Chapter 7
What Does Radiation Do to Us?

The technology used to detect if vehicles are carrying radioactive material is so sensitive it can tell if a person recently received radiation as part of a medical procedure.
Timothy Murphy 1952–

Any radiation exposure can potentially have negative effects on health. This can be considered as the basic principle of radiation protection. It is therefore no surprise that damage due to ionising radiation was first observed very soon after the discovery of radioactivity by Becquerel. The biological effect of ionising radiation is a consequence of the energy transfer, by ionisation and excitation, to cells in the body.

The biological effects of radiation absorption are shown in Figure 7.1 in detail. Figure 7.2 shows a very rough classification of different types of radiation damage. Usually they are divided into three different categories (early, delayed and genetic), as discussed in the next three subsections.

7.0.1 Early Effects

This radiation damage occurs immediately after the irradiation, and only appears for high radiation doses. From a whole-body dose of 0.25 Sv upwards, it is possible to see the effects using a blood test (haemogram). For doses of around 1 Sv, clear symptoms of radiation sickness are to be expected. However, the recovery of the patients is nearly guaranteed if sufficient medical care is available. For a whole-body dose of 4 Sv, the chance of survival is 50 %. This dose is called the lethal dose. For a dose of 7 Sv, the death rate is nearly 100 % (see Figure 7.3).

For high radiation doses, the symptoms of radiation sickness occur within a few hours of irradiation. The symptoms are headaches, nausea and vomiting. These symptoms normally disappear after some time. After a quiet period of several days almost without any symptoms, the second phase of the radiation sickness starts. The

C. Grupen and M. Rodgers, *Radioactivity and Radiation*,
DOI 10.1007/978-3-319-42330-2_7

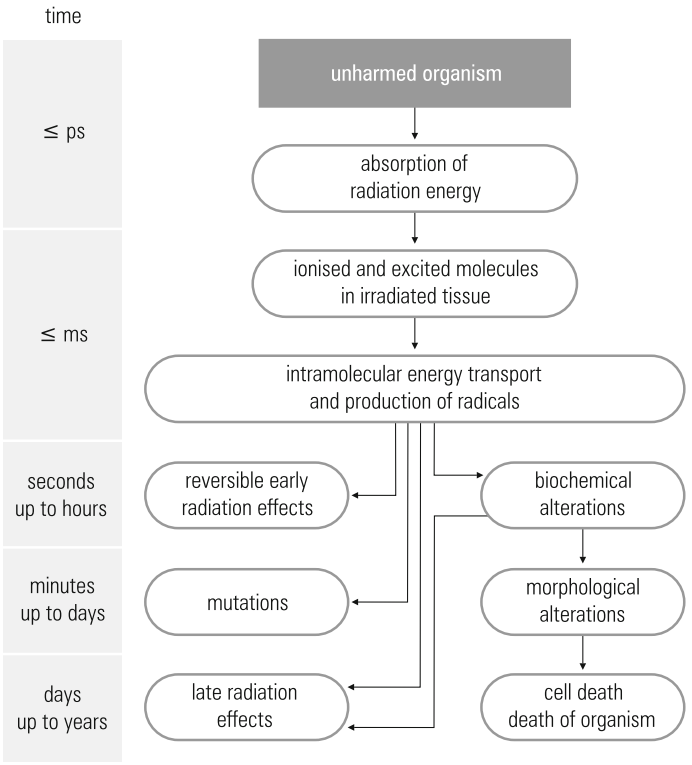


Fig. 7.1 Timings of the biological effects of absorbed radiation energy

symptoms are then fever, haemorrhage, vomiting of blood, bloody faeces and loss of hair. For the highest radiation doses, the quiet phase will be shorter or may even not occur. If the exposed person survives for eight weeks, there is good reason to expect a complete recovery from radiation sickness. However, in some cases death can occur after several months.

Biological tissue has several different repair mechanisms, giving it some ability to rectify damage. Therefore, there is a threshold dose for early effects after irradiation. This means that below a certain dose, no lasting damage is observed. This threshold dose depends on how the dose was distributed in time, and which parts of the body are affected. The smallest value of the threshold is about 0.5 Sv (which is when the exposure all happened at once), and it is closer to 1 Sv if the dose is spread over a longer period. Radiation exposure from natural radiation is certainly far below this threshold. According to current (cautious) thinking there is, however, no threshold dose for the other two effects, discussed below.

