From FA Smith, Applied Radiation Physics

1.4.3 Cyclotron

A conventional cyclotron uses resonance radio-frequency (rf) acceleration of heavy charged particles in a uniform dc magnetic field. This is achieved by placing an ion source at the centre of two "D"-shaped semicircular, hollow electrodes. The rf field is applied across the electrodes such that each "D" goes alternately positive and negative at the rf frequency, f_0. The cyclotron can only be used to accelerate heavy ions (p, d, He, H^- ....).

The motion of a particle of mass m and charge ze having velocity v, moving in a magnetic induction *B* with radius r, is governed by the Lorentz and centripetal forces.

$$B\ ze\ v = m\frac{v^2}{r} \tag{1.15}$$

The total energy *E* of the particle having kinetic energy *T*, is :

$$E = T + W_0 = T + m_0c^2 = mc^2 = \frac{m_0c^2}{\sqrt{(1-\beta^2)}} \tag{1.16}$$

where W_0 is the rest energy of the particle and $\beta = v/c$. Using Eqs.(1.15) and (1.16), the orbital frequency of the ion can be written :

$$f_i = \frac{v}{2\pi r} = \frac{ze\,B}{2\pi m} = \frac{ze\,B\,c^2}{2\pi(W_0 + T)} \qquad (1.17)$$

The resonance acceleration can be maintained only for a constant frequency, f_0. Since the kinetic energy of the particle increases with each crossing of the gap, the condition $f_i = f_0$ is only possible if $T << W_0$, *i.e.* at low energies.

A positive ion which emerges from the source in Fig.(1.12) is accelerated when the right-hand D is in the negative half cycle. There is no accelerating field within the hollow D so the particle experiences only the magnetic field. The particle follows a semicircular path until it reaches the edge of the D. If the time to traverse this path is the same as the time necessary for the left-hand D to become negative, the acceleration process will continue. For continuous acceleration the phase of the rf field must be slightly ahead of the phase at which the particle crosses the gap. This is the resonance condition.

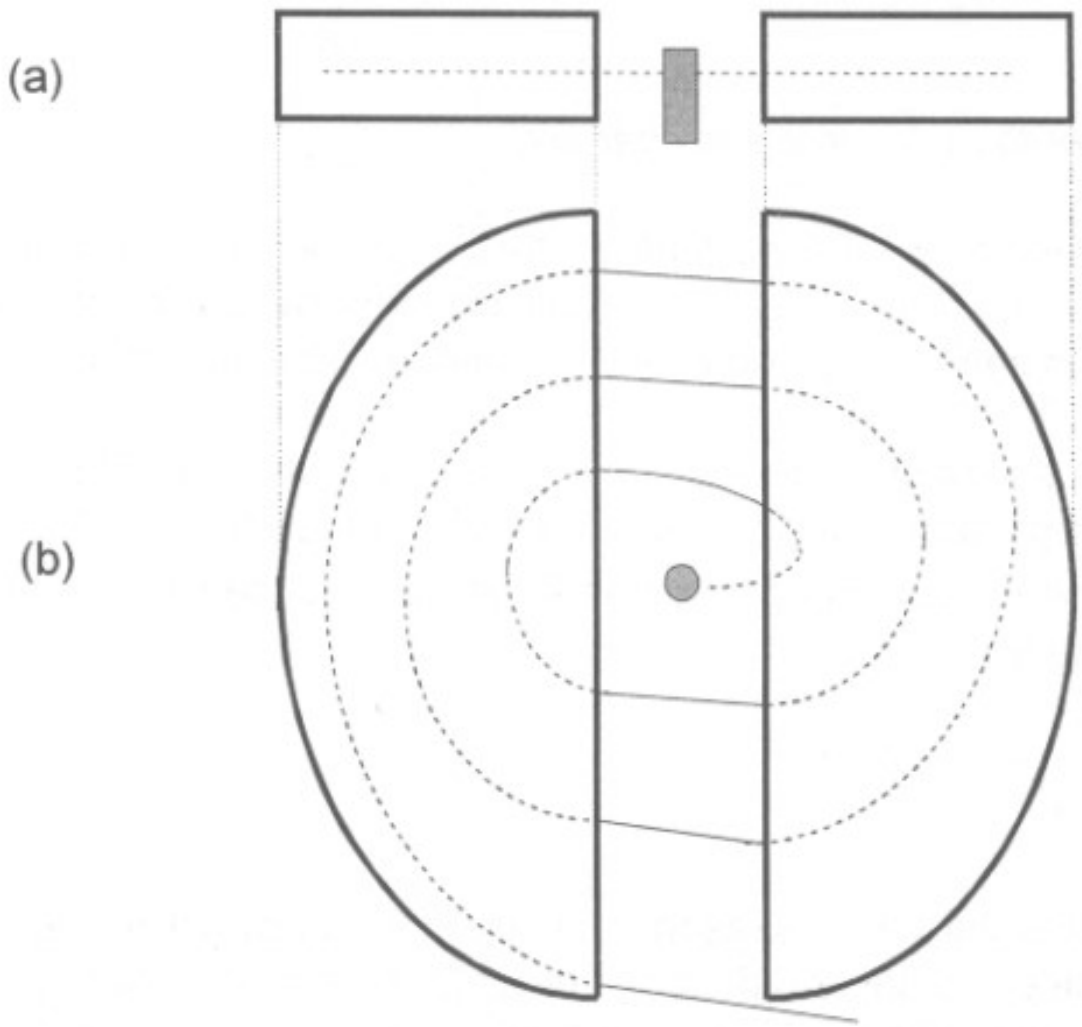

Fig.(1.12) Schematic diagram of the "D" structure of a cyclotron. (a) cross section (b) plan view. The ion source is at the centre and the dc magnetic field lines are perpendicular to the plane of the diagram, [7].

Weak-focusing

The requirement is that the lines of magnetic induction are always concave inwards towards the centre of revolution of the ions. This is achieved by the introduction of shim material, Fig.(1.13), in the central regions of the field together with the shaping of the pole pieces at the extremities of the field.

