Radiation Effects on Plants and Animals

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Accompanying Notes

The following material supplements the PowerPoint presentation on Radiological Effects to Biota. From reading both, students should obtain an introductory level understanding of the following:

1. radioactive decay and ionization as it relates to effects of radiation
2. that the primary target for the induction of biological effects is DNA
3. the broad similarities in radiation responses among organisms
4. the wide variation in responses among organisms
5. the generation of free radicals and their role in biological effects from radiation
6. repair of damage from radiation
7. mis-repair of damage and the fate of mutations within a population of organisms
8. fundamental differences in human versus ecological risk analyses from the perspective of radiation effects
9. a general idea of the state of knowledge about radiation effects and some of the major data gaps that need to be addressed

I have plagiarised freely in putting this text together. Some of the material is taken directly from a chapter I am co-authoring with Peter Airey. The book, *Tropical Radioecology*, is edited by John Twining at ANSTO in Australia, and should be published in 2011 by Elsevier. I also copied material from:

Hinton, T. G. 1998. Estimating human and ecological risks from exposure to radiation. Chapter 7 IN: *Risk Assessment: Logic and Measurement* (Ed: M. Newman and C. Strojan). Ann Arbor Press, Chelsea, MI. pp 143-166.

Hinton, T. G., R. Alexakhin, M. Balonov, N. Gentner, J. Hendry, B. Prister, P. Strand, D. Woodhead. 2007. Radiation-induced effects on plants and animals. Findings of the UN Chernobyl Forum. *Health Physics.* 93:427-440.

Additionally, I borrowed freely from an excellent IAEA publication that was just issued. I highly recommend it for those more interested in the radiation biology perspectives:

IAEA, 2010. *Radiation biology: A handbook for teachers and students*. IAEA-TCS-42. ISSN 1018-5518. Vienna, Austria.

Radioactivity

Radioactivity is a natural phenomenon. It occurs when overly excited atoms seek stability by emitting energy in the form of radiation. The amount of energy and the forms of radiation emitted vary tremendously among the radioactive elements. It is due to this variation that the uses of radiation range from powerful tracers of biological, physiological, and geological cycles; to healing medicine; to weapons of mass

destruction. In this introductory material you will learn about what happens following exposure to radiation. The latter is fundamental in determining when radiation has medicinal characteristics versus lethal ones. Understanding such fundamentals is required if we want to confidently evaluate the human and environmental risks from radiological exposures.

Radioactive decay is accompanied by the emission of high energy radiation. Radioactive decay is associated with the transition of the nucleus from a higher to a lower energy state, and occurs at a rate which is described by a decay constant (λ), which is a property of the nucleus and totally independent of its surroundings. The process of radioactive decay transforms one element into another. There are long chains of naturally occurring transformations that occur within most ecosystems. For example, uranium-238 undergoes radioactive decay and transforms into thorium-234, thorium-234 changes into protactinium, and eventually (approximately 10^{10} years later and having undergone 14 different transformations) the original radioactive U atom is ultimately transformed into stable lead. At each step the resulting product loses all the characteristics of the parent element and acquires the characteristics of the newly formed daughter element. Characteristics such as colour, melting point, hardness, even physical state change with each transformation. For example, within the U-decay series, radium, a solid, is transformed by radioactive decay into radon, a gas. Radioactive decay is nature's alchemist (Hinton, 1998). The web-based table of isotopes has decay schemes for all known isotopes linked to a periodic chart of the elements at http://ie.lbl.gov/toi/perchart.htm .

Units of energy: Einstein's famous equation showed us that energy can be expressed in units of mass, and vice versa. In nuclear and radiation science, energy is normally expressed as changes in atomic mass units, μ, or as electron volts, eV. One eV is equivalent to 1.783×10^{-36} kg. The energy released during radioactive decay is measurable and can reach several million electron volts (MeV). Radiation in the form of alpha particles is often in the MeV range (*e.g.*, plutonium-239 emits an alpha particle with an energy of 5.2 MeV); whereas gamma emissions are generally less energetic, some thousand of ev (*e.g.*, cesium-137 emits gamma radiation with energy of 662 keV).

Interaction of radiation with matter

The interaction of radiation with matter leads to the excitation and ionisation of the target material (tissue). The measurement of the absorbed energy is known as dosimetry. In SI units, the unit of absorbed dose is the Gray (Gy), where one Gy = one Joule of absorbed energy per kg material (J kg^{-1}). The effects of dissipating radiation energy in the target tissue include:

- temperature increase (highly sensitive calorimetry is the only primary method for measuring dose from a radioactive source)
- excitation and ionisation of atoms
- the breaking of chemical bonds
- biological effects

Biological effects

DNA is the primary target for the induction of biological effects from radiation in all living organisms. There are broad similarities in radiation responses from different organisms, and yet wide differences in radiation sensitivity. The range in lethality from acute exposure to radiation various by three to four orders of magnitude among organisms,

with mammals being among the most sensitive and viruses being among the most radioresistant (Whicker and Schultz, 1982).