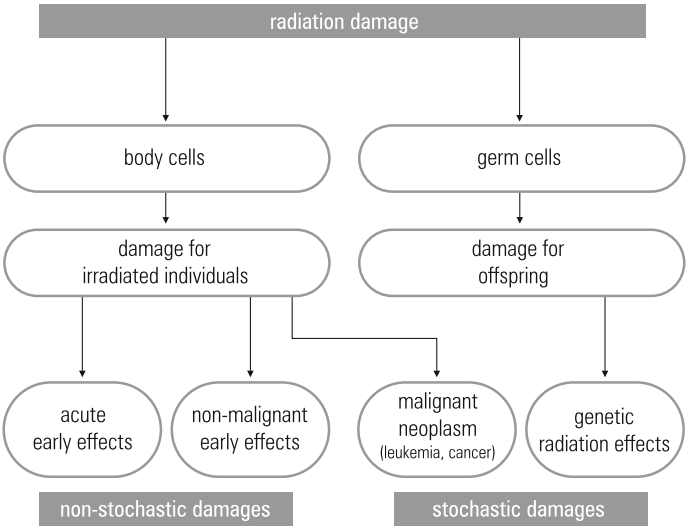


Fig. 7.2 Overview of the different kinds of radiation damage. The early non-malignant effects are symptoms such as temporary reddening of the skin

7.0.2 Delayed Radiation Damage

Delayed (or "late") effects are those which occur after a long dormant period, which can be several decades long. The most common and most frequently-discussed late effect is cancer. In contrast to early effects, whose severity is related to the dose received, delayed radiation-damage effects represent a so-called stochastic risk. This means that the probability of each negative outcome rises with increasing dose, but the severity of each outcome does not (see Section 7.5). The total cancer risk per absorbed dose of 10 mSv is estimated to be to about 5×10^{-4}. This means that out of 10 000 people being irradiated with 10 mSv, on average five of these will later develop cancer due to that exposure.

It is generally assumed that the relationship between the probability to develop cancer and the absorbed dose is a simple straight line (see Figure 7.4). In addition to the assumption of a linear dependence it is argued that there is no threshold for radiation damage: i.e. there is no amount of radiation small enough that it has no damaging effect at all. In combination, these assumptions are called the LNT (Linear No-Threshold) hypothesis, and are quite conservative, as discussed in Section 7.5. Some scientists even assume that humans have no sense organ to warn against ionising radiation because they do not need one since low doses do not present any risk.

It is interesting to compare the cancer incidence as a result of exposure to radiation with other risks. The probability that any person is the victim of a fatal accident (say, a traffic accident, or an accident in the home) in any one year is about 5×10^{-4}, which is comparable to the total cancer risk after a whole-body exposure of 10 mSv.

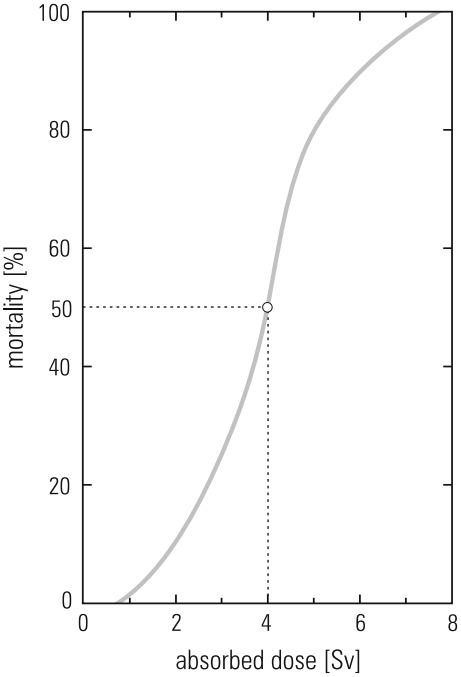


Fig. 7.3 Early effects: mortality (death rate) after 30 days for different whole-body doses for humans