To set up the condition of weak-focusing, the magnetic induction B_0 at a final orbit radius of r_0 is first specified. At any other radius, r, the magnetic induction must then be given by :

$$B = B_0\left(\frac{r_0}{r}\right)^n$$

where $0<n<1$ for particles travelling near the final orbit radius. The consequence of this restoring force is that there are oscillations of the ion in both radial and axial di

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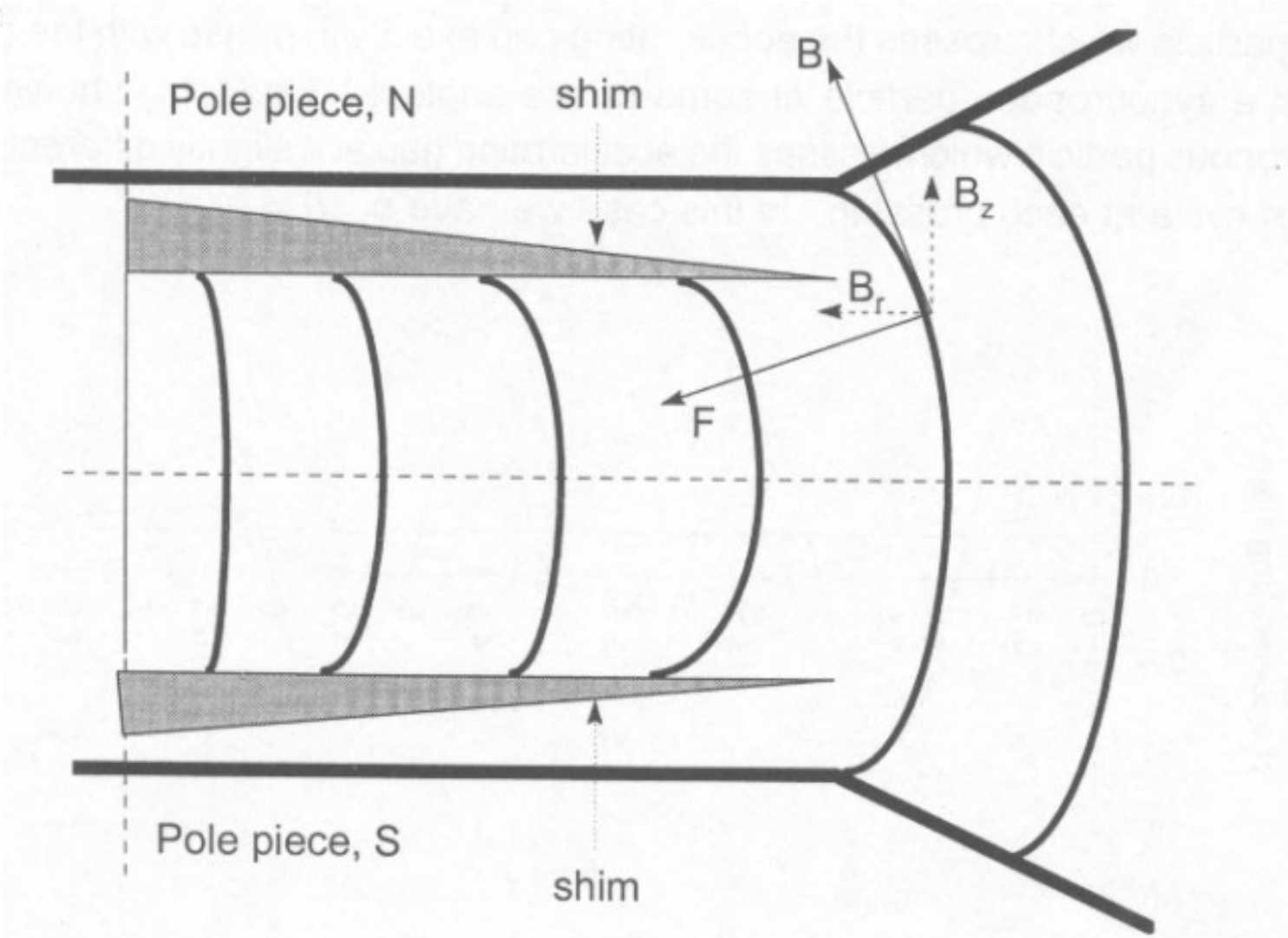

Fig.(1.13) Lines of magnetic induction in a weak-focusing field. A particle which is not travelling on the central plane will experience a force F which always tends to restore it into the central plane. This has an axial component F_z towards the central plane and a radial component F_r towards the centre of revolution, [7].

These oscillations have frequencies, [7] :

$$\text{Axial: } f_z = n^{1/2} f_i$$

$$\text{Radial: } f_r = (1-n)^{1/2} f_i$$

and are always smaller than the ion frequency, hence the name 'weak-focusing'. The overriding requirement is that the magnetic induction decreases as the radius increases.

Phase stability

The above requirement for weak-focusing is not consistent with the resonance

condition in Eq.(1.17). As T becomes significant with respect to W_0 the ion frequency f_i decreases. In order to maintain the resonance condition in a constant magnetic field, either :

- the rf frequency f_0 must also decrease or,
- if f_0 remains constant, B must increase as r increases. Certainly it cannot decrease, as in weak-focusing.

RADIATION PROTECTION

11.1 Introduction

The International Commission on Radiation Protection (ICRP) aims to provide a general system of radiological protection that can be applied to any situation in which humans are, or are likely to be, exposed to radiation [1]. It publishes recommendations, the latest being ICRP 60 [1], which draw on scientific evidence as well as using value judgements to assess the relative risks of radiation exposure.

At low dose levels, which are the main concern for protection of the general population, it is most important to establish whether or not there exists a dose threshold below which no effect is present. If there is not, then a finite risk must always be accepted however low the dose. Conversely, if there is a threshold, it no longer becomes possible to apply the following proportional relationships that are the basis of practical radiation protection [2]. These are that :

- the dose received by an organ or tissue can be averaged over the organ or tissue,
- doses received at different times can be added, and
- the dose from one source can be considered independently of the dose from any other source.

11.2 Units and Special Parameters

In section 9.4 reference was made to the concept of Relative Biological Effectiveness (RBE). This was used to relate the biological response of tissue to absorbed dose from radio-therapeutic beams of different qualities. Publication 60 of the ICRP [1] redefined this relation by making use of two parameters :

- the Equivalent Dose in an organ, and
- the Effective Dose.

For the sole purpose of expressing dose limits, Equivalent Dose and Effective Dose have been recommended by ICRP as being the best way to correlate radiation exposure with the risk of developing cancer.

In this chapter, the use of the term "dose", in units of Sievert (Sv), is applied to Effective Dose unless otherwise stated. It applies to all exposure conditions, whether caused internally or externally, and represents an absorbed dose weighted for different radiations and for the carcinogenic biological sensitivity of different organs.

11.2.1 Equivalent dose

The Equivalent Dose $H_{T,R}$ (in Sv) received by tissue T which has been exposed to a dose $D_{T,R}$ (in Gy) of radiation R is then specified using :

$$H_{T,R} = w_R \; D_{T,R}$$

Table (11.1) Radiation Weighting Factors from [1], [3]. Both Reports give the identical weighting factors apart from the value for protons. Note how the radiation weighting factor reflects the distribution of radiation quality, see Fig.(9.34).