Damage from radiation is initiated by ionization. Ionization occurs if the radiation has sufficient energy to eject one or more orbital electrons from the atom in which it interacts. Ionizing radiation is characterized by a large release of energy (approximately 33 eV per event), an amount that is more than enough to break strong chemical bonds (e.g. only 4.9 eV are required to break a C=C bond; IAEA 2010)

The ionization process and resulting charged particles can subsequently produce significant damage to biological cells. Such damage is often referred to as direct effects. Much of the biological damage from radiation, however, is due to indirect effects from free radicals (**Figure 1**). Free radicals are the fragments of atoms that remain after being ionized. Free radicals have an unpaired or odd number of orbital electrons, resulting in a high degree of chemical instability. Such free radicals can easily break chemical bonds, and are a main cause of damage from radiation exposure. Free radicals react with cellular molecules within fractions of a second after their formation, and thus have a short life. The OH• free radical, formed by the ionization of cellular water, is among the most common because of the abundance of water in all biological tissues (about 80% of the mass of a living cell is water). To appreciate the quantity of free radicals produced, consider their concentration (expressed in terms of a G-value, defined as the number of radicals produced per 100 eV of energy absorbed in the medium). The G-value for the OH• radical is 2.6 (IAEA, 2010). Thus, if a 5 MeV alpha particle were to dissipate all of its energy within cellular water, some 50 000 free radicals of OH• could theoretically be produced.

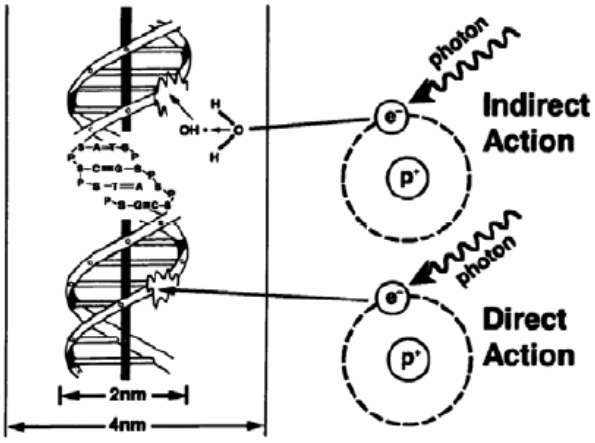

Figure 1. Direct versus indirect effects caused from free radicals (in IAEA 2010)

Free radicals are not unique to radiation, but are produced in response to many stressors: smoking, air pollution, exposure to solar UV radiation, tissue inflammation, and metabolism---all produce damaging free radicals. Such free radical production results in humans experiencing approximately 10^4 to 10^5 endogenous oxidative damages per cell per day among the 3 x 10^9 bases in the genome (IAEA, 2010). Damage caused from the free radicals is so abundant that very efficient repair mechanisms have evolved within all biological species, from yeast to humans, to counter their effects.

Radiation and the free radicals produced can damage DNA by causing several different types of lesions (e.g. single strand breaks, double strand breaks, base changes, interstrand crosslinks). The number of DNA lesions caused by a dose of 1 to 2 Gy is some 1000 base damages, 1000 single strand breaks (SSBs), and some 40 double strand breaks (DSBs; IAEA, 2010). DSBs are central to radiation-induced damage and their numbers correlate with radiosensitivity and the probability of cell survival. There are efficient DNA repair processes specific to each type of lesion. For DSBs the two primary repair pathways are non-homologous-end-joining (NHEJ) and homologous recombination (HR). The mechanisms of the two repair pathways are such that NHEJ is much more prone to errors during the repair process (IAEA, 2010).

Errors in repair can result in cell death through apoptosis, chromosome aberrations or mutations. The fate of mutations and their impacts within a population are dependent on the type of cell in which they occur. Two general types of cells are germ and somatic. Germ cells refer to the primordial cells from which eggs or sperm are derived. All other tissues (bone, muscle, blood, etc.) are derived from somatic cells. A mutation within a somatic cell can lead to cell death, or if the DNA damaged cell has undergone mis-repair such that the cell is still viable, then the mutation in the somatic cell can lead to cancer. Mutations in reproductive germ cells can decrease the number of gametes, increase embryo lethality, or be inherited by the offspring, resulting in their alteration. For humans, the risk of hereditary effects in offspring of exposed individuals is about 10% of the cancer risk to the exposed parents. The risk of non-fatal cancer for humans has been estimated at 1×10^{-5} per mSv. For non-human biota the risk of hereditary effects is unknown.

Most mutations are deleterious, offer no advantage to the individual that possesses it, and are subsequently removed from the population. Some mutations are neutral, have no apparent effect on the individuals that possess it, and can persist over many generations within a population. Rarely, a mutation might offer a selective advantage (e.g. increase the efficiency of water absorption in the roots of a plant that contains the mutation). Such selective advantages would spread in a population.

The deleterious effects of ionising radiation to biological systems have been known from the earliest days to be primarily dose dependent. Effort has therefore been expended over the years in defining the effective dose to a biological system. The issue is complex because the effective dose depends not only on the gross energy deposited, but also on the quality of the radiation and the radiation sensitivity of the affected tissue.