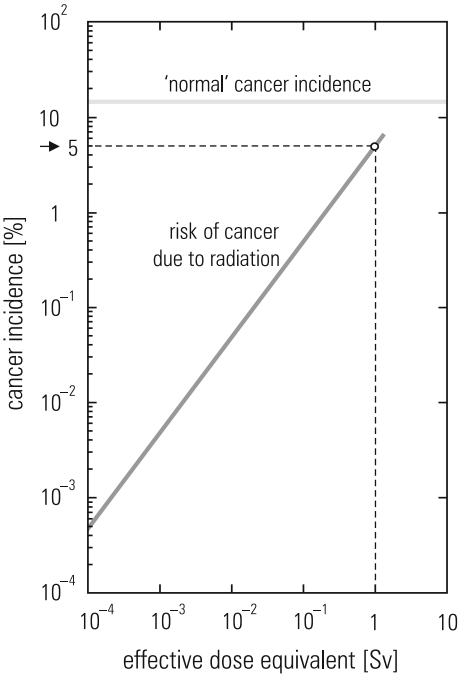


Fig. 7.4 Dependence of the radiation risk on the absorbed whole-body dose in comparison to the 'normal' incidence of cancer (LNT model)

"10 000 nuclei decay per second in our bodies,
and we worry about the price of petrol!"

7.0.3 Genetic Damage

Radiation absorption in germ cells (the precusors to eggs and sperm) can result in mutations. For the irradiated person, the mutations are not recognisable. They will only manifest themselves in the following generations. During the reproductively significant age of humans (up to the age of 40), about 160 germ-cell mutations occur due to environmental factors. A radiation exposure of 10 mSv will add another 2 mutations on average: this corresponds to less than two per cent of the natural rate of mutations. The average risk factor for these radiation effects is estimated to be 10^{-4} per 10 mSv (i.e. one sufferer would be expected if 10 000 people received 10 mSv). This number includes the effect on the first two generations. After this, the probability of transmission is very small.

7.1 Radiosensitivity

The sensitivity to radiation of a piece of tissue is increased if the cells are reproducing rapidly, and also if there is a large variety of different cell types in a small region.

If the cells are dividing rapidly, there is less time available to repair damage before the next division of the cell occurs. If there are many cell types in a small area, it follows that if a few of one type happen to be damaged, there will not be many of that type nearby to replace them. These effects combine to ensure that the time when humans are most sensitive to radiation is as embryos: the cells are dividing rapidly for the embryo to grow, and the nascent organs only contain small numbers of cells. This means that the costs and benefits of perfoming medical procedures involving radiation must be weighed particularly carefully for the case of pregnant women.

It should be mentioned that there are some chemical substances which can modify the biological effect of radiation quite substantially. For example, oxygen, bromouracil and fluorouracil increase the radiosensitivity, i.e. they make human tissue more susceptible to radiation. The water content in the cell also has quite a large influence on the radiosensitivity because potentially damaging free radicals are produced by the ionisation of water molecules. Some carcinogenic (cancer-causing) substances act by increasing the sensitivity of tissue, rather than by causing damage themselves directly.

In the same way that sensitising substances exist, there are also radioprotective substances. For example, mice will survive a radiation dose of 7 Sieverts if they receive an injection of cystamine before the irradiation, while this dose is normally lethal for mice. If an exposure to radiation is fractionated, i.e. received as multiple sub-doses separated by periods of time, its effect is reduced. Clearly, regeneration mechanisms come into play which repair radiation damage between the individual fractions. Also, there is a higher resistance to a large dose if it is preceded by another dose which is small, but significantly above background level (‘pre-irradiation’). The use of fractionated irradiation or pre-irradiation has been shown to reduce early radiation damage, and there are some tentative indications that it can reduce the cancer risk.