Radiation and energy range	w_R (NCRP-116)	w_R (ICRP-60)
X-and γ-rays, electrons, positrons, muons	1	1
Neutrons, $E_n < 10$ keV	5	5
$10 < E_n < 100$ keV	10	10
$0.1 < E_n < 2$ MeV	20	20
$2 < E_n < 20$ MeV	10	10
$E_n > 20$ MeV	5	5
Protons, $E_p > 2$ MeV (not recoil protons)	2	5
α - particles, fission fragments, etc	20	20

Table (11.2) Tissue Weighting Factors from references [1] and [3]

Tissue	w_T
Gonads	0.20
Red bone marrow	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Structural bone	0.01

Since many radiation fields consist of more than one component – for example, photons and neutrons with a range of energies – the overall equivalent dose is given by :

$$H_T = \sum_R w_R \; D_{T,R} \tag{11.1}$$

11.2.2 Effective dose

To account for the different radio-sensitivity of different body tissues, a tissue weighting factor w_T is also used, Table (11.2). This then defines the Effective Dose, *E*.

$$E = \sum_T w_T H_T \tag{11.2}$$

In the compilation of a table of tissue weighting factors it is implicitly assumed that w_T does not depend on radiation quality. It is therefore assumed to be independent of w_R [4].

11.3 Background Levels

Figures given in Table (1.1) for contributions to the average radiation background in the UK hide important variations for certain sub-groups of the population. The National Radiological Protection Board (NRPB) provides estimates for some of these regional and occupational distributions [5]. Changes in the estimates for the years prior to 1986 and those up to 1994 are due largely to :

- the improvements in the monitoring of local radon concentrations,
- the more widespread use of computerized tomography in diagnostic radiology, and
- reduced discharges and radioactive fallout to the environment.

Variations in geographical region and occupational grouping within the UK can result in significant changes from the mean annual background dose burden.

The following figures and table show variations due to :

- a reduced radioactive fallout with time, Fig.(11.1)
- the increased cosmic ray contribution with altitude above mean sea level, Fig.(11.2)
- the geographical variation of radon contribution to the total burden, Table (11.3)
- the reduced radioactive discharge to the environment in a particular region, Fig.(11.3)

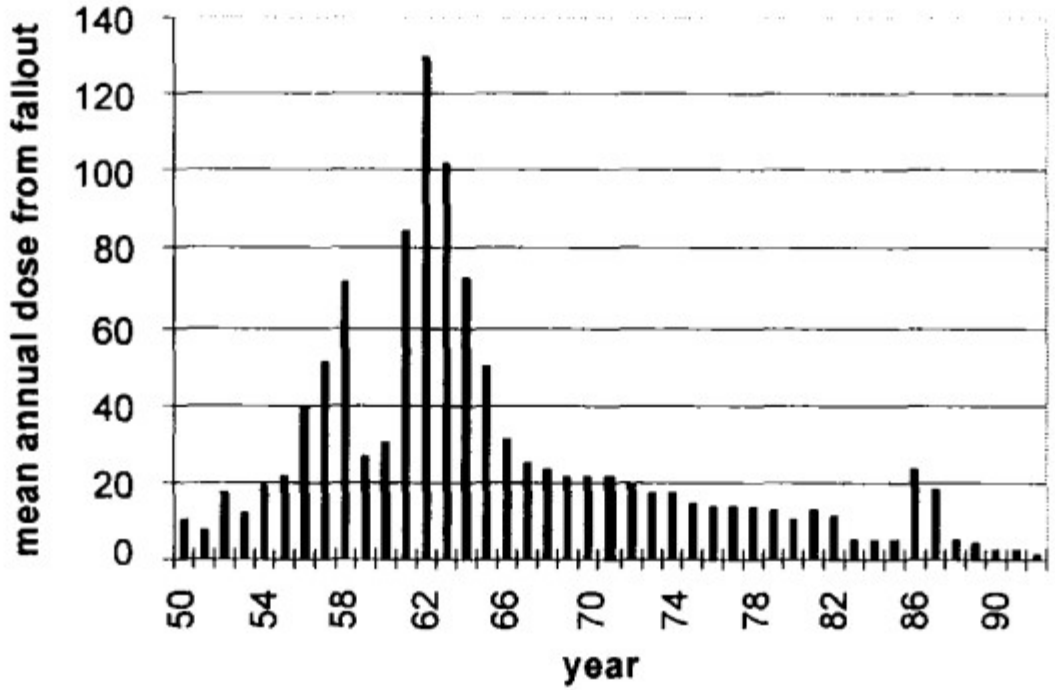

Fig. (11.1) The annual mean dose to the UK public (μSv) from radioactive fallout. The large peaks in the late 1950's and early 1960's are due to weapons testing. The smaller peak in 1986 is due to the Chernobyl accident. Although the current mean is ~ 5 μSv, areas with heavy average rainfalls receive doses up to 15 μSv [5]. With permission from NRPB.

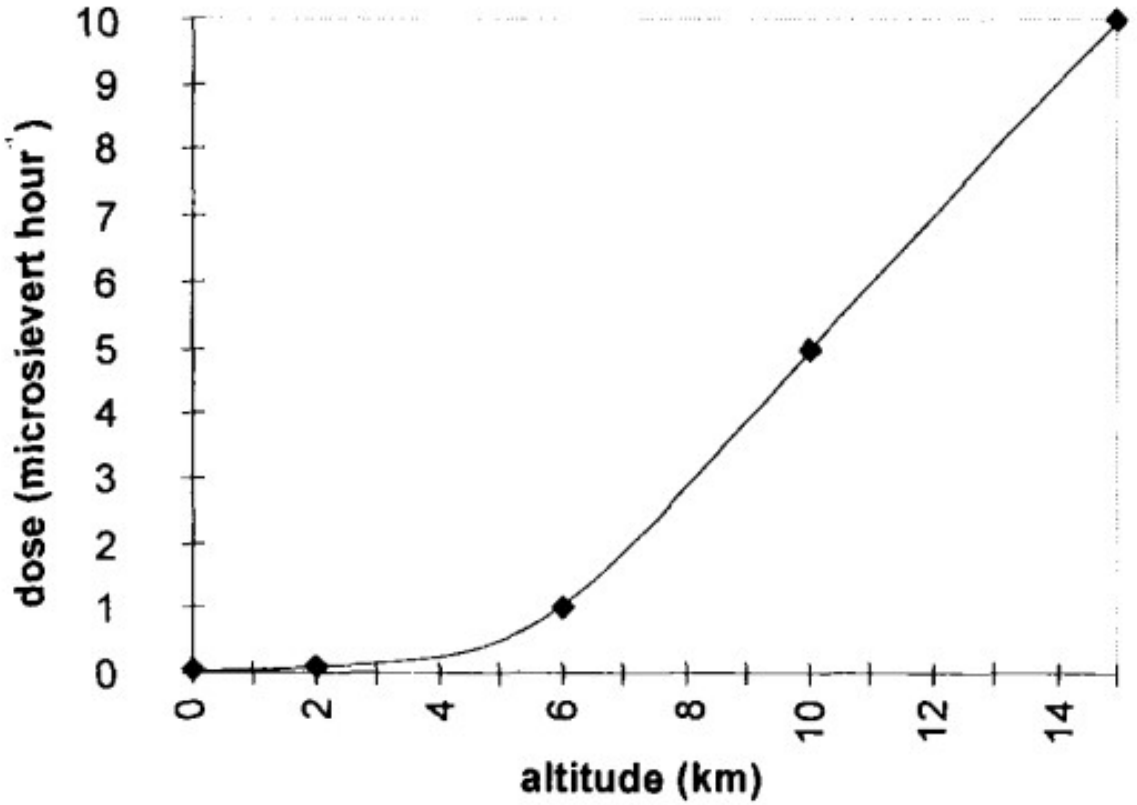

Fig.(11.2) Increase in dose due to cosmic rays (μSv hr^{-1}) with altitude. The annual dose at mean sea level varies with latitude in the range 200 - 300 μSv. Points correspond to the altitudes above mean sea level, a high altitude city such as Mexico, a Himalayan Peak, and subsonic and supersonic airliner travel. Frequent air travellers can raise their mean annual dose from cosmic rays from 260 μSv to ~700 μSv [5]. With permission from NRPB.

