Background material on nature of radioactive contamination at the University of Manchester and possible health risks: Task 1 report

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FOREWORD

Concern has been raised by staff at the University of Manchester about possible health effects from radioactive and chemical contamination in certain locations on the campus. These locations were used by Rutherford in carrying out early work with radioactive material and residual radioactivity has been found. An Inquiry has been set up under the independent chairmanship of Professor David Coggon to examine this possibility and The Radiation Protection Division of the Health Protection Agency (HPA-RPD) has been asked to carry out a risk assessment in support of the Inquiry with regard to the radioactive contamination.

This report contains four papers to meet the requirements of Task 1.1 “preparation of background material on the nature of the radioactive contamination at the University of Manchester and possible risks to health”.

The first paper provides an introduction to radiological protection and gives definitions of basic quantities that are used. This paper gives an introduction to the radionuclides thought to have been used in the old Physics buildings at the University of Manchester and the levels found naturally in the environment.

The second paper provides more detailed information on the radionuclide decay chains thought to be present. It includes information on which exposure routes are important and which members of those decay chains are likely to give the highest doses over the period of interest.

The third paper provides an overview of the behaviour of the radionuclides of concern when they are in the body. It includes a discussion of the organs likely to receive the highest doses following the ingestion or inhalation of radionuclides and also considers doses to the pancreas and brain.

The fourth paper provides an overview of the possible radiation induced health effects, specifically looking at risks of pancreatic and brain cancer from ionizing radiation.

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1 PAPER 1: BASIC CONCEPTS IN RADIATION PROTECTION

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1.1 Introduction to radioactivity

An atom is the basic building block of elements which combine together to form all material. Each atom contains a central “nucleus” consisting of protons and neutrons. Around this nucleus is a cloud of electrons. The number of protons in a nucleus is called the atomic number and is unique to each element. The sum of the number of protons and neutrons within a nucleus is termed the mass number. An element is defined by the atomic number, that is, the number of protons within its nucleus. For example, lead will always have 82 protons in its nucleus. However, different forms of an element can have different numbers of neutrons and these different forms are called isotopes. Each isotope therefore has a different mass number and it is this number that is often written with an element name to define it. For example, common lead isotopes are lead-210 and lead-212, with the first isotope having 82 protons and 128 neutrons and the latter having 82 protons and 130 neutrons.

Although many isotopes are stable, that is they do not change with time, some will undergo radioactive decay. This is where an atom is unstable and it emits energy in the form of radiation to enable it to become more stable. This emitted radiation takes the form of a “particle”, for example the emission of an electron is termed “beta” radiation whilst the emission of an “alpha particle” consists of 2 protons and 2 neutrons. When a particle is emitted from an atom it will change its atomic number as protons are either lost from the nucleus (for example, with alpha radiation) or a proton will be added to the nucleus when, for example, a neutron changes to a proton and an electron with the electron then being emitted as beta radiation. Emission of a particle often leaves the atom with too much energy and therefore, radioactive decay by beta or alpha particle emission is often accompanied by the emission of a gamma ray. A gamma ray is a discrete amount of electromagnetic energy without mass or charge. The act of atomic transformation is termed radioactive decay and the isotope that emits the radiation is termed a radionuclide.

Radiation is typically classified according to the type of effect it has on matter, for example, organs in the human body. There are two classes; ionising and non-ionising radiations. Ionising radiation has sufficient energy to produce ionisation as it passes through tissues of the body whereas non-ionising radiation does not. Non-ionising radiation includes radio and micro waves and is not of concern for this work at the University of Manchester. This work relates to ionising radiation and this type of radiation includes the emission of alpha and beta particles and gamma rays.

1.1.1 Characteristics of ionising radiation

Alpha particles are not particularly penetrating and are stopped by a sheet of paper, or the outer dead layer of skin of the body, and are therefore unable to irradiate internal

organs unless they are emitted from within the body. Alpha particles, being slower and more heavily charged than beta particles, lose their energy much more densely along their paths. This is important as if the radionuclide is selectively taken up by an organ or tissue then that organ can have a relatively large amount of energy deposited within it.

Beta particles are stopped by a thin sheet of metal and, except for the most energetic particles, they are also stopped by the skin. This means that a beta particle emitted outside of the body will generally not deposit energy in internal organs. It should be noted that ionising radiations differ in the way in which they interact with biological materials, so equal absorbed doses (meaning equal amounts of energy deposited) do not necessarily have equal biological effects. For instance 1 Gray (this is the unit that is used to quantify the amount of ionising radiation deposited in a unit mass of matter – see the glossary for more information) to tissue from an alpha particle is much more harmful than 1 Gray from beta radiation because an alpha particle, being slower and more heavily charged than beta particles, loses its energy much more densely along its path.