7.2 Decorporation

It is also possible to remove incorporated radioactive substances from humans by administering suitable drugs. These methods of *decorporation* work by helping the body flush out the incorporated radioactive substance. One possibility is for the drug to bind the atoms of the radioisotope together (chelation), so that the kidneys can remove them in urine. Decorporation drugs can also help by stopping the body absorbing the radioisotope through the gut, so that it passes out in faeces. The best results for decorporation have been obtained with DTPA (diethylenetriamine pentaacetate) and EDTA (ethylenediamine tetraacetate). Figure 7.5 shows the excretion rate of plutonium from the human body after an incorporation caused by an accident. There were two treatments with DTPA (the first very soon after the accident), which increased the excretion rate markedly.

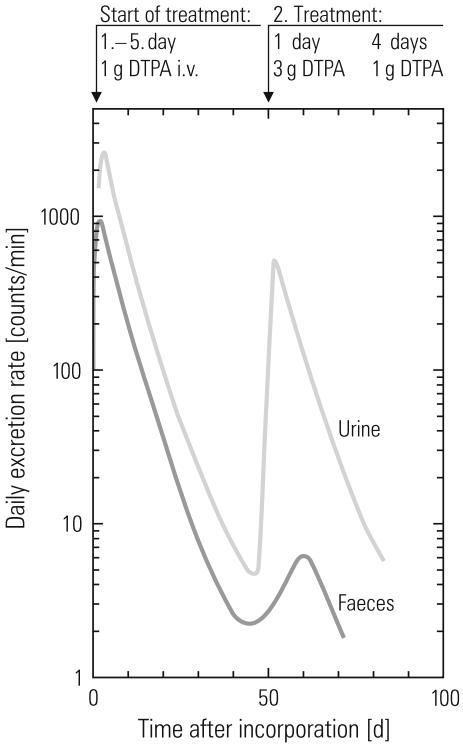


Fig. 7.5 Excretion of plutonium from the human body. There were two treatments with DTPA after an incorporation caused by an accident

The human body will have some ability to excrete incorporated material, and this ability will vary according to the substance involved. Because this excretion will reduce the amount of the radioactive substance in the body, and therefore its activity, the concept of a biological half-life is useful. This is the time required for the body to excrete half of a quantity of radioactive substance. This is distinct from the (normal) physical half-life. Indeed, both the excretion effect and the decay of the material will act to reduce the activity of the substance in the body over time, so there is a concept of the effective half-life, which is the time for the activity of a particular substance to reduce to half inside the human body. The effective half life is always lower than the other two half-lives. For example, for yttrium-90 (a β-emitter used in radiotherapy), the physical half-life is 64 hours, the biological half-life is 56 hours, and the effective half-life is 30 hours.

Figure 7.6 shows the decrease of ^{137}Cs (physical half-life: 30 years) stored in the bodies of humans and some other mammals. The biological half-life of ^{137}Cs for humans is 110 days, giving an effective half-life of 109 days. The other mammals listed have faster metabolisms, increasing the rate at which the caesium is excreted.