It is apparent from Fig.(11.2) that radiation levels for astronauts in earth orbit (~300 km) are well in excess of sea level values. Special limits are therefore likely on the Maximum Permissible Dose for annual and career-accumulated exposures [6], [7]. Doses of ~150 mSv have been measured for a six month mission in orbit.

11.4 Stochastic and Deterministic Effects of Radiation

Exposure of living organisms to radiation gives rise to two types of effect :

- The possibility, however small, that increased exposure to radiation will result in an increased probability of genetic mutations or cancer induction. These are the stochastic (random) effects of radiation and they may not become evident for some considerable time after the exposure.

- Where the severity of the response to radiation increases with dose. This is a deterministic (formerly non-stochastic) effect. In the case of a radiation accident, for example, where the doses may well have been appreciable, deterministic effects such as skin erythema (reddening and breakdown) or cataract formation become evident quite soon after the exposure. It is likely that a non-stochastic effect will also be accompanied by stochastic effects.

Table (11.3) The variation of average radon dose with region in the UK. High levels in South West and Central England and North East Scotland are due to the relatively large (but still small in absolute terms) deposits of uranium in the geological strata. The country-wide mean dose from radon is 1.3 mSv. Levels out of doors and in buildings with under-floor ventilation are considerably less than the quoted figures [5]. In any one area, radon dose can vary by several orders of magnitude.

Region	Radon Dose (mSv)	Total Dose (mSv)	%
Cornwall	6.4	7.8	82
Devon	4.0	5.3	75
Somerset	3.3	4.5	73
Northants	2.6	4.0	65
Derbyshire	2.4	3.7	65
London	0.9	2.1	43

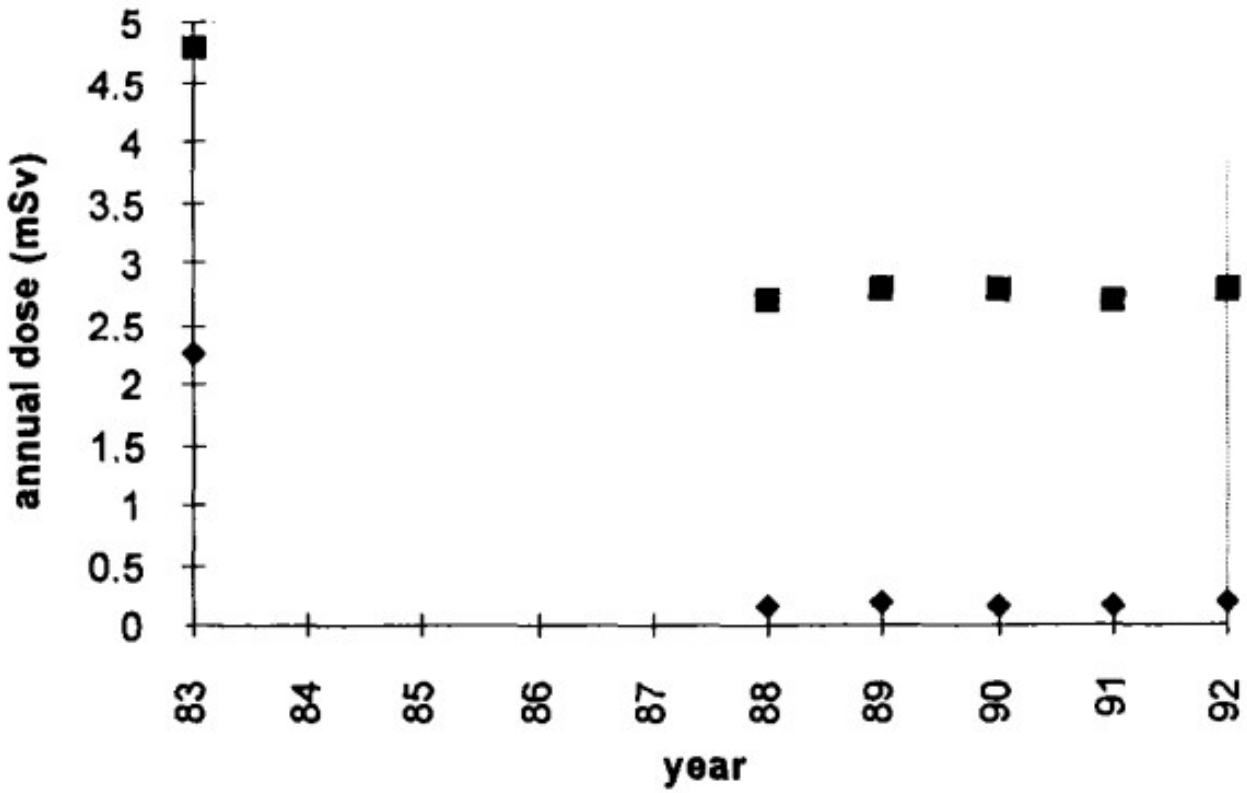

Fig. (11.3) Annual dose received by heavy consumers of seafood in Cumbria. ■ total dose: ◆ dose due to radioactive discharges from nuclear industry: The current dose burden for this group due to discharges continues to be in the range 150 - 200 μSv. The current national average due to discharges from all sources (nuclear industry and medical) is 0.4 μSv [5]. With permission from NRPB.

The delayed reaction to a stochastic effect is expressed by a risk factor (also known as a probability coefficient). These factors can be individually assigned to different conditions (leukaemia) or grouped together to include an overall category (solid cancers of all types).

If the risk factor for leukaemia induction is 3×10^{-6} mSv^{-1}, for example, then a population of 10^6 irradiated with 1 mSv would be expected to yield 3 extra cases, on average, over and above the normal incidence. Alternatively, the risk factor for the excess number of solid cancers over a lifetime in the population at large is expressed as 50×10^{-6} mSv^{-1}. The interpretation is that 50 extra patients with solid cancers would be expected in a one million population irradiated with 1 mSv. Risk Estimates agreed by both ICRP and NCRP [1],[3] are summarized in [4],[8].

An example of the progression from severe deterministic to stochastic effects of radiation was provided by a case of industrial exposure of a worker's hand. The dose to the hand was probably ~100 Gy and to the whole body ~10 Gy over a time

span of about a decade. Outward evidence of exposure was first provided by redness and swelling of the right index finger. This was followed by dermatitis and heavy infection which eventually required amputation of the finger. Several years later acute myeloid leukaemia eventually culminated in the death of the person [9].

