and 0·5 Sv per year in members of the general public.

The stochastic effects are not preventable but their frequency of occurrence can be limited. It is important, therefore, that unnecessary exposure shall be avoided and unavoidable exposure shall be as low as reasonably achievable (ALARA). The system of dose limits that is adopted, therefore, indicate maximum values which should never ordinarily be exceeded. The radiation safety of an occupation depends on the risks corresponding to the average annual exposure received. In order to compare the safety of occupations involving radiation exposures with those not involving, accounts should be ideally taken of the whole range of occupational injuries, diseases, fatalities, the mutagenic or carcinogenic exposures if any, and a variety of transient or permanent disabilities, etc (ICRP 1977b). Radiation exposures which amount to an average annual value of 5 mSv may cause a component of harm equal to about 3–4 accidental deaths annually per 100,000 workers. If on the other hand the working conditions are such that the annual dose limit of 50 mSv was commonly approached each year by most workers leading to an average dose of 30 mSv per year, then the radiation harm would be equivalent to about 20 accidental deaths annually per 100,000 workers (Pochin 1980).

A coherent system of dose limits must have knowledge of and suitably equate risks of irradiating individual body organs, relative to that of uniform whole body exposure to ensure compliance with the limits. To achieve this the ICRP (1977a) has used weighting factors based upon the risks of fatal cancers of different organs or genetic risks. Thus

radiation protection has come a long way from the earlier empirical though safe, dose limits to the recent more scientifically derived sound basis. A lot more data and quantitative information on several aspects of radiation effects are required to make radiation protection still more authoritative.

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References

Baral E, Larsson L E and Mattsson B 1977 *Cancer* **40** 2905

Beebe G W, Kato H and Land C E 1977 Studies of the mortality of A-bomb survivors, 8. Mortality experience of A-bomb survivors, 1950–74, Radiation Effects Research Foundation, RERF, Tech. Rep. 1–77.

Boice J D Jr, Land C E, Shore R E, Norman J E and Tokunaga M 1979 *Radiology* **131** 589

Cohen B L 1981 *Health Phys.* **40** 305

Conrad R A *et al* 1975 A 20-year review of medical findings in a Marshallese population accidentally exposed to radioactive fallout, Rep. BNL 50424, Brookhaven National Lab., Upton, New York, USA

Court Brown W M and Doll R 1965 Leukemia and aplastic anaemia in patients irradiated for ankylosing spondylitis, UK Medical Res. Council Spl. Rep. Series No. 295, HM Stationery Office, London (1957); 1965 *Br. Med. J.* **2** 1327

Darby S L and Keeney R L 1981 *Risk Analysis* **1** 217

Fischhoff B, Slovic P and Lichtenstein S 1979 *Environment* **21** 17

Glasstone S and Sesonske A 1963 *Nuclear reactor engineering* (New Jersey: Van Nostrand) (By permission: USAEC)

Goldman M 1982 *Western J. Med.* **137** 6

Hempelmann L H, Pifer J W, Hall J W, Phillips M, Cooper R A, and Ames W R 1975 *J. Natl. Cancer Inst.* **55** 519

ICRP 1977a Radiation protection, *Ann. ICRP*, Vol. 1 No. 3, International Commission on Radiological Protection (ICRP), Pub. No. 26

ICRP 1977b Problems involved in developing an index of harm. *Ann. ICRP*, Vol. 1 No. 4, ICRP Pub. 27 (Oxford: Pergamon)

ICRU 1980 Radiation quantities and units, ICRU Rep. No. 33, ICRU, Washington DC, USA

ICRP 1982 General principles of monitoring for radiation protection of workers. *Ann. ICRP*, Vol. 9 No. 4, ICRP Pub. 35

ICRP 1983 Cost-benefit analysis in the optimisation of radiation protection. *Ann. ICRP*, Vol. 10 No. 43, ICRP Pub. 37

Jain V K and Soman S D 1978 SI units in radiation protection, BARC Rep. 964, BARC, Bombay

Lowe W E 1982 A-bomb survivor dosimetry update. UCRL-86275, Lawrence Livermore National Lab., Livermore, Calif., USA

Matanoski G M, Seltzer R, Sartwell P E, Diamond E L and Elliott E A 1975 *Am. J. Epidemiol.* **101** 188, 199

Modan B, Baidatz D, Mart H, Steinitz R and Levin S G 1974 *Lancet* **1** 277

NCRPM 1980 Influence of dose and its distribution in time on dose-response relationships for low-LET radiations, Rep. No. 64, National Council on Radiation Protection and Measurements, Washington DC

NAS 1980 The effects on populations of exposure to low levels of ionizing radiations. Rep. of Committee on Biological Effects of Ionizing Radiations, National Academy of Sciences, BEIR III

Oftedal P and Searle A G 1980 *J. Med. Genet* **17** 15

Paretzke H G, Dennis J A and Massue J P 1982 *Ionizing radiation and protection of man, Vol. 2, A book of teaching modules* (Neuherberg, FRG: Gesellschaft fur Strahten und Umweltforschung Abteilung)

Pizarrello D J 1982 Ed. *Radiation Biology*. CRC Press Inc., Boca Raton, FL, USA

Pochin E E 1969 Long-term hazards of radioiodine treatment of thyroid carcimoma. Thyroid cancer, UICC Monograph Series Vol. 12 Hedinger C Ed. Springer-Verlag Berlin

Pochin E E 1980a *Phys. Med. Biol.* **25** 1

Pochin E E 1980b *Atomic Energy Rev.* **18** 779
Raabe O G, Parks N J, Brook S A 1981 *Radiat. Res.* **86** 575
Rossi H H and Kellerer A M 1972 *Science* **175** 200
Rowland R E, Failla P M, Keane A T and Stehney A F 1971 Tumor incidence for the radium patients. Radiological Physics Division *Ann. Rep.* ANL 7860, Part II, Argonne National Lab., USA
Searle A G 1977 Use of doubling doses for the estimation of genetic risks. *Proc. Europ. Symp. on Rad. Equivalence*, CEC Ser. on Radiological Protection EUR 5725 e, 133
Shore R E, Albert R E and Pasternack B S 1976 *Arch. Environ. Health* **31** 21
Shore R E, Hempelmann L, Kowaluk E, Mansur P S, Pasternack B S, Abert R E and Haughie G E 1977 *J. Natl. Cancer Inst.* **59** 813
Smith P G and Doll R 1976 *Br. J. Radiol.* **49** 224
Smith P G, and Doll R 1978 Late biological effects of ionizing radiation, *Proc. Symp. Vienna*, Vol. 1, IAEA, Vienna, p. 205
Spiess H and Mays C W 1973 Radionuclide carcinogenesis, US Atomic Energy Agency Symp. Ser. No. 29, Wash. p. 437
Starr C 1969 *Science* **165** 1232
Starr C 1970 Benefit cost relationships in sociotechnical systems. Environmental aspects of nuclear power stations, IAEA Symp., New York, p. 895
Starr C 1981 *Risk Analysis* **1** 117
Starr C, Rudman R, Whipple C 1976 *Annu. Rev. Energy* **1** 629
Stewart A and Kneale G W 1970 *Lancet* **1** 1185
UNSCEAR 1977 & 1980, Report of United Nations Scientific Committee on Effects of Atomic Radiation, United Nations, New York, USA
Watson S R 1981 *J. Soc. Radiol. Prot.* **1** 21