Radiobiology Principles

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Ionising radiation has the potential to disrupt the structure of organic molecules in cells. All cell organelles can be damaged by radiation, but the principal target of radiation exposure is the genetic material of the cell, the DNA[1,2]. Three major types of radiation-induced DNA lesions occur in an irradiated cell: base damage (BD), single strand breaks (SSB) and double strand breaks (DSB). The critical lesion is the DSB, because it leads to a disruption of the DNA molecule. Cells use specialised signalling pathways to sense, respond to and repair DNA damage[3]. About half of radiation-induced DSB are misrejoined, which may lead to changes in the gene sequence of a DNA strand. Moreover, 30-40% of DSB induced after gamma radiation are complex and this type of damage creates serious problems for the DNA repair machinery with an increased probability of misrepair. Epigenetic changes can also be induced by radiation exposure. These are heritable changes in gene expression such as DNA methylation or acetylation/methylation of residues in histone proteins which wraps up the DNA[4]. Irradiated cells may die or survive with mutated DNA. The mechanisms of death are the active, programmed death, termed apoptosis, or in cases of high radiation doses and especially lack of oxygen or energy, by necrosis. Cells can also go into permanent cell cycle arrest, senescence.

The effects of ionising radiation at the level of tissues and organisms can be divided into deterministic and stochastic events[1,2,5]. Deterministic effects originate from cell death events. If a high number of irradiated cells die, this will lead to necrosis of the tissue. Hence, deterministic effects show a dose threshold which corresponds to the dose which kills a sufficiently high number of cells for the tissue to break down. Stochastic effects originate from cells which survive a dose of radiation with mutated DNA which, in turn, can lead to neoplastic transformation. They are probabilistic in nature in that it is impossible to predict whether a particular cell will carry a mutation or not. Consequently, stochastic effects have no threshold of dose[5].

The principles of radiological protection are developed by the ICRP[5]. ICRP assumes that there is no "safe" dose of radiation below which the risk of stochastic effects is zero. This dose-response model is generally known as "linear-non-threshold" or LNT. Whilst the LNT model remains a scientifically plausible element in its practical system of radiological protection, its adoption is to a large extend guided by the requirement to follow the precautionary principle which is generally used in health protection.

References:

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