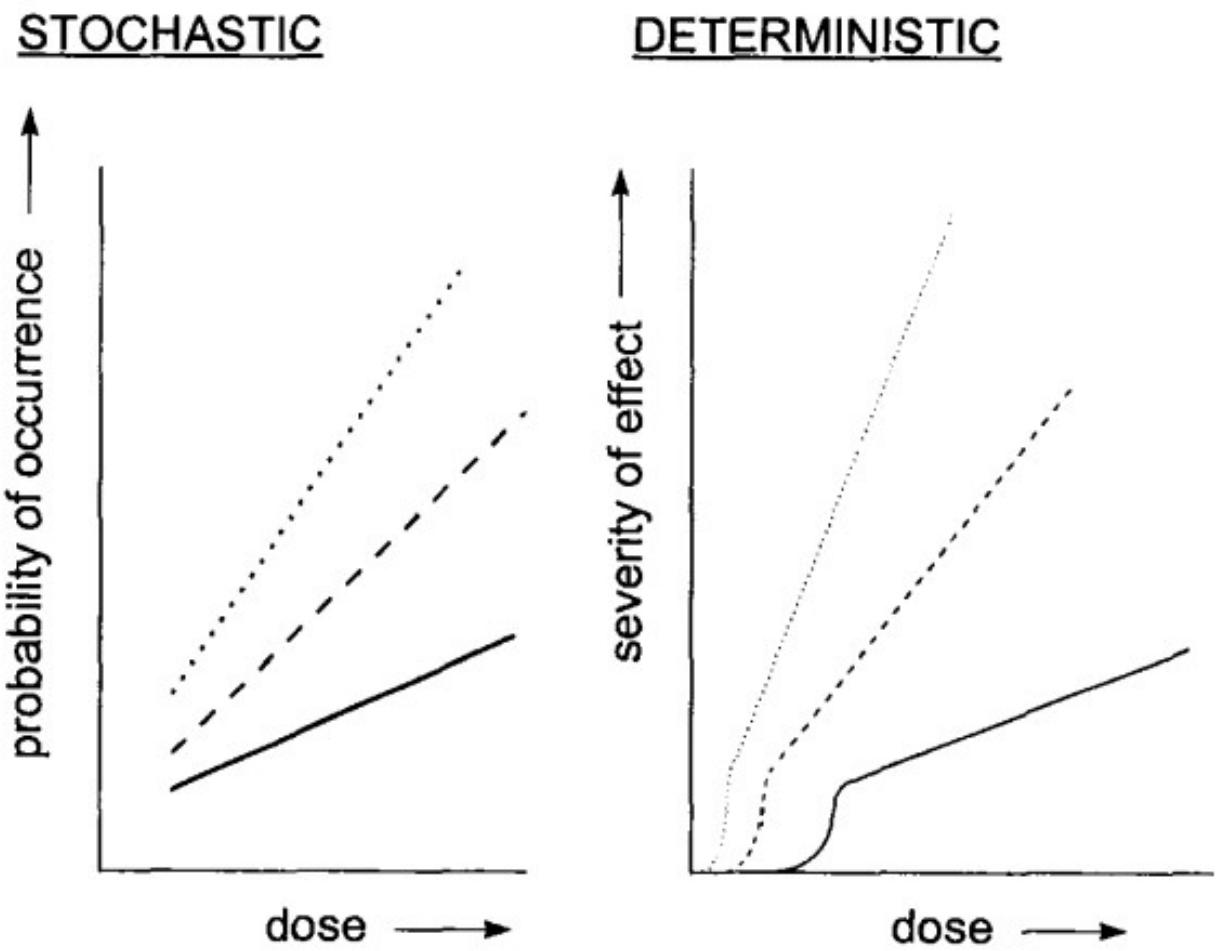

Fig.(11.4) The different lines on the stochastic graph refer to different conditions, e.g. leukaemia, chromosome aberrations, etc. Different lines on the deterministic graph refer to the response of different groups of people, e.g. to skin damage, cataract formation, etc. Note the presence of a threshold in the deterministic effects.

Table (11.4) Risk Estimates for Stochastic Effects. The figures refer to the lifetime risk of contracting one of the outcomes for all ages (Whole Population) and for the working population (Adult Worker).

Outcome	Whole Population	Adult Workers
Fatal Cancer	50×10^{-6} mSv^{-1}	40×10^{-6} mSv^{-1}
Non-fatal Cancer	10×10^{-6} mSv^{-1}	8×10^{-6} mSv^{-1}
Genetic Effects	13×10^{-6} mSv^{-1}	8×10^{-6} mSv^{-1}

11.5 Radiation Carcinogenesis

The data sets which are used as the bases of radiation protection come from a variety of sources [8],[10]. Some of these are :

- the study of disease and mortality rates of the Hiroshima and Nagasaki atomic bomb survivors,
- the survival rates of patients who had suffered from ankylosing spondylitis,
- tuberculosis patients given chest fluoroscopies,
- irradiation of children for ringworm of the scalp,
- the national registers of radiation workers.

In many of these cases, the greatest difficulty in associating the pathological condition with the radiation exposure lies in the precision of the dosimetry. The data therefore have to be complemented by controlled radiation biology experiments on both cellular and mammalian systems.

11.5.1 Dose : response relationships

The mechanisms of radiation action are a continuing area of study. Many factors are responsible for the large uncertainty between the exposure to radiation and the ensuing biological response. This is especially true at the low doses experienced under radiation protection conditions. These factors are :

- the stochastic nature of energy deposition resulting from both primary and secondary charged particles,
- the complexity of the target (ultimately the DNA) and the manner in which the energy deposited manifests itself in a pathological condition,
- the dynamic nature of the target system,

- the large number of end-points (chromosome aberrations, cell death, *etc.*).

Radio-biology experiments have established certain dose:response functions to describe the incidence of different end-points in a number of cellular as well as mammalian systems irradiated by different types of radiation. These functions are formulated on the basis of models which consider the spatial correlation between single- or multihit events within the target. These models are applied in the following circumstances :

- In radiotherapy the target is a collection of tumour cells, sometimes at a specific location and sometimes distributed throughout the body. The cell death end-point is clearly of relevance to the high doses delivered here. Analysis of cell survival is described adequately using a linear-quadratic function :

$$N = N_0 \exp\left(-\alpha D - \beta D^2\right) \qquad (11.3)$$

This gives the number, N, of surviving cells after irradiation to dose D. The coefficients α and β are generally regarded as being associated with non-repairable and repairable damage respectively, with values in the range 1—0.1 Gy^{-1} and 0.1— 0.01 Gy^{-2}.

