

Introduction:

As stated by the International Commission on Radiological Protection (ICRP), the primary aim of radiological protection is to “provide an appropriate standard of protection of humans against ionizing radiations without unduly limiting the beneficial practices giving rise to radiation exposure”[1].

Radiological Protection and the practice of Health Physics is concerned with protecting man against the harmful effects of radiation. This course focuses on principles and techniques to be applied in the practice of Health Physics, but it is useful start with a review of the harmful effects of ionizing radiation.

Recently, there has been extensive discussion and debate regarding the expansion of the scope of radiological protection to “non-human biota” – but this aspect of radiological protection is outside the scope of this course.

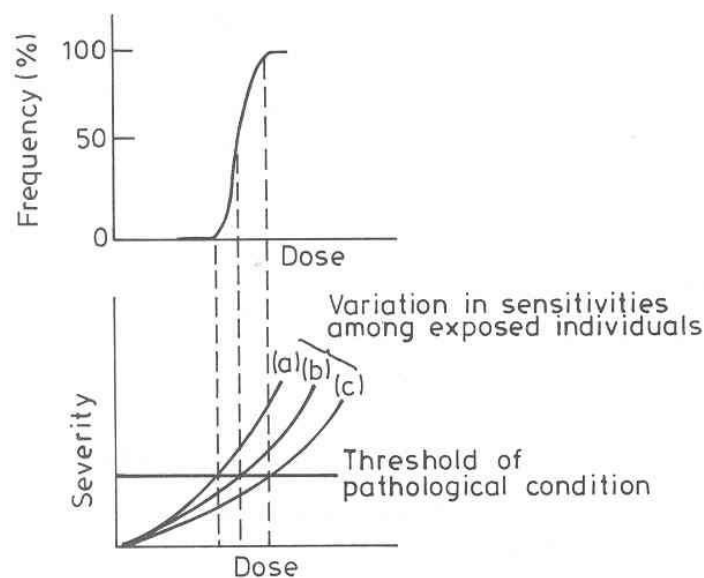
Harmful Effects

Harmful effects of radiation exposure in man are broadly divided into two groups – Deterministic and stochastic effects

Deterministic Effects

Deterministic effects result from cell killing that leads to clinically detectable impairment of the function of a tissue or organ. This the main processes involved in deterministic effects but radiation can also interfere with a variety of tissue functions adding to the severity of the deterministic effect. There is a threshold of radiation exposure below which the loss of cells is too small to result in a clinically observable effect. Below the threshold – the deterministic effect does not occur. Above the threshold, the surviving fraction of cells decreases and the severity of the effect increases. Deterministic effects are sometimes described as “radiation injuries”.

Note that the threshold for a given pathological condition varies from individual to individual – giving rise to an overall “S” shaped dose response curve in the population. This is illustrated in the following figure from ICRP 60



Source – ICRP 60

Deterministic effects can be avoided by keeping exposures below the corresponding threshold. As this is one of the main goals of radiobiological protection practices, deterministic effects are typically associated with comparatively large and (usually) acute exposures encountered in accidents.

Effects can be early or late, depending on the tissue(s) irradiated. Cell death normally occurs at the time the cell next attempts to divide. Irradiation of tissues such as bone marrow with rapidly dividing stem cells leads to early manifestation of effects. Irradiation of tissues with low rates of cell division, such as the liver, leads to late effects.

Examples of thresholds for deterministic effects in radiosensitive tissues are provided in the following table from ICRP 60 [3].

Deterministic effects in the skin are another category that Health Physicists should be aware of. The following is summarized from ICRP 59.

