RADIATION PROTECTION NOTE NO 4: RADIATION DOSE AND THE BIOLOGICAL EFFECT OF IONISING RADIATION

MEASUREMENT OF "RADIATION DOSE"

In Note No 2 we have seen that a lightly ionising charged particle, such as a beta particle (or the photo-electron or Compton electron produced by a gamma ray photon) ionises about one in a thousand of the atoms in its path. An alpha particle on the other hand ionises practically every atom it meets. The situation where we irradiate a cell nucleus in biological material is depicted schematically in Figure 1 with dots representing ionisation events.

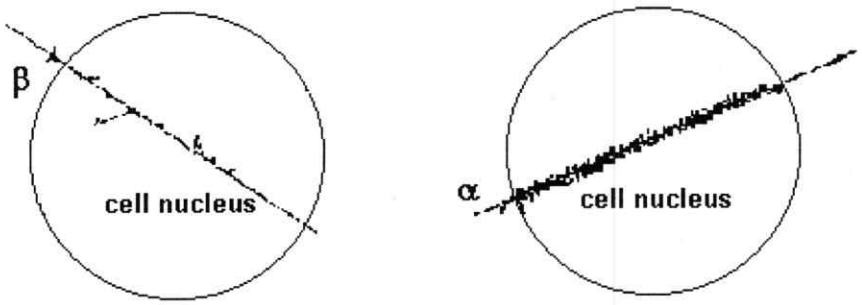

Figure 1

(a) β - particle, 50 ionisations/nucleus
Radiation dose 1 mGy*

(b) α - particle, 12500 ionisations/nucleus
Radiation dose 250 mGy*

* *One ion track per cell nucleus in irradiated tissue is produced by a radiation dose of approximately 1 mGy of beta radiation and 250 mGy of alpha radiation.*

As mentioned previously, ionisation of a molecule invariably leads to its disruption producing the "direct" damaging effect of ionising radiation.

However, there is also an "indirect" effect. The fragments of molecule produced by the ionisation are 'free radicals' and these may diffuse away from the site of their production and attack other important molecules such as DNA in the cell nucleus. It is probable that this effect depends upon the concentration of the free radicals so that the indirect effect is perhaps more important in (b) above than in (a).

We see that, as a first approximation, the amount of biological damage will be proportional to the number of ionisations produced and it might be sensible to consider the number of ionisations per unit mass as a measure of radiation dose. It turns out that the average energy required to produce an ionisation does not depend greatly on the energy of the radiation or its type, so a measurement of the dissipated energy/unit mass by the incoming radiation is also a measure of the number of ionisations per unit mass. We call this quantity - energy dissipated per unit mass the **Absorbed Dose, D_T**.

The unit of Absorbed Dose is the **gray** (Gy).

1 **gray** = Dissipation of 1 Joule/kg

When we perform experiments on the amount of damage produced in biological systems by ionising radiation, we find that comparing the results of 1 gray of Cobalt-60 radiation and 1 gray of alpha radiation on a cell culture, a particular damage effect is fifteen times more pronounced in the case of the alpha irradiation. A similar experiment comparing the effects of the two types of radiation on the blood forming cells of mice might indicate that alpha radiation is twenty-two times more effective.

In the two experiments, we would say that the relative biological effectiveness (RBE), of the alpha radiation is 15 and 22 respectively. Reviewing all the experiments of this type, the International Commission on Radiological Protection (ICRP) in 1990 assigned a Radiation Weighting Factor "W_R" of 20 to alpha radiation. Formerly, this quantity was called the "Quality Factor". Table 1 overleaf lists the values of W_R assigned to the various radiations.

Radiation Type and Energy	W_R
Photons, all energies	1
Electrons, (beta particles), muons, all energies	1
Neutrons < 10 keV	5
" 10 keV – 100 keV	10
" > 100 keV – 2 MeV	20
" > 2 MeV – 20 MeV	10
" > 20 MeV	5
Alpha particles, fission fragments	20

Table 1: Radiation Weighting Factors, W_R

It is seen that an appropriate measure of the biological damage sustained would be obtained by multiplying the absorbed dose D_T in a tissue T by the radiation weighting factor W_R.

This new quantity is called the **Equivalent Dose**, H_T.

ie, $H_T = W_R D_T$

The unit of equivalent dose, H_T, is the **sievert** (Sv)

ie, 1 Gy of beta radiation => 1 Sv
1 Gy of alpha radiation => 20 Sv

We will see later that where an individual has received a "whole body" dose of radiation we can make an estimate of the probability that that individual may eventually die as a result of a radiation induced malignancy such as leukaemia.

How do we cope with the situation where only a part of the body is irradiated? To make a similar probability calculation we introduce the concept of **Effective Dose**. Using data obtained from a study of hospital patients exposed to therapeutic doses of ionising radiation, the ICRP has established the relative radio-sensitivity of most organs in the body and has introduced a Tissue Weighting Factor, W_T.

We define Effective Dose, E, as the weighted sum of the equivalent doses, H_T, received by all the organs (T) in the body, each organ equivalent dose being weighted by its appropriate factor, W_T.

In mathematical terms, Effective Dose, $E = \sum_T W_T H_T$

The values of W_T assigned by ICRP in their 1990 review of the data are given in Table 2 overleaf.

Summing all the tissue weighting factors in Table 2 gives unity, so a given whole body dose (ie where each tissue in the body receives the same dose) has the same magnitude as the calculated "effective dose" and of course both are measured in Sv.

We use "effective dose" where there is non-uniform or partial irradiation of the body. For example, many radioisotopes when ingested or inhaled are concentrated in particular organs. If we know the metabolism of the chemical involved and its transfer between the different compartments of the body, we can calculate the "effective dose" for any given intake.