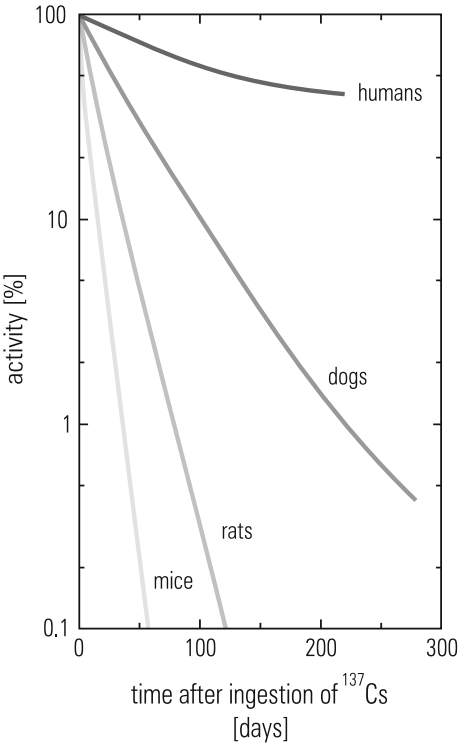


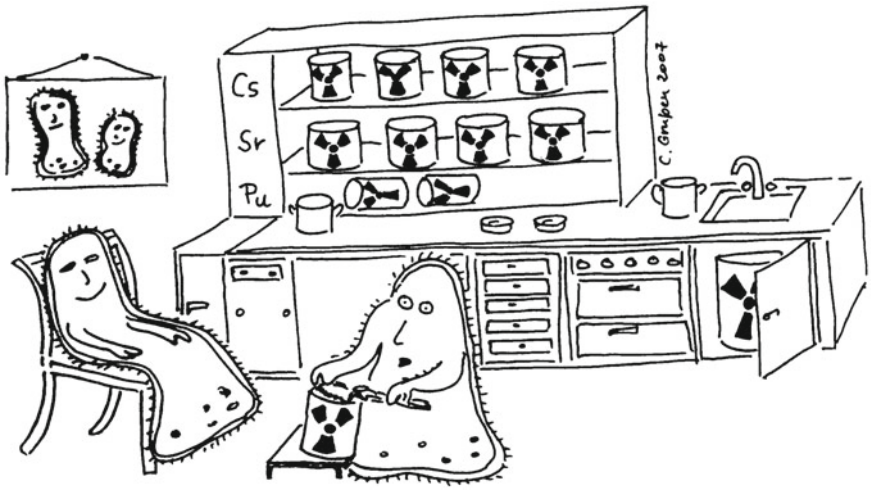
Fig. 7.6 Decrease of the accumulated ^{137}Cs in the body for humans and for some other mammals

7.3 Non-Human Organisms

Different organisms are resistant against ionising radiation to different extents. For example, the lethal dose, remembering that doses are inherently per-kilogram measures, for all mammals is about the same (humans: 4 Sv, dogs: 4 Sv, monkeys: 5 Sv, rabbits: 8 Sv, marmots: 10 Sv). In contrast to that, spiders (with a lethal dose of 1000 Sv) and viruses (2000 Sv) are much more resistant against ionising radiation. If there were a nuclear holocaust,[1] it would probably only be survived by spiders, viruses, bacteria and certain types of grass. The idea that cockroaches have an extremely high radiation tolerance is a myth: although higher than that of humans, their resistance is similar to that of many invertebrates.

The bacteria deinococcus radiodurans and deinococcus radiophilus can survive enormous doses (30 000 Sv) because of their extraordinary ability to repair radiation damage. They have even been found in the hot reactor cores of nuclear power plants. These bacteria somehow manage to repair DNA damage with the help of a special enzyme system, even if the helix structure of the DNA exhibits about one million breaks. Deinococcus radiodurans is able to make chemical changes to highly radioactive waste, which make the process of disposing of it easier and more efficient.

[1] Let's hope not!



"So, Deino, shall we have some delicious caesium-137 for dessert?"

© by Claus Grupen

For this reason, there is active research into using these bacteria to clean up the radioactively contaminated areas which result from nuclear accidents, military use and earlier generations of nuclear power plants. Because of the high level of resistance to radiation, and also to extreme temperatures, these organisms can survive in meteorites under space conditions over a long time. Consequently they can also propagate over large distances. It is even conceivable that life on Earth was initiated by the impact of meteorites containing such organisms (a hypothesis called 'panspermia').

7.4 Radiation-Absorbing Fungi

The fungus cryptococcus neoformans appears to exhibit the astonishing ability to transform the energy of ionising radiation into usable energy. The fungus was described in detail for the first time in 1976. Its special accomplishment became generally known when it was found after the Chernobyl accident in the sealed nuclear reactor. In general, fungi are rather radiation resistant: they can survive radiation doses of up to 30 000 Sv. However, cryptococcus neoformans goes a dramatic step further: its metabolism increases significantly under irradiation. It appears that the fungus achieves this by using melanin, a class of pigments also common in plants and animals, to derive usable energy from the γ radiation, but the mechanism is not well-understood. In principle, this is a similar technique to that of normal plants: plants transform electromagnetic radiation from the visible range into chemical energy (by photosynthesis), and the fungus seems to be doing the same with radiation from the γ energy range.

As an aside, cryptococcus neoformans can be harmful for humans: it can cause meningitis, especially as a secondary infection for AIDS patients.