Estimates of Thresholds for Deterministic Effects in the Skin of Adult Humans

Effect	Threshold	Notes
Erythema or dry desquamation	3 – 5 Gy	Symptoms appear after about three weeks. Early erythematous reaction may be seen within a few hours and will subside in 24 – 48 hours.
Moist desquamation	20 Gy	Blistering after about four to six weeks
Tissue necrosis	50 Gy	Appears after about 3 weeks

Estimates of Thresholds for Some Deterministic Effects in Adult Humans (from ICRP 60)

Tissue and Effect	Threshold		
	Total dose equivalent received in a single brief exposure (Sv)	Total dose equivalent received in highly fractionated or protected exposures (Sv)	Annual dose rate if received yearly for many years (Sv)
Testes			
Temporary sterility	0.15	NA	0.4
Permanent sterility	3.5-6.0	NA	2.0
Ovaries			
Sterility	2.5-6.0	6.0	>0.2
Lens			
Detectable opacities	0.5-2.0	5	>0.1
Visual impairment	5.0	>8	>0.15
Bone Marrow			
Depression of hematopoiesis	0.5	NA	>0.4

Acute Radiation Syndrome and Early Death after Acute Whole Body Exposure

Acute whole body exposures may lead to a variety of syndromes, depending on the magnitude of the exposure. The following table summarizes the various syndromes and the associated dose range.

Table 15.1 Acute Radiation Syndrome
(After IAEA 88 1988)

Grouping	Range of Doses (Gy)
1. Hematopoietic	1–10
a. From 0 to 0.25 Gy: No clinical symptoms but a slightly increased frequency of chromosome aberrations may be detected in lymphocytes.	
b. From 0.25 to 1 Gy: Either no symptoms or transient nausea. Biological tests may reveal a lymphopenia accompanied in some cases by slight thrombopenia. Cytogenetic changes in lymphocytes are readily detected. Some studies have shown slight changes in the patient's electroencephalogram.	
2. Gastrointestinal	10–20
3. Cardiovascular or Toxemic	20–50
4. Nervous System	Above 50 Gy

Source - Health Physics and Radiological Health Handbook

For doses below 1 Gy to a population of individuals, no individuals would be expected to die. As the dose is increased, more and more individuals would be expected to die. The Survival-dose response can be described by three values: The $LD_{5,60}$, $LD_{50,60}$, and $LD_{95,60}$ – the doses at which 5, 50 and 95% of the population would be expected to die within sixty days of exposure, respectively. For a healthy adult human, the $LD_{50,60}$ is 3 – 5 Gy (without medical intervention).

Stochastic Effects

Two general types of stochastic effects are well recognized – the first results in potential cancer induction in the irradiated person and the second results in potential hereditary disorders in the progeny of the irradiated individual.

Hereditary effects have not been observed in any human population but are believed to be possible based on animal (mainly mouse) and cell culture experiments. Observations of the Japanese survivor progeny provide only upper bounds to the risk estimate. Risks of hereditary effects are often based on the “doubling dose method”. This is the dose necessary to produce as many mutations as those that occur naturally in a generation. The current best estimate of doubling dose as described in ICRP 60, based on mouse data, is 1 GY. This is also the lower 95% confidence value for the estimate based on the absence of findings in the progeny of the Japanese survivors.

Induction of cancer by low dose, low dose rate irradiation cannot be directly observed. The effect is too small to be observed amongst the other variables in cancer rates. Thus, information has been extrapolated from high dose, and typically high dose rate, situations where effects are clearly observable. By far the most important group studied is the Japanese A-bomb survivors.

The observed dose response relationship in highly exposed individuals is thus extrapolated down to provide estimates of the risk of harmful effects at occupational levels of exposure, making allowance for the assumed increase in the slope of the dose response curve at higher dose and dose rates. The assumptions in the extrapolation are:

- There is no threshold associated with the induction of stochastic effects
- The risk of stochastic effects increases proportionally with exposure.

This is the “Linear No-Threshold” or “LNT” model. It is the subject of a great deal of controversy and debate. However, it is the risk model recommended by the ICRP and effectively incorporated into Canadian law and it is the model that the professional health physicist should base judgments and advice on in occupational and public radiological protection.

As mentioned, no threshold associated with stochastic effects. They are random in nature, initiated by damage to DNA in a small fraction of the irradiated population of somatic (carcinogenesis) or germinal (hereditary effects) cells.

In the case of carcinogenesis, DNA damage is the initial event in a multi-stage (poorly understood) process that may lead to cancer after a latency period. The minimum latency period for acute myeloid leukemia is two years and the mean is about eight years. For solid cancers the minimum latency period is of the order of 5 to 10 years and the mean is of the order of 16 to 24 years.