A very simple case is the inhalation of radon. Each year inhalation of radon in the air results in an "equivalent dose" to lung tissue of 10 mSv. The "effective dose" in this case is 10 x 0.12 = 1.2 mSv, ie, the probability of future death from lung cancer as a result of this annual effective dose from radon is the same as that which would result from an annual whole body dose of 1.2 mSv.

Tissue or Organ	W_T
Gonads	0.20
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
Bone surface	0.01
Remainder	0.05

Table 2: Tissue Weighting Factors, W_T (ICRP 60)

THE EFFECTS ON HEALTH OF EXPOSURE TO IONISING RADIATION

Two situations arise. In the first case we are concerned with the clinical effects that result from exposure to ionising radiation. The radiation doses involved here are usually substantial and delivered over a short space of time and there is a threshold dose below which no clinical effect is observed: these type of effects are called deterministic. In the second case, we have the situation where there is only a probability of the effect occurring. It is a stochastic or chance process - induction of malignancy and hereditary effects come into this category. Unlike the deterministic effect, there is no threshold dose below which the probability is zero and in the simplest approach we adopt the hypothesis that "probability" is proportional to dose received.

Deterministic Effects

An example of this type is the reddening of skin (erythema) following exposure to ionising radiation. Radiation doses of less than 3 mGy produce no observable effect but once this dose is exceeded, the effect is more or less certain to occur hence the name deterministic. Table 3 below lists some of the other effects that occur following acute radiation exposure ie, large radiation doses received over a short time scale. Generally speaking, cells which have the most rapid turnover are the most radiosensitive and so radiation sickness due to irradiation of the intestines and changes in the blood picture are early signs of exposure to ionising radiation.

Dose (Sv)	Clinical Effect
0 → 0.25	No obvious injury
0.25 → 0.50	Possible blood changes
0.50 → 1.00	Blood cell changes, some injury, no disability
1.00 → 2.00	Injury, possible disability, nausea/vomiting within 24 hrs
2.00 → 4.00	Injury and disability certain, death possible
> 4.00	50% probability of death

Table 3: Clinical Effects of Acute Radiation Exposure (ICRP 60)

Stochastic Effects

When we compare the health of a population of smokers with a population of non-smokers, we find that the smokers have a higher incidence of lung cancer and the more cigarettes smoked, the greater your probability of contracting the disease. There is no certainty, however, that heavy smokers will succumb to the cancer.

The same scenario is found to apply in the case of exposure to ionising radiation. Extensive study of the health of the survivors of Hiroshima and Nagasaki, and some other groups exposed to relatively large radiation doses, enable us to evaluate the probability of death at a later date from radiation induced cancer for a given radiation dose.

Two approaches are possible. We can say that a given dose produces a risk that is constant with time - this is the additive model, or we can say that a given dose produces a risk which is a constant multiple of the pre-existing spontaneous risk of cancer – the multiplicative model. Until relatively recently, the additive model was the most extensively used but since the 1990 review (ICRP 60), the second multiplicative model has been used.

At high radiation doses, ICRP calculate that where a general population of one thousand people are exposed to a whole body dose of 1 Sv, one hundred cases of fatal cancer will occur in succeeding decades. Where a working population is exposed, the corresponding figure is eighty fatal cancers. The "spontaneous" incidence of cancer is highest in later life and it is then that most of the radiation-induced cancer will appear according to the multiplicative model.

Among the Japanese bomb survivors it is possible to determine that for fatal leukaemia, the risk per Sv for those receiving doses less than 0.5 Sv is half that of those receiving doses of 1 to 2 Sv. We also know from some laboratory animal experiments that the risk per Sv is much less at low doses and dose rates. Accordingly, ICRP introduced a factor called the DDREF (Dose and Dose Rate Effectiveness Factor) for assessing the risk of fatal cancers occurring amongst the general population and radiation workers. It is only applicable in the case of lightly ionising radiation such as beta, X and gamma radiation. Using the DDREF, ICRP estimate the risk of fatal cancer for a radiation worker is 40 per 1000 per Sv ie, 4% per Sv.

In 1990, ICRP also assessed the risk to health from radiation induced non-fatal cancers. Non-fatal cancers are weighted by the lethality faction for that cancer on the grounds that the higher the lethality, the lower the quality of life for those who survive. On this basis, the risk of non-fatal cancer for radiation workers is estimated to be 1.2% per Sv.

Irradiation of the gonads can produce changes in the genetic material with serious consequences for future generations. This is also a stochastic effect. Studies of exposed human populations provide no estimate of the risk of radiation induced hereditary disease and estimates are therefore made from studies on laboratory animals.

It is interesting that a careful study of ten thousand children of the Hiroshima/Nagasaki survivors did not reveal any departure from the normal incidence of hereditary disease. Using the animal data it is estimated that, following irradiation in reproductive life, the incidence of serious hereditary disease in all future descendants will be twelve per thousand per Sv of which three cases would occur in children and grandchildren. For a working population, there is a different age distribution, and the average risk is six per thousand per Sv, ie, 0.6% per Sv for hereditary defect in all succeeding generations.

Taking the risk figures for (i) fatal cancer 4% per Sv, (ii) non-fatal cancer 1.2% per Sv, iii) hereditary disease 0.6% per Sv, we arrive at a risk factor for detriment to health of 5.8%

per Sv. This can be compared with the corresponding figure of 1.65% per Sv in the 1977 ICRP recommendations (ICRP 26) which did not include detriment for non-fatal cancer, and only considered risk of hereditary disease in children and grandchildren.