Gamma rays are stopped by thick layers of highly dense materials such as lead. A gamma ray is able to pass right through the body and so will irradiate all organs within the body regardless of whether the gamma ray is emitted internally or externally to the body. However, as gamma rays are more penetrating than alpha or beta particles the amount of energy deposited in any organ it passes through, and therefore the potential damage caused, will be less than that for an alpha particle with the same energy. This is shown in Figure 1.1.

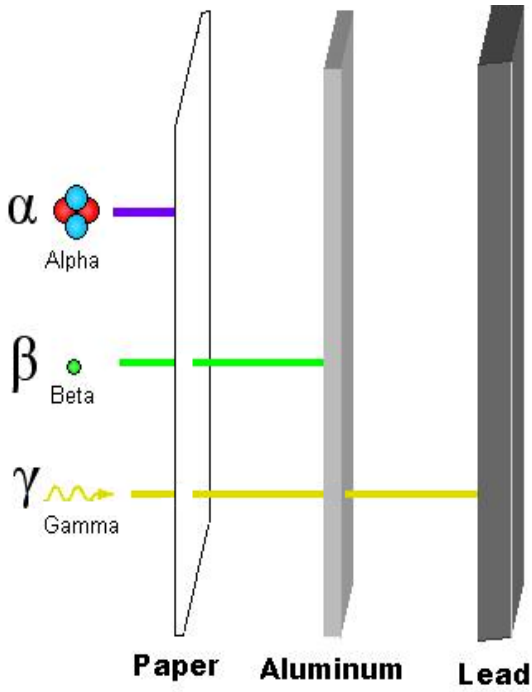

Figure 1.1 The penetrating power of ionising radiation

1.1.2 Units of radioactivity

The energy of alpha and beta particles and gamma rays is usually expressed in the unit of the **electron volt** (eV). Multiples of the eV are often used, for example a million eV is expressed as a **MeV** (mega electron volt).

The rate at which spontaneous transformations occur in a given amount of a radioactive material (radioactive decay) is known as its activity. Activity is expressed in a unit called the **Becquerel** which has the symbol **Bq**, where 1 Bq is equal to one transformation per second. As for the unit of energy, multiples of the Bq are often used to describe the number of transformations that a radioactive material will undergo, with 1 million transformations being expressed as a mega-Becquerel (**MBq**).

The time taken for the activity of a radioactive material to fall to half of its original value is termed the half-life. This represents the time for half of the nuclei in a sample to decay. Each radionuclide has its own specific half-life, with times ranging from fractions of a second to many millions of years. An illustration of half-life is given in Figure 1.2 for a radioactive material that has a half-life of 1 hour, where the activity of this material is shown at hour intervals.

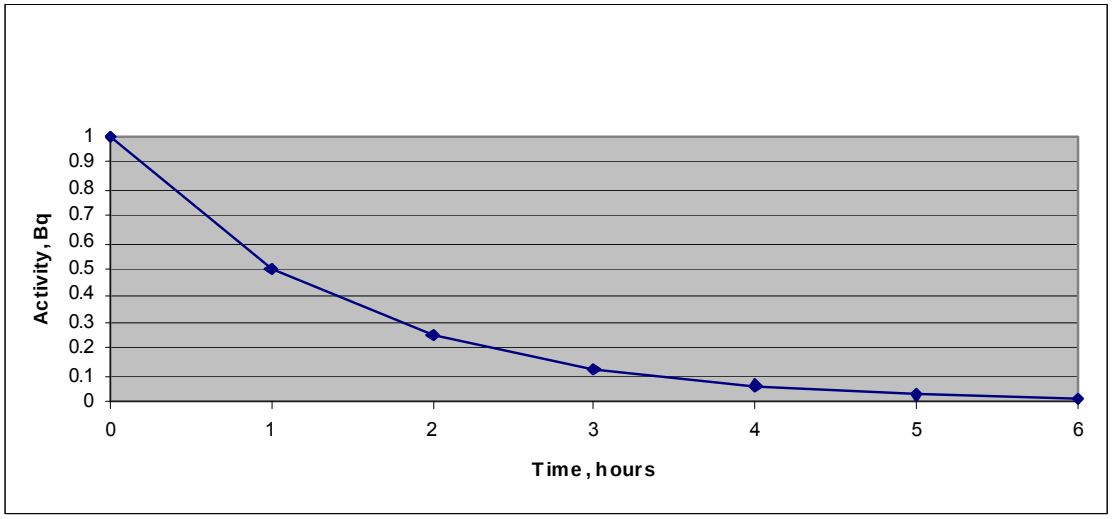

Figure 1.2 Radioactive decay curve for a radioactive material with a half-life of 1 hour

From this figure it can be seen that after 1 hour the activity of the material is at 0.5 Bq which is half that present at the start (1 Bq). After another half-life (2 hours) the activity present has fallen in half again (to 0.25 Bq).