Detriment

Detriment is the conceptual value proposed by the ICRP to reflect the overall harm to an exposed population arising out of stochastic effects. The ICRP recommendations are based on limiting the total detriment to an exposed population. Detriment is taken to have four components:

- The risk of fatal cancer in all relevant organs
- Allowance for different values of latency leading to different values of life lost
- Allowance for morbidity resulting from induced non-fatal cancers
- Allowance for risk of serious hereditary disease in all future generations

Relative Contribution of Organs to the Total Detriment

Organ	Prob. of fatal cancer F per 10 ⁴ people/Sv	Relative length of life lost (l/L)	Relative non-fatal contribution (2-k)	Product F(l/L)(2-k) per 10 ⁴ people/Sv	Relative contribution
Bladder	30	0.65	1.50	29.4	0.040
Bone Marrow	50	2.06	1.01	104.0	0.143
Bone Surface	5	1.00	1.30	6.5	0.009
Breast	20	1.21	1.50	36.4	0.050
Colon	85	0.83	1.45	102.7	0.141
Liver	15	1.00	1.05	15.8	0.022
Lung	85	0.90	1.05	80.3	0.111
Oesophagus	30	0.77	1.05	24.2	0.034
Ovary	10	1.12	1.30	14.6	0.020
Gonads ¹	100	1.33		133.3	0.183
Skin	2	1.00	2.00	4.0	0.006
Stomach	110	0.83	1.10	100.0	0.139
Thyroid	8	1.00	1.90	15.2	0.021
Remainder ²	50	0.91	1.29	58.9	0.081
Total	500			725.3	1.000

¹ Severe hereditary effects.

² The remainder consists of adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus.

RISK FACTORS

As mentioned, the major source for estimating the risk factors for stochastic effects is the study of the Japanese A-bomb survivors. Statistically significant (95% level) excess number of cancers can be found only at doses exceeding about 0.2 Sv. These doses were accumulated at very high dose rates. Data have also been obtained from patient treatment studies, but it is not clear that these groups are representative of the general population. Other groups are the early Ra-226 workers and uranium miners. However, the dosimetry is not certain, exposure to other carcinogens may have occurred, and the doses were local and from alpha particles.

The conservative approach is to assume a linear relationship without threshold for stochastic effects and dose. Allowances are made for the fact that in radiation protection we are concerned with protracted low doses over time, whereas the Japanese population received an acute dose.

For most types of cancer, the excess mortality seems to exhibit a latent period and then have the same pattern in time as the naturally occurring cancer. If this is true, then there will be a simple proportion between the natural cancer mortality and the excess due to radiation for the whole time after the latent period. This model is referred to as “the multiplicative risk projection model”. An alternative model is the “additive risk projection model” that assumes the excess mortality is independent of the natural mortality. After the initial latent period, the rate would rise over a number of years and then remain constant, or in the case of leukemia and bone cancer, fall.

The fact that the relative risk model fits the data better does not imply any particular mechanism for the induction of cancer and no deep biological conclusions should be inferred. Use of the relative risk model greatly increases computational difficulties. It is necessary to know the age (and sex) distribution of the population considered, and the natural incidence of the various cancers as a function of age and sex. The effects of smoking should also be taken into account since this alters the distribution and incidence of cancers.

The risk factors adopted by the ICRP in 1990 are given below.

Exposed population	Detriment (10^{-2} Sv^{-1})			Total
	Fatal cancer	Non-fatal cancer	Severe hereditary effects	
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

References:

1. Harvey, Physics 776 Course Notes, McMaster University, 1998.
2. ICRP 75, General Principles for the Radiation Protection of Workers, Annals of the ICRP Volume 27 No. 1, 1997.
3. ICRP 60, 1990 Recommendations of the International Commission on Radiological Protection, Volume 21 No. 1-3, 1991.
4. Shleien, B., The Health Physics and Radiological Health Handbook, Scinta Inc., 1992