- End-points such as point mutations and chromosome aberrations in cells might be more relevant to initiation events which eventually lead to cancer. Experiment shows that these mutational events in cells are induced with a linear-quadratic, $(\alpha_1 D + \alpha_2 D^2)$, dependence on dose. Thus, cancer induction data might be modelled using an induction term for mutational events in cells and a cell killing term as in Eq.(11.3). This results in Eq.(11.4) [8].

$$P = \left(\alpha_0 + \alpha_1 D + \alpha_2 D^2\right) exp\left(-\beta_1 D - \beta_2 D^2\right) \qquad (11.4)$$

Here, the α terms describe defect induction resulting from spontaneous, (α_0), 1-hit and 2-hit components, while the β terms describe 1-and 2-hit components for cell death.

11.5.2 Effects of dose, dose-rate and LET in cancer induction

Experimental verification of radiation action has only been obtained for doses large enough to have produced a measurable and statistically significant response. For most biological systems, however, these doses are greater than those of concern in radiation protection. Since the ultimate purpose of radiation protection is to assess the probability of cancer induction from the known dose received by an individual, it is first necessary to extrapolate the effects at high dose down to the low doses of interest. The task is then to relate all factors which influence the pathway between energy deposition in the prime target - DNA in the cell nucleus - and its subsequent expression as a cancer. This is done with a model such as Eq.(11.4).

The relevant factors to be considered are :

- Whether the action is direct. In this case the energy is deposited directly into the DNA by the primary or secondary radiation.
- Whether energy is deposited initially into a neighbouring molecule - producing an $OH^{\bullet}$ radical in water, for example - which then interacts with the DNA target. This is called indirect action.
- The efficiency of damage repair processes.
- Whether one or more hits are produced by the same primary particle in the same target molecule.

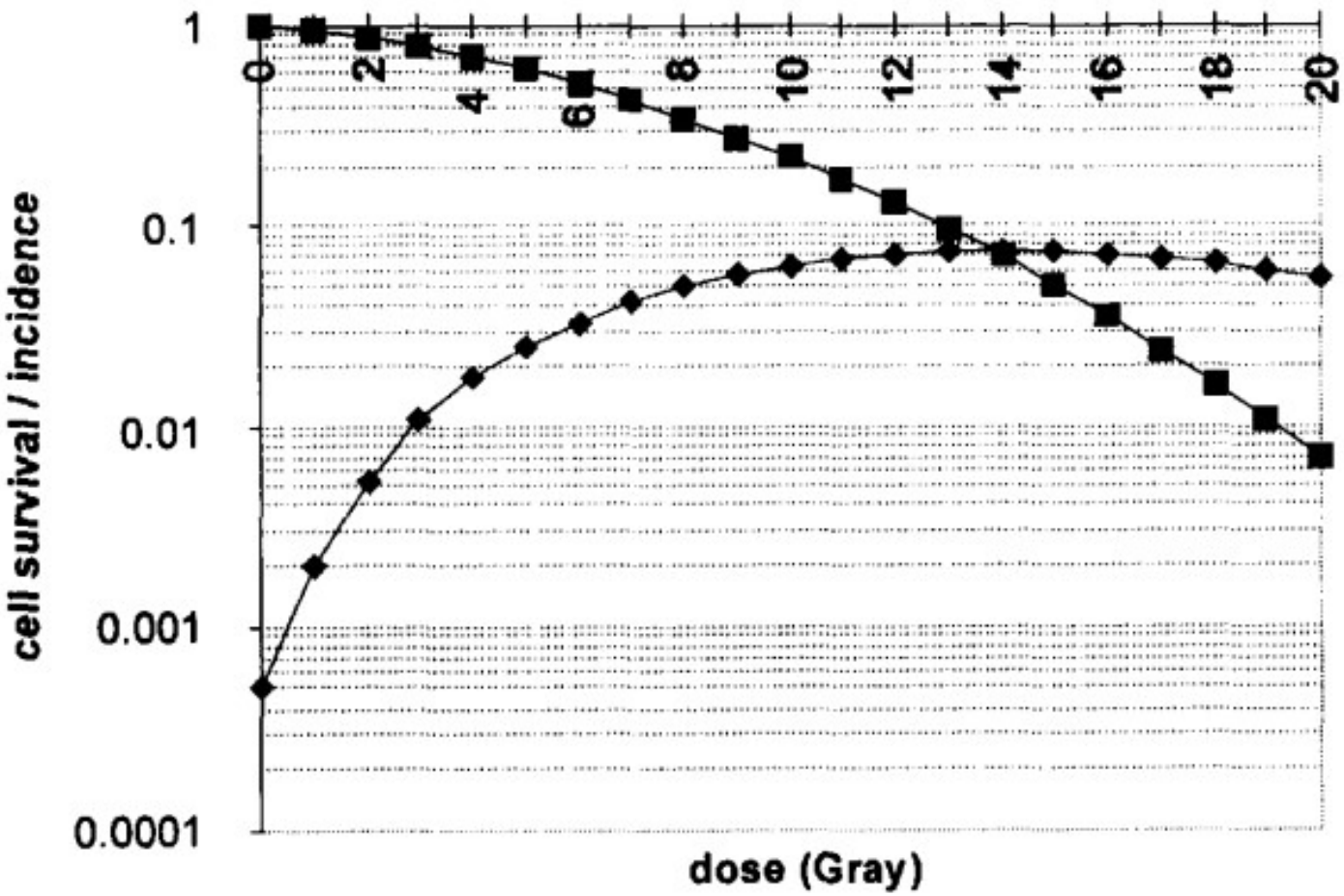

Fig. (11.5) Typical survival and incidence curves against dose (Gy) in the range 0 - 20 Gy.
(■) Cell survival using Eq.(11.3) and $\alpha = 0.05$ and $\beta = 0.005$:
(◆) Incidence of a defect and eventual cell death using Eq. (11.4) together with $\alpha_0 = 0.0005$, $\alpha_1 = 0.0005$, $\alpha_2 = 0.001$, $\beta_1 = 0.001$ and $\beta_2 = 0.005$.

The physical parameters that govern the above factors are total dose, dose-rate and LET (section 9.4). Fig. (11.6) illustrates the link between cancer incidence and the α and β coefficients in Eq. (11.4).

Most experimental evidence of cancer induction comes from high doses of low LET radiation at high dose-rate. In this case, the small probability of a multihit event comes either from low energy electron tracks which traverse a DNA strand or from a high spatial concentration of individual radiation spurs (section 6.6). The probabilities of induced effect (α terms) and cell death (β terms) are therefore both small except at large doses, as shown in curve C, Fig.(11.6). At very low doses, the induction is determined solely by the α_1 term (= 0.0002) which is generally assumed to be dose-rate independent.

However, if a linear relation were to be used to fit the high dose experimental data, as in line B, the induction at low doses would be very much higher than if only the linear term of curve C were used (line E). Thus, a dose of 2 Gy would have an induction coefficient of ~ 0.01 for line B and 0.0004 for line E. This has considerable importance in the definition of what might constitute a "safe" dose of radiation. The ratio of these slopes (line B/line E) is sometimes used as an estimate of the reduction factor to be applied to data obtained at higher doses and high dose-rates in setting risk estimates for radiation protection.