7.5 Radiation Risk Factors

To make predictions about the likely effects of an exposure, it is useful to have a set of numbers to represent the cancer risks associated with certain doses. However, in determining these numbers, an assumption had to be made. This is because there is good evidence for the relationship between exposure and risk at the high end of the scale, but the measurement of small risks from small exposures is very difficult.

The solid evidence comes from cancer incidence in the aftermath of radiation accidents and after the dropping of nuclear bombs on Hiroshima and Nagasaki. After these horrific events, there were many people with high, measurable exposures, and many cases of cancer, so it was easy to work out the relationship between them, and to summarise it in a number called the risk factor, different for each cancer type.

To estimate the risk factors for lower doses, the conservative assumption is made that the LNT (Linear No-Threshold) hypothesis holds. This hypothesis is that there is a linear relationship between the absorbed dose and the probability of radiation-induced cancer, and that there is no threshold dose: i.e. no dose is small enough that the risk from it is effectively zero. This means that each additional millisievert of exposure gives an identical increase in risk, regardless of whether the total dose is high or low. The "no-threshold" component is particularly conservative, as the bodies of all living things are known to have some ability to repair minor radiation damage.

Alternative models have been suggested in which the increase in risk with increasing exposure starts slowly, but the risk increases more strongly above a few tens of millisieverts. If this model held, it would mean that an additional millisievert on a low exposure causes little additional damage, but an additional millisievert on a higher dose is more harmful.

Initially, the risk factors were developed based on cancer types with relatively short latency periods, like leukaemia. With the help of computer models of the biochemical behaviour, these results have been extrapolated to other types of cancer. The results of approximations to low doses are, however, somewhat problematic. Additional hypotheses are required to arrive at risk factors which are believed to be reliable for low doses (say, 20–50 mSv), which are of interest for radiation-protection purposes. In the early 1980s, the risk factors determined in this way led to values for radiation-induced cancer of 1.3 % per Sievert. Based on new results, better models and the LNT assumption, the somewhat higher risk factor of 5 % per Sievert is now usually used. This means that if 10 000 people receive a dose of 20 mSv, it would be expected to cause ten additional cancer cases. The risk factors for different types of radiation-induced cancer have also been determined (e.g. 0.5 % per Sievert for leukaemia and 0.85 % per Sievert for lung cancer).

If a radiation worker is exposed to the maximum total dose (in the EU) of 400 mSv over an entire lifetime, there is an additional total risk of 2 % (i.e. of 1000 people working under these conditions, about 20 will develop a radiation-induced cancer). This size of occupational risk appears to be acceptable.

It is possible to use risk factors to illustrate the stochastic nature of cancer risk, namely that an increase in dose increases the probability of each negative outcome, but does not increase the severity of any outcome. Take for comparison cancers of the oesophagus (risk factor 0.3 %/Sv) and of the thyroid (risk factor 0.08 %/Sv). Oesophagal cancer is widely considered to be among the most serious of cancers. In contrast, although it is still a serious medical condition, thyroid cancer is among the least severe cancers, particularly if caught early. A 10 mSv whole-body exposure gives a 3×10^{-5} chance of oesophagal cancer, and a 8×10^{-6} chance of thyroid cancer. A 250 mSv whole-body dose gives 7.5×10^{-4} for the oesophagus, and 2×10^{-4} for the thyroid. The probability of each cancer has risen with the higher exposure (and the probabilities have risen in line with each other), but the potential medical conditions themselves are the same for the two exposures.

Risk factors are statistical, which means that they affect the probabilities of certain outcomes, but some people will be luckier and others less so. There are some good examples of this from the early days of research into radioactivity. Marie Curie, who handled significant quantities of polonium and radium in her laboratory, eventually died of leukaemia. In contrast, Otto Hahn, who dealt with similar quantities of radioistopes, lived until old age. The fact that he did not share the same fate as Marie Curie was considered a miracle. An American colleague visiting Otto Hahn expressed this feeling as: “After having received such high doses from your experiments it is really a shame that you are still alive!”