1.1.3 Radioactive decay chains

For some radionuclides, radioactive decay does not form a stable atom. The new atom formed, termed the progeny, is also radioactive and will itself undergo radioactive decay. This then forms what is termed a radioactive decay series or chain where the first radionuclide is termed the chain header or "parent" radionuclide and this is followed by a series of "progeny" radionuclides all of which radioactively decay until a stable atom is reached. See Figures 1.5, 1.6 and 1.7 at the end of this paper for examples of decay chains for the radionuclides uranium-238, thorium-232 and uranium-235.

1.1.4 Secular equilibrium

Secular equilibrium is a special state of a radioactive decay chain where all radionuclide members have the same activity. This state exists after sufficient time has passed that the activity of a radioactive isotope remains constant because its production rate (due to the decay of a parent isotope) is equal to its decay rate. This is shown graphically in Figure 1.3.

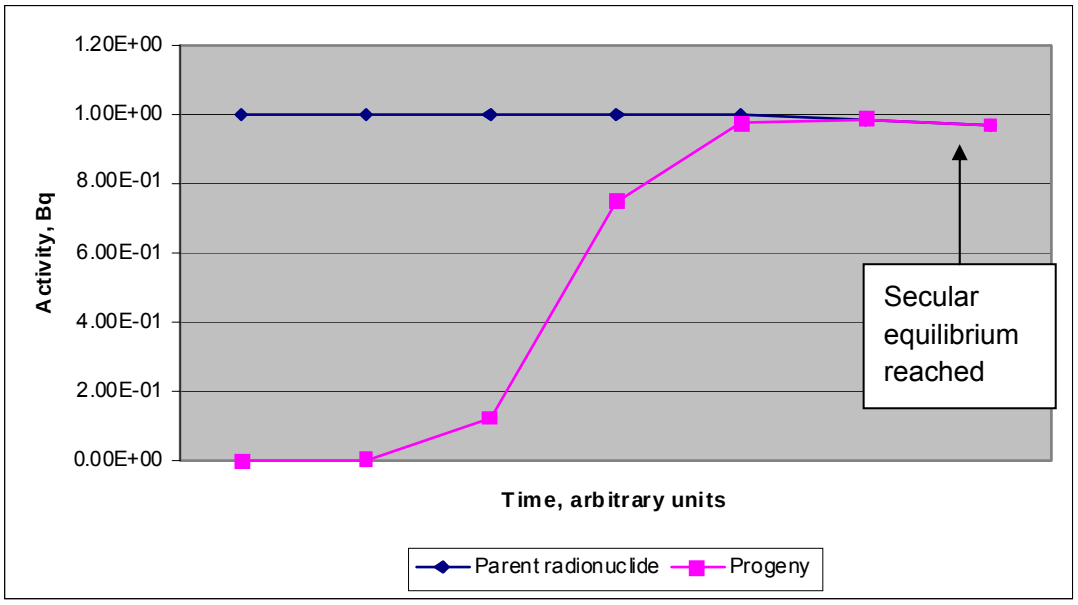

Figure 1.3 An example of a progeny radionuclide reaching secular equilibrium with its parent radionuclide

This state only occurs when the progeny has a short half-life when compared with the half-life of the parent radionuclide. Many factors can alter the relative amounts of activity within a decay chain found in the environment. For example if a chain contains a radionuclide that exists as a gas that radionuclide would be able to leave an area of radioactive contamination and any associated progeny would not be produced in that area. The measured activity of higher members of such a decay chain may therefore be very different to the activities of lower members due to this movement of radioactivity.

1.2 Dosimetric quantities

The fundamental dosimetric quantity in radiological protection is the **absorbed dose** which is the energy absorbed per unit mass. This quantity has the unit joules per kilogram, which is given the name Gray (Gy).