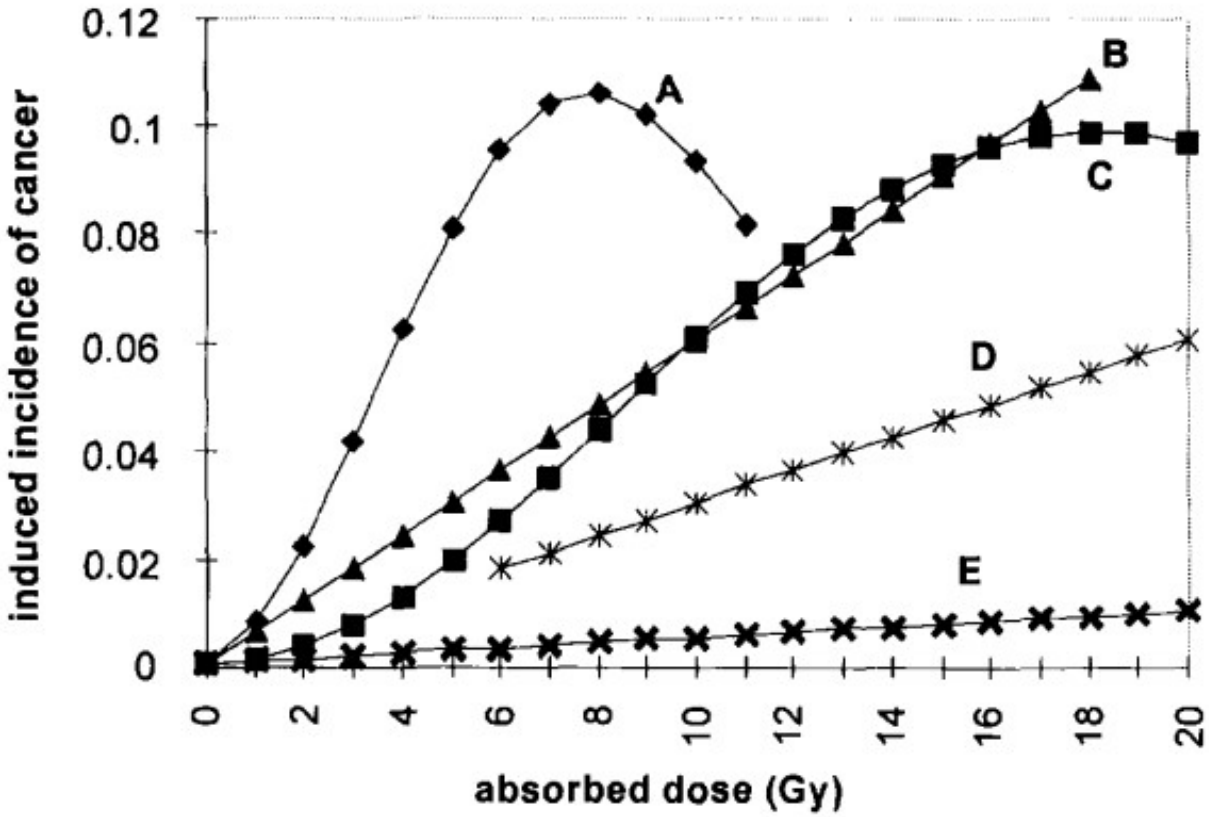

Fig. (11.6) Dose-response relationships for cancer induction using Eq. (11.4) and spontaneous incidence coefficient $\alpha_0 = 0.0005$: Data taken from [8] with permission.

◆ high LET: $\alpha_1 = 0.004$, $\alpha_2 = 0.004$, $\beta_1 = 0.005$, $\beta_2 = 0.015$. Curve A.

▲ low LET: linear, no threshold: $\alpha_1 = 0.006$. Line B.

■ low LET: high dose-rate: $\alpha_1 = 0.0002$, $\alpha_2 = 0.0008$, $\beta_1 = 0.0004$, $\beta_2 = 0.003$. Curve C.

✱ low LET: low dose rate: $\alpha_1 = 0.003$. Line D.

✖ low LET: limiting slope for low dose-rate. $\alpha_1 = 0.0002$. Line E.

Information on this critical dose:response relation at low doses can be obtained from experimental low LET studies at different dose-rates. These point to the effectiveness of the repair mechanisms which tend to reduce the biological effect following a given dose delivered at a low dose-rate, Fig.(11.7). Ultimately the dose-rate becomes so high that the damage is irreparable and this puts an upper limit on the initial slope of the dose:response relation, Fig.(11.6). Many of the experimental data which underpin present knowledge of low dose effects may have been performed at dose-rates which were not sufficiently high to reach this limit. It is therefore present practice to allocate a response function – such as line D – which has a slope (=0.003) approximately between the two extremes (0.0002 and 0.006). This factor of ~ 2 reduction for the effects of dose-rate in the extrapolation of high to low doses is referred to as the Dose-Rate-Effectiveness Factor (DREF) [8].

There is a higher probability that a densely ionizing particle traverses a DNA target molecule in high LET radiation. This then produces a greater fraction of multihit events and gives rise to much larger α_1 and β_1 coefficients in Eq.(11.4). Curve A in Fig.(11.6) indicates the likely dose:response relation. In general, a larger probability of cancer induction is accompanied by a larger probability of cell death. Cell death is the more likely consequence at high doses however. Exposure to low doses of high LET radiation (α-particles, neutrons..) are therefore more likely to induce cancer than the same absorbed dose at low LET.