The additional exposures of smokers to radiation (predominantly because natural radioactive elements from inhaled air stick to the tar within smokers’ lungs) are discussed in Section 6.5.2. The equivalent dose for the bronchi of a very heavy smoker (two packets of cigarettes daily) might be as high as 5 Sv over a period of 25 years. This in itself would lead to a risk of a cancer of the lungs or bronchi of something like 5 %. If, in addition, the carcinogenic effect of nicotine and tar is considered, the number is more like 30 %. This high value is obtained because the cancer risks due to ionising radiation and due to chemical effects will reinforce each other.

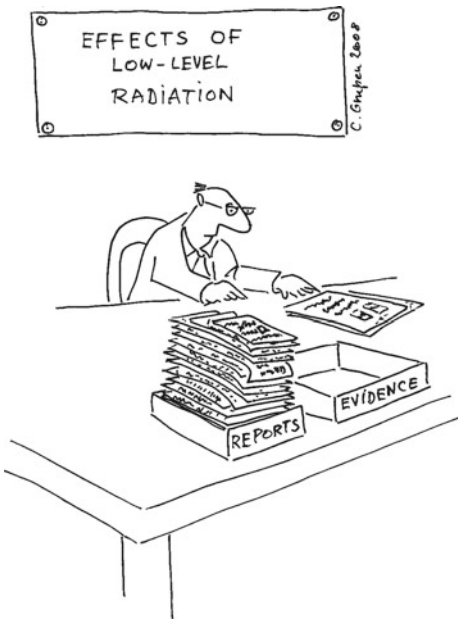
7.6 Low Radiation Doses

The risk factors for malignant late radiation damage are very low for doses in the range of a few millisieverts, as they must be because background radiation exposures are at least 2 mSv per year. In individual cases it is, for all practical purposes, impossible to establish correlations between an observed sickness and a possible irradiation of this scale, because the normal cancer rate (for people without the additional exposure) is so much higher than the additional risk from the exposure. So the fact that the risk is low makes it hard to observe its scale.

Apart from damage due to ionising radiation, favourable effects after modest radiation exposures have also been observed. This effect is called hormesis. It is suggested that low doses of non-natural radiation might increase the lifetime of cells. The idea is that cells are able to repair minor damage (on the same scale as is caused by natural radioactivity) and that cells become more resistant to damage in general if they are regularly stimulated to repair themselves by being exposed to additional non-natural low-level radiation.

An interesting study was published recently, describing a group of people who accidentally received significantly raised (but still low) radiation doses over a period of multiple years, and appear to have received a health benefit. In Taiwan in the early 1980s, radioactive cobalt-60 (half-life 5 years) was accidentally included in some steel building materials, and these materials were then used to build 1700 apartments. Over the course of two decades, some 10 000 people occupied these apartments, receiving doses between a few millisieverts per year and a few tens of millisieverts per year. After the problem was discovered, the individuals were traced, with the expectation that there would be an increased cancer rate. The researchers found not only no increase in cancer rates, but actually significantly lower incidences of cancer (and also of birth defects). If these results are supported by other findings, then the limits given in radiation protection regulations should be reconsidered, and in particular the LNT hypothesis should be rejected for low to moderate doses.

Nonetheless, for the purposes of radiation protection it must be assumed that any additional irradiation should be avoided if possible.



7.7 Eradication of Insect Pests

A breakthrough in fighting the tsetse fly on the island of Zanzibar was achieved by the so-called sterile-insect technique. The tsetse fly is endemic across large parts of sub-Saharan Africa, and carries a parasite which causes trypanosomiasis (sleeping sickness), a disease which can devastate both livestock and human populations. Using the sterile-insect technique this fly has been practically eradicated on Zanzibar. In this method, tsetse flies are bred in a research laboratory in large numbers, and the male flies are sterilised with low-level γ rays (since it is next to impossible to separate male from female insects, all the flies bred are irradiated). The infertile males are released from planes over the territory. They mate with wild females, which are not then fertilised, and so have no offspring. The release of 8 million sterile male tsetse flies on Zanzibar decreased the rate of trypanosomiasis among cattle from 20 % to below 0.1 %. The success of this sterile-insect technique was aided by the fact that out of a total of 22 different species of this fly, only one type existed on Zanzibar. In addition, the isolated location of an island is particularly well-suited for this kind of endeavour.