As discussed, ionising radiations differ in the way in which they interact with biological materials, so that equal absorbed doses (meaning equal amounts of energy deposited) do not necessarily have equal biological effects. For example, an alpha particle depositing 1 Gy to a tissue is more harmful than 1 Gy deposited by a beta particle as the alpha particle, being slower and more heavily charged, loses its energy much more densely along its path, therefore causing more localised damage. In order to put all ionising radiations on a equal basis with regard to their potential for causing harm the absorbed dose is multiplied by a factor termed the *radiation weighting factor* that accounts for the way a particular type of radiation distributes energy in a tissue. The absorbed dose multiplied by the radiation weighting factor results in a quantity termed the **equivalent dose**, which has the unit of Sievert (Sv). For gamma rays and beta particles the radiation weighting factor used is 1, while for alpha particles the factor is set as 20 (ICRP, 2007).

The equivalent dose can be further multiplied by a factor, termed the *tissue weighting factor*, which takes into account the risk associated with irradiating a particular tissue or organ within the body. For example, some organs are more susceptible to the risk of radiation-induced health effects such as cancer and these will have a higher tissue weighting factor than other organs. The quantity used to describe the absorbed dose weighted by both a *radiation weighting factor* and by the *tissue weighting factor* is called the **effective dose** which has the unit of Sievert (Sv). This is summarised in Figure 1.4.

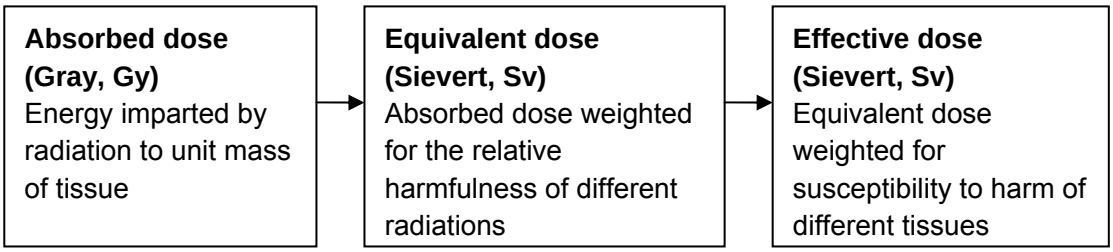

Figure 1.4 Summary of dose quantities

1.3 Radionuclides used in the old Physics buildings at the University of Manchester

From information about the work conducted in the old Physics buildings at the University of Manchester (Todd, 2008) it is thought that the majority of the radionuclides likely to

have been used during the early years of the last century were members of the natural decay series, that is, radionuclide members of the uranium-238, uranium-235 and thorium-232 decay chains. An introduction to these decay series is presented in the following sections. It is also thought that tritium, a radioactive form of hydrogen, may have been produced during the middle to late part of the last century.

1.3.1 Uranium and thorium decay chains

The decay chains headed by the radionuclides uranium-238, uranium-235 and thorium-232 are often called natural or primordial radionuclides as these decay chains exist naturally in the Earth. They were created in supernovae and subsequently incorporated within the developing Earth. Due to their long half-lives they are still found today in soil, rocks and other materials.

The full radioactive decay chains headed by the radionuclides uranium-238, uranium-235 and thorium-232 are given in Figures 1.5, 1.6 and 1.7, respectively, at the end of this paper. A summary of the most important radiological information about the radionuclide members of these decay chains, including radiological half-life, main radiation emissions and the energies of these emissions, is given in Tables 1.4, 1.5 and 1.6, also to be found at the end of this paper. It can be seen that these decay chains have a large number of radioactive progeny and that radioactive members of these decay chains produce a range of radiations including alpha and beta particles and gamma rays. The energy of these radiations varies from the relatively very low to the relatively very high. This means that the potential dose from exposure to radionuclides within these decay chains will be very dependent on which radionuclides are present and their relative activities.

From information about the work that was being carried out at Manchester (Todd, 2008) it is evident that several radionuclides within these decay chains were used in experimental work. For some of these radionuclides, the long half-life of either the chain header or one of its progeny will mean that, even though any potential spills of radioactivity may have occurred a hundred years ago, some radionuclides may still be present today. Other radionuclides used in work at Manchester may have undergone sufficient radioactive decay over the last 100 years such that there may not be any measurable trace of them left today. A review of the potential activity left today for each of these decay chains is given in Paper 2 of this report.