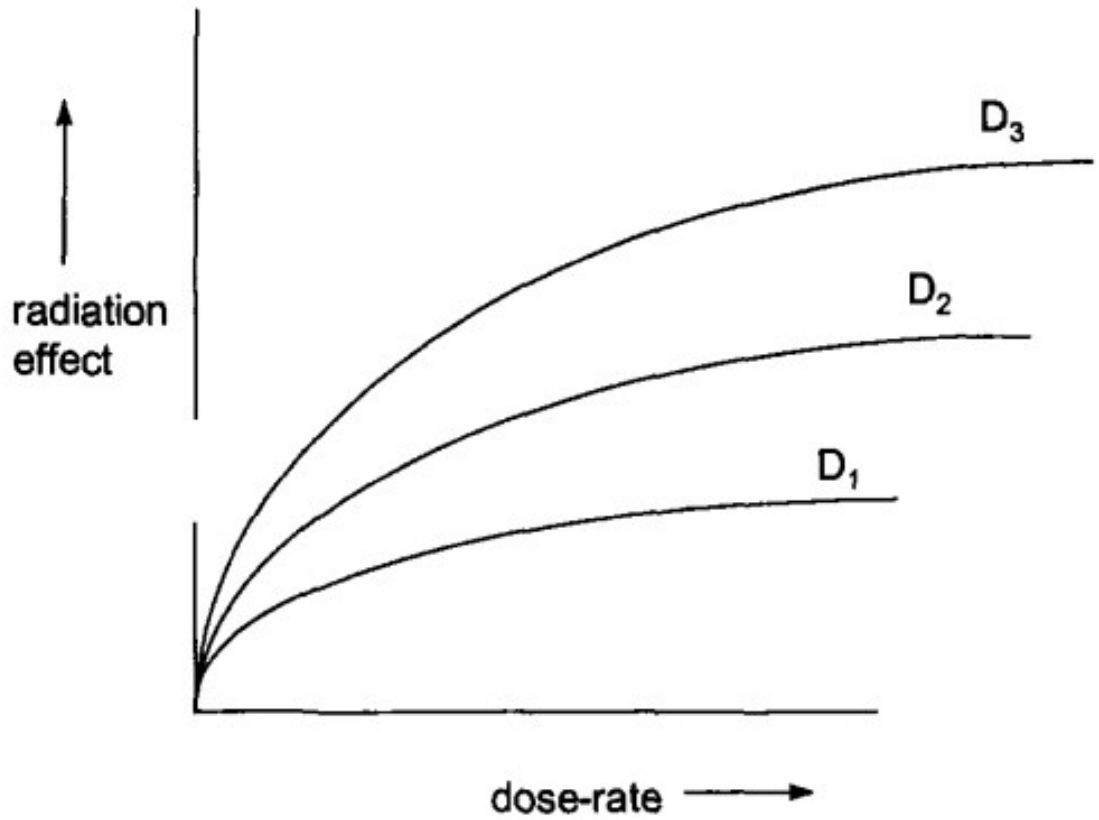

Fig. (11.7) The effect of dose-rate on the radiation response for total doses $D_3 > D_2 > D_1$. For a given total dose at low dose-rates, repair mechanisms are able to reduce the biological response. Eventually the dose-rate becomes so high that the biological damage becomes irreparable.

The existence of protective, as distinct from repair, mechanisms has been studied by a small number of workers. If such processes are initiated by radiation exposure there is the possibility that low doses of radiation may be life enhancing. This effect is known as Radiation Hormesis. There is no general acceptance that available data can be interpreted in these terms.

11.6 Maximum Permissible Levels of Exposure

The principles of radiation protection are enshrined in the following :

- Any practice using ionizing radiation must be justified. The advantages must outweigh the disadvantages and there must be no other way of achieving the stated objective.
- Doses both to workers and to the public must be optimized. This means that doses must be reduced until it is no longer economic to reduce them further.
- Doses must be below the prescribed limits which are judged to be at the boundary between acceptability and intolerability. If the dose limits cannot be met the practice cannot be justified.

Recommended dose limits are set at a judged level of risk. The two categories for which exposure limits are set are :

- the public at large, and
- those who receive exposure through their occupation.

Evidence of detrimental effects of radiation exposure are gathered from many sources before assessment by a number of national (NRPB in the UK, NCRP in the US) and international (ICRP) bodies. Reports are issued periodically to suggest the adoption of limits of exposure for the two categories above [1],[3]. These are summarized in Table (11.5) [4].

The recommended dose limits are set at a judged level of risk. These are that a worker should not be expected to accept a lifetime risk of cancer greater than 3 - 4 % while a member of the public should not have to accept a risk greater than 0.3 - 0.4%. Using risk estimates for fatal cancer from Table (11.4) and the lower of these numbers in each case, we have :

- Workers with a working life of 40 years: 0.03 = (40 years) × (40 × 10^{-6} mSv^{-1}) × (dose limit), giving a dose limit of ~ 20 mSv yr^{-1}.
- Member of the public with a life-span of 70 years: 0.003 = (70 years) × (50 × 10^{-6} mSv^{-1}) × (dose limit), giving a dose limit of ~ 1 mSv yr^{-1}.

11.7 Practical Methods of Reducing Dose

The three main principles of protection from an external (to the body) source of radiation are :

- to minimize the time spent by the person in the vicinity of the source,
- to maximize the distance between the person and the source,
- to use sufficient and appropriate shielding material between the source and the person.

While the precepts of dose reduction are self-evident in many ways, judgement is always necessary in selecting the most appropriate line of defence from exposure. For example, in seeking to keep the total exposure within permissible levels :

- long handled tongs may be used to increase the distance between a worker and a task which needs to be performed on or near an intense source. If the task is so delicate or precise and the tongs insufficiently manipulative that the worker spends a considerable length of time making unsuccessful attempts, it may be less detrimental to forego the use of tongs and decrease the distance

  for the shorter time it takes to carry out the task by hand.
- the appropriate selection of shielding, if employed, is paramount. Secondary radiation can be produced in the shield material itself and reduce its effectiveness in contributing to overall protection.

Points to remember when selecting the most suitable shielding material for the following different types of radiation are :

- β-particles and electrons. The higher the energy the more likely it is that bremsstrahlung photons will be produced. The most appropriate initial shielding is therefore low Z material (e.g. perspex) to degrade the electron energy by collision loss. Secondary shielding of lead can then be used if necessary to provide further protection against photons.

- Positrons. Positrons of whatever energy will always be a source of annihilation photons at 511 keV. Lead shielding is most effective.
- Neutrons. These should first be moderated to thermal energies using material with a high concentration of H or C atoms. A layer of B, Cd or Gd, all of which have high absorption cross-sections for thermal neutrons, then absorbs the neutron flux. Both of these functions are sometimes combined by the use of borated polyethylene or similar. Secondary γ-rays, both prompt and decay, are then generated. These are attenuated using a further layer of lead or concrete shielding.
- Photons. Shielding against MeV photons is most easily (and less expensively) achieved using high density concrete rather than lead. This is because MeV photons interact primarily by Compton scatter and not photoelectric absorption. A large electron density is therefore more appropriate than a high Z nucleus.
- Photon scatter is most easily reduced by minimizing the amount of high Z material visible to the source and by the avoidance of a direct line of sight. This technique is employed in the construction of interlocking maze entrances to linac rooms.

Table (11.5) Summary of Maximum Permissible Levels of Exposure for the public at large and occupationally exposed radiation workers from [1] and [3].
All limits apply. The limits to specific organs are necessary to prevent deterministic effects to some organs even when effective dose is not exceeded.

Parameter	ICRP 60 [1]	NCRP 116 [3]
<u>Public</u>		
Effective Dose:		
Annual	1mSv: 5y average $\leq$ 1 mSv	1 mSv: if continuous 5 mSv: if infrequent
Equivalent Dose:		
Annual	15 mSv: eye lens 50 mSv: skin, hands, feet	15 mSv: eye lens 50 mSv: skin, hands, feet
<u>Occupational</u>		
Effective Dose:		
Annual	50 mSv	50 mSv
Cumulative	$\leq$100 mSv in 5 yr	10 mSv $\times$ age (yr)
Equivalent Dose:	150 mSv: eye lens	150 mSv: eye lens
Annual	500 mSv: skin, hands, feet	500 mSv: skin, hands, feet