The sterile-insect technique has also successfully been used to eradicate the screw-worm fly (cochliomyia hominivorax) in areas of North America. There have also been many successes in controlling species of fruit flies using this technique. This biological method can also be considered when the desire is to control the population of a species rather than eradicating it.

7.8 Metabolism of Plutonium

In the course of the construction of the first nuclear bombs in the United States (the Manhattan Project), the workers building the bombs were exposed to dust particles containing plutonium. Naturally, questions arose about the biological effects of inhaled plutonium. Of the two bombs dropped on Japan at the end of the Second World War, one was made of enriched uranium (mostly ^{235}U) and the other of ^{239}Pu.

There were warnings of potential health risks and it was suggested that a study be undertaken immediately to understand the metabolism of plutonium. A small fraction of the plutonium that had been produced was allocated for animal studies. The plutonium was injected into different animals, and the excretion and retention rates were studied. Since these rates differed substantially for different species, it was difficult to correlate animal excretion and retention data to humans. As a result, there was a proposal to administer small amounts of plutonium to humans to obtain reliable data.

In this context, plutonium was injected into hospital patients at Rochester and Chicago (USA) in the late 1940s. The patients were thought to be either terminally ill, or to have a life expectancy of less than ten years either due to age or to chronic diseases. Different quantities between a few μg and about 100 μg were administered, corresponding to activities of up to 220 kBq. After injection, samples of blood, urine and faeces were analysed at Los Alamos. The physicists and physicians felt reasonably certain that there would be no additional harm to the patients given their preexisting medical conditions.

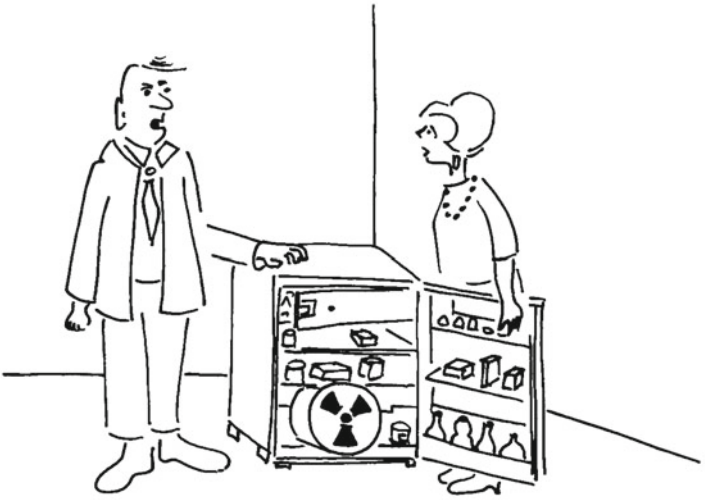
The urinary excretion data showed a rapid initial excretion rate, although much slower than for radium. This rate levelled off to a constant amount per day after a few weeks. It was found that significant quantities of plutonium were retained in the body in the long term, making the problem of chronic plutonium poisoning a matter of serious concern. Because of this retention, and the significant quantities of plutonium used, the patients received doses of hundreds of millisieverts per year for the rest of their lives.

Out of the 16 patients tracked, ten died within ten years. Four patients survived more than 20 years. Three of the four survivors were examined in 1973, 28 years after the injections had taken place, providing long-term patterns of plutonium retention and excretion. The results of these studies were used as source for estimating permissible limits in the framework of radiation-protection regulations.

Naturally, these experiments with radioactive substances on humans raised serious questions about medical ethics, especially because of the absence of informed consent from the patients selected.

Summary

Biological consequences of ionising radiation are subdivided into early and late effects. Early effects are only observed for doses larger than 250 mSv. In this case, the seriousness of the consequence is directly related to the dose. The lethal dose (50 per cent mortality) is around 4 Sieverts for humans. Late effects (mostly cancer) occur a long while after the exposure, typically 20 years. Here, the severity of the outcome does not depend on the dose, but rather the probability of occurrence of each outcome does. Ionising radiation can also cause mutations in germ cells.



"The radioactive generator not only powers the fridge, it also sterilises the food!"