1.3.1.1 Natural uranium decay chains and its decay products

Generally, contamination by uranium ores such as pitchblende includes radionuclides in the entire uranium-238 chain, including radium-226, plus corresponding natural levels of radionuclides in the uranium-235 chain. Gamma and beta emissions from members of the decay chain are readily detectable, and some alpha emissions may also be detectable. Chemically-separated uranium has a much lower radium-226 level and produces lower gamma emissions than natural unprocessed ores and minerals. However, there would be some detectable gamma emissions as well as detectable beta emissions from protactinium-234m if there were large quantities of the material; the alpha emissions from uranium-238 and uranium-234 may also be detectable.

Thorium-230 (previously called “Ionium”) is essentially a pure alpha emitter (half-life 77,000 years) and is the parent of radium-226. “Ionium” preparations are understood to have been used at Manchester. However, these are likely to contain other isotopes of thorium and probably other radio-elements that are gamma and beta emitters, thus enabling contamination to be detected. Further, after nearly a hundred years, the in-grown radium-226 activity will be detectable.

Sources containing significant levels of actinium-227 were historically prepared from materials containing natural uranium. Actinium-227 has a half-life of 21.8 years and is in the uranium-235 decay series. Its relatively long half-life means that such activity may still persist even now. The presence of actinium-227 is detectable by the limited gamma emissions from its decay products. Additionally surface contamination measurements would detect the associated beta and alpha emissions. In addition, other contaminants might be present in the prepared actinium, such as radium and thorium. Detection of actinium-227 might also imply the presence of its immediate parent, protactinium-231, which also has a long half-life.

1.3.1.2 Radium and its shortlived decay products

Any residual contamination by radium-226 (half-life 1600 years) could clearly remain and would occur with its immediate short lived decay products as well as its longer-lived decay products lead-210, bismuth-210 and polonium-210. The ratio of lead-210 to radium-226 in such contamination will vary depending on the age of the contamination and on the proportion of radon-222 gas escaping from the contaminated materials. For well-fixed and sealed contamination (eg, activity present under layers of paint) radon loss might be relatively limited and in the worst case the activity levels for the long-lived products would be the same as that of the parent radium.

Radium contamination is easily detectable by the gamma emissions from several of the radium decay products, and surface contamination is usually detectable by the beta emissions and often also the alpha emissions.

1.3.1.3 Radon

Isotopes of radon are present in all of the natural decay chains. Radon-222 is part of the uranium-238 decay chain, radon-220 is part of the thorium-232 decay chain and radon-219 is part of the uranium-235 chain. Radon-222 has the longest half-life of these radon isotopes at 3.82 days. Radon-220 has a half-life of 56 seconds whilst the half-life of radon-219 is 4 seconds. Of the three isotopes, radon-222 is the most significant as its relatively long half-life enables it to migrate quite significant distances within the geological environment before decaying.

HPA radon detectors, which have been used by the University of Manchester to measure the radon levels throughout the campus, are designed to measure radon-222 as the half-life for entry into the detector (25 minutes) is short compared with the radon-222 half-life, but long compared to the half-lives of radon-220 and radon-219.

In general a progeny of a radionuclide will be found in the same place as the radionuclide from which it formed. As such whole decay chains may be present in an

area. However, radon is an inert gas and is able to leave the surface of objects and enter the atmosphere. This has the effect of potentially disrupting the balance of activity along the decay chain as radon, and its progeny, may move to an area which is remote from the higher members of the decay chain. The amount of activity lost from an area when radon is formed and enters the atmosphere is hard to estimate.

The amount of radon able to leave a material depends on a number of parameters such as the porosity of the material, reflecting how easy it is for radon gas to enter air, and the movement of air directly above the surface of the material, which relates to how fast any gas created within a material can leave that material. Therefore it is impossible to give a general value for the amount of radioactivity that could be lost to atmosphere with the creation of radon as this is very site dependent.

Once in the air radon disperses like any other gas. If the gas is located within a building that has poor ventilation then radon can concentrate at high levels. However, if the building has high ventilation, for example if the windows are open, then radon can leave the building and the build up of radon, and its decay products, will be much reduced.

1.3.1.4 Lead-210 supported activity

The relatively long (22.3 years) half-life of lead-210, known also as “Radium-D”, means that if this isotope had been separately extracted then, even after a long time interval, there could still be lead-210 and its decay products at higher levels than any surrounding traces of radium-226 activity. Of some importance here is that that the decay chain lead-210/bismuth-210/polonium-210 does not have significant gamma emissions and so lead-210 contamination cannot be detected using these. The types of gamma-radiation survey meters employed for this type of work have a low detection efficiency for lead-210 gamma rays (energy 47 keV) and are therefore not adequate for the detection of lead-210.