In SI units, the effective dose to humans is the Sievert (Sv), which is the absorbed dose (Gy) adjusted by two dimensionless weighting factors: the radiation weighting factor w_R to account for the biological effectiveness of the absorbed radiation; and the tissue weighting factor w_T to account for differences in the radiation sensitivities of different organs of the body. These weighting factors have been developed solely for human radiation biology—no such factors exist for non-human biota. Thus, dose to non-human biots is expressed in Gy, rather than Sv.

Biological effects of radiation are classified as deterministic or stochastic effects. Readers are referred to a detailed description in IAEA (2010), and to the general information in the associated powerpoint slides.

Environmental radiological protection

There are fundamental differences in determining the risk to humans following exposure to radiation and the risks to a radioactively contaminated environment. Human risk analyses largely focuses on cancer risks to individuals. Dose-response relationships are sufficiently established that risk factors (i.e. probability of lethality from cancer per unit of dose) are established. In contrast, ecological risk to non-human biota is seldom concerned with individuals, but instead, to populations of plants and animals. Management of the environment centers on a viable population of organisms, not on single individuals within the population. Endpoints for ecological risks are not cancer oriented, but instead include a wide assortment of effects ranging from chromosomal damage to reduced reproductive success. The dose-response relationships for these endpoints are not established, and therefore there are no risk factors that equate dose to the probability of an outcome.

The criteria for determining if an ecosystem is at risk from radioactive contamination are currently changing. Traditionally, the paradigm for protecting the environment was that if humans are protected then so is the rest of the environment (IAEA, 1992). That is, the protection criterion for humans (1 mSv / year) was considered to be sufficiently restrictive that populations of non-humans living in the same environment would be sufficiently protected. The International Commission on Radiological Protection (ICRP) recognised the need to provide more quantitative advice on environmental protection, and that a clear framework was required to assess the relationships between exposure and dose, dose and effects, and any consequences of effects. The ICRP has stated that the framework they are developing for environmental protection should complement the approach used for the protection of humans (ICRP, 2009). Consequently, the ICRP has suggested a similar reference-model approach as used for humans (*i.e.* "Reference Man") for non-human biota. They have, therefore, proposed a small set of "Reference Animals and Plants" (RAPs) for which reference dosimetric models have been developed and knowledge on radionuclide uptake and radiation effects collated.

The endpoints considered to be most relevant in determining risks to non-human biota are increased mortality, increased morbidity and decrease reproductive output. Of the three, changes in reproduction are thought to be the most sensitive to radiological exposures. Much more data are needed, however, before we can confidently predict population level impacts to non-human biota as a function of radiological exposures. Data are particularly scarce for chronic, low-level exposures; exposures over several generations; and when radiological exposures are combined with other types of contaminants or stressors.

The ability to predict population level effects under such scenarios are complicated by the large natural variation in sensitivities to radiation among the individuals within a population. Additionally, indirect effects occur, compensating mechanisms exist, and adaptation to the radiological exposures can take place. An example of an indirect effect is the greater abundance of resources (i.e. food, water, light, etc.) available to radioresistant individuals when radiosensitive individuals decline within a population. The same analogy holds relative to a greater abundance of resources available to radioresistant populations within a community when radiosensitive populations decline (i.e. one species of insect declines leaving more resources to a radioresistant insect species occurring within the same community). Such interactions are extremely difficult to predict. Likewise, compensating mechanisms have been documented in populations

of exposed animals that complicate the prediction of effects. An example of a compensating mechanism is provided by Blaylock et al. (1969). They documented an increased mortality of fish embryos exposed to a dose rate of 4 mGy/d in a contaminated lake. This effect, however, was compensated for when the fish produced larger brood sizes, with the net result that no effect was observed to the population.
Several organizations and research groups are actively seeking to improve our knowledge of radiation impacts on the environment, and to derive benchmarks of acceptable dose rates that will be considered protective of the structure and function of ecosystems. Consolidation of data within a common database is augmenting their efforts. A radiation effects database, called FREDERICA, has been developed and is freely available on line at www.frederica-online.org (Copplestone et al. 2008). See the accompanying lecture notes and power points slides of David Copplestone on the derivation of benchmarks for non-human biota, and Hinton and Whicker (1997).

Considerable uncertainty and controversy remains relative to the effects from chronic, low-level exposures to radiation. Much can be learned from the Chernobyl accident and the multiple generations of biota that have been exposed within the contaminated 30-km zone since 1986. A United Nations subcommittee reviewed the environmental effects from the Chernobyl accident (Hinton et al., 2007), and their conclusions form a major component of my accompanying power point presentation. Other scientists have since documented effects at Chernobyl from dose rates previously considered safe to biota (see power point slides). Much healthy debate exists on this topic. Major data gaps undoubtedly exist in the following areas:

- What are the effects from chronic, low-level exposures to radiation?
- What is the extent of inherited, transgenerational effects to populations?
- What is the significance of molecular effects to individuals and populations of biota?
- How are effects from radiation altered when organisms are exposed to other stressors?

These questions are not unique to radiation ecology, but are also taxing the scientific abilities within ecotoxicology relative to other types of contaminants (Eggen et al. 2004).

Additional References

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