Where present as surface contamination, lead-210 supported activity is normally detectable from the beta emission from bismuth-210 and, possibly, the polonium-210 alpha emission (but see later comments on absorption). Bismuth-210 will be in equilibrium with lead-210 and polonium-210. The expected equilibrium between lead-210 and polonium-210 means that polonium-210 provides a “marker” for possible lead-210 contamination, which is useful because laboratory radiochemical techniques more easily determine polonium-210 than lead-210. The half-life of polonium-210 is 138 days. Separated polonium-210 (or “Radium F”) activity is no longer replenished by the decay of its parent, lead-210, so it will decay away completely in less than ten years.

Lead-210 contamination might arise from direct spills of prepared lead-210 solutions or other materials containing lead-210, but it could also arise by “plate out” in poorly ventilated areas which are used to store “leaky” radium-226 sources for long periods; the vector for this contamination is radon-222 gas emitted by the radium. However, the potential importance of this transport mechanism in a large building is unclear. The effect is seen in small cabinets and containers but the deposited activity, while detectable, tends to be only a very small fraction of the parent radium activity.

1.3.1.5 Natural thorium and its decay products

Any thorium activity found would be expected now to be thorium-232 in essentially complete equilibrium with all its decay products, including the radium-228/actinium-228/thorium-228 sequence. Thorium is readily detectable by gamma emissions from various radionuclides in the thorium-232 decay chain, and any surface contamination would have associated beta and alpha emissions. Given the elapsed time, had radium-228 (half-life 5.8 years) been separated from materials containing thorium the excess radium activity would have decayed by now to essentially the same level as any remaining supporting thorium-232 activity. Any separated thorium-228 activity (half-life 1.9 years) would have completely decayed within a few tens of years.

1.3.2 Tritium

The possibility of tritium contamination at the University of Manchester has been mentioned in reports detailing the work of the physics department (Todd, 2008). Tritium, which is a radioactive isotope of hydrogen with a half-life of approximately 12 years, emits only weak beta particles and does not generally contribute significantly to radiation exposures unless large quantities are present, which does not appear to be the case at Manchester. It has therefore been included in this report for completeness rather than because it is likely to pose a significant radiological hazard.

Tritium was potentially created in the early 1960s when linear accelerators were installed at the University of Manchester. The use of this equipment may have continued until the 1970s. Assuming that only radioactive decay affected the loss of tritium then the amount of tritium present today would be between two and four times less than that present at its creation. However, tritium is very mobile in the environment as it behaves like water. This means that, in addition to the loss of activity through radioactive decay, there would be losses associated with evaporation which would be very significant over timescales of many tens of years. The overall effect of evaporation and radioactive decay is that there is unlikely to be any significant amounts of tritium present today apart from that which is present naturally.

1.4 Natural levels of radioactivity

This section provides information about natural levels of radioactivity of the decay chains headed by uranium and thorium found in the UK. Table 1.1 summarises some of the information given.

Table 1.1 Some examples of natural levels of radioactivity

Material	Levels	
Soil (UK average)	40 Bq kg^{-1} uranium-238 decay chain	25 Bq kg^{-1} thorium-232 decay chain
(UK range)	2 – 330 Bq kg^{-1} uranium-238 decay chain	1 – 180 Bq kg^{-1} thorium-232 decay chain
Mussels	42 Bq kg^{-1} radium isotopes	
Brazil nuts	30 Bq kg^{-1} radium isotopes	
Radon (UK average outdoor)	4 Bq m^{-3}	
(UK average indoor)	20 Bq m^{-3}	
(UK highest - indoor)	17 000 Bq m^{-3}	

1.4.1 Uranium and thorium

In the UK, the average activity concentration of members of the uranium-238 decay chain in soil is 40 Bq kg^{-1} and that for the thorium-232 decay chain is 25 Bq kg^{-1} (UNSCEAR, 2000). However, there is a wide range in these activity concentrations across the UK with the range for uranium-238 activity concentration being between 2 and 330 Bq kg^{-1} whilst that for thorium-232 being between 1 and 180 Bq kg^{-1} (UNSCEAR, 2000). As these radionuclides are present in soil they are able to be taken up by plants and can then be ingested by humans. Some members of these decay chains can be more concentrated in certain foodstuffs as a result of natural processes and some examples of the activity concentrations in some common foods grown in the UK are given in Table 1.2.

Table 1.2 Typical activity concentrations, Bq kg^{-1}, in common foodstuffs found in the UK for members of the natural decay chains (from Watson et al, 2005)

	^{238}U	^{234}U	^{230}Th	^{226}Ra	^{210}Pb	^{210}Po	^{232}Th	^{228}Ra	^{228}Th	^{235}U
Nuts	$6.2\ 10^{-3}$	$7.0\ 10^{-3}$	$1.0\ 10^{-2}$	$9.4\ 10^{-2}$	$1.1\ 10^{-1}$	$2.9\ 10^{-2}$	$3.0\ 10^{-3}$	$9.4\ 10^{-2}$	$3.0\ 10^{-3}$	$1.0\ 10^{-3}$
Potatoes	$4.8\ 10^{-3}$	$5.0\ 10^{-3}$	$6.0\ 10^{-3}$	$2.3\ 10^{-2}$	$1.6\ 10^{-2}$	$9.0\ 10^{-3}$	$3.0\ 10^{-3}$	$2.0\ 10^{-2}$	$5.0\ 10^{-4}$	$1.0\ 10^{-4}$
Root vegetables	$1.2\ 10^{-2}$	$7.0\ 10^{-3}$	$9.7\ 10^{-3}$	$6.0\ 10^{-2}$	$3.0\ 10^{-2}$	$2.1\ 10^{-2}$	$7.0\ 10^{-3}$	$2.0\ 10^{-2}$	$5.0\ 10^{-4}$	$1.0\ 10^{-4}$
Green vegetables	$9.8\ 10^{-3}$	$4.9\ 10^{-3}$	$6.0\ 10^{-3}$	$9.6\ 10^{-3}$	$3.1\ 10^{-2}$	$9.0\ 10^{-2}$	$4.0\ 10^{-3}$	$4.0\ 10^{-2}$	$1.5\ 10^{-2}$	$1.0\ 10^{-3}$
Cattle meat	$4.9\ 10^{-3}$	$2.0\ 10^{-3}$	$2.0\ 10^{-3}$	$1.2\ 10^{-2}$	$7.2\ 10^{-2}$	$1.1\ 10^{-1}$	$1.0\ 10^{-3}$	$1.0\ 10^{-2}$	$1.0\ 10^{-3}$	$5.0\ 10^{-5}$
Milk	$1.2\ 10^{-4}$	$1.0\ 10^{-3}$	$5.0\ 10^{-4}$	$3.0\ 10^{-3}$	$3.5\ 10^{-2}$	$1.5\ 10^{-2}$	$3.0\ 10^{-4}$	$5.0\ 10^{-3}$	$3.0\ 10^{-4}$	$5.0\ 10^{-5}$
Cheese and butter	$4.9\ 10^{-3}$	$2.0\ 10^{-3}$	$2.0\ 10^{-3}$	$5.6\ 10^{-2}$	$7.2\ 10^{-2}$	$1.1\ 10^{-1}$	$1.0\ 10^{-3}$	$1.0\ 10^{-2}$	$5.6\ 10^{-2}$	$5.0\ 10^{-5}$
Cereals	$6.2\ 10^{-3}$	$7.0\ 10^{-3}$	$1.0\ 10^{-2}$	$5.2\ 10^{-2}$	$9.3\ 10^{-2}$	$2.9\ 10^{-2}$	$3.0\ 10^{-3}$	$6.0\ 10^{-2}$	$3.0\ 10^{-3}$	$1.0\ 10^{-3}$
Drinking water	$1.0\ 10^{-3}$	$1.0\ 10^{-3}$	$1.0\ 10^{-4}$	$5.0\ 10^{-4}$	$1.0\ 10^{-2}$	$5.0\ 10^{-3}$	$5.0\ 10^{-5}$	$5.0\ 10^{-4}$	$5.0\ 10^{-5}$	$4.0\ 10^{-5}$

Using radionuclide activity concentrations in foods, such as those presented in Table 1.2, an average dose to members of the UK population has been estimated (Watson et al, 2005). The most significant contributor to the dose from the ingestion of these foods was from radionuclide members of the uranium decay chains, notably lead-210 and polonium-210. The average annual dose to a member of the UK population from the presence of uranium and its decay products in food is approximately 67 micro Sv (μSv) whilst that from the ingestion of thorium and its decay products is approximately 7 μSv.

In some foods certain radionuclides are found to concentrate at relatively high levels. Mussels, for example, can contain relatively high concentrations of polonium-210 with the average concentration found in a study carried out in 2001 being 42 Bq kg^{-1} (EA et al, 2004). The consumption of an 80 g jar of mussels containing this activity concentration of polonium-210 would lead to a dose of approximately 4 μSv. If such a jar of mussels was consumed each week for a year the annual dose received would be about 200 μSv.

Brazil nuts can contain elevated levels of radium isotopes (Turner et al, 1958)), sometimes up to a few hundred Becquerel per kilogram. A study (Hiromoto et al, 1996) found average concentrations of about 30 Bq kg^{-1} of both radium-226 and radium-228. The consumption of 100 g of these nuts could give rise to a dose of about 4 μSv, or 200 μSv per annum if such an amount was consumed each week.

1.4.1.1 Radon and thoron

Radon in air

Radon, as it is a gas, moves freely with the atmosphere and so its activity concentration is not able to build up significantly in outside air. The population-weighted average concentration of radon in outdoor air has been estimated as 4 Bq m^{-3} (Wrixon et al, 1988).

Radon in buildings

Radon tends to concentrate in buildings as the radon is drawn into them from the ground by processes such as indoor heating, where the air rises and the radon-laden air is pulled into the building from the ground, and the action of wind passing over the building. The main source of indoor radon is from the ground below, but it can also be carried into homes in the water and natural gas supplies, and released when these are used. Also, uranium in building materials can be a minor source of radon.

The activity concentration of radon in buildings is very variable as the concentration will depend on the ventilation of the building, what the building is made of, and the type of material on which the building was built as some soils and rocks contain a much higher activity of radium-226 than others. Due to this large range in activity concentrations an Action Level is used in the UK to determine whether action needs to be taken to reduce the radon concentration. For buildings used as dwellings the UK Action Level is set at annual average radon concentration of 200 Bq m^{-3} (DoE, 1990). For buildings that are above ground and that are used as places of work a 24-hour average radon activity concentration above 400 Bq m^{-3} will mean that the Ionising Radiation Regulations 1999

applies (HMSO, 1999). The higher value for work buildings is set to account for the lower occupancy of these buildings compared with dwellings.

A number of surveys have been carried out since the early 1980s to investigate the radon activity concentration in houses in the UK, for example see Wrixon et al (1988) and Green et al (2002). These surveys have found that the average indoor radon activity concentration in the UK is 20 Bq m^{-3}. Based on these surveys it is thought that about 0.5% of the UK housing stock, that is some 100,000 homes, have an average annual radon activity level above 200 Bq m^{-3} (NRPB, 1990). The highest radon activity concentrations so far found in domestic dwellings are for two houses in Cornwall, one with 17,000 Bq m^{-3} and the other with 12,000 Bq m^{-3} (NRPB , 2004).

Doses from radon and thoron

Radon decays into a series of radionuclides that can become attached to particles of dust in indoor air. Inhalation of radon gas and its decay products gives rise mainly to a dose to the lungs, which is almost all from the decay products rather than radon gas itself. The dose to the lungs can be converted into an effective dose, using the appropriate weighting factor. This can then be expressed as an effective dose rate per unit of radon gas concentration. However, this quantity depends on many factors, such as the degree of equilibrium between radon gas and its decay products, the fraction of decay products that are attached to dust particles and the size of particles to which those radionuclides are attached.

For dwellings a conversion convention (NRPB, 1987) was adopted that gives rise to a dose of about 1 mSv y^{-1} at the UK average indoor concentration of 20 Bq m^{-3}. Taking the UK as a whole, the population-weighted average annual dose from radon-222 is around 1.2 mSv, with individual annual doses varying from about 0.3 mSv to a few hundred millisieverts in homes with the highest radon levels. In the dwelling with the highest radon concentration found, at 17,000 Bq m^{-3}, the annual dose was estimated to be approximately 850 mSv.

In addition to radon exposure there is a small annual dose from thoron (radon-220), which was estimated to be 0.1 mSv on average in the UK (Cliff, 1996), with a range between about 0.05 to 0.5 mSv. Some recent measurements (Proctor, 2004) of thoron in homes at a number of locations in the UK support this general range, and suggest that in homes with the highest thoron levels, annual doses from this source can be around 1 mSv. The average annual dose from both radon isotopes in the UK is therefore about 1.3 mSv (1,300 µSv).

1.4.2 Doses from exposure to natural radionuclides

The average annual dose in the UK from all natural sources is about 2,230 µSv. In addition to the doses discussed in the previous sections, other natural sources of exposure include that from other naturally occurring radionuclides such as potassium-40 which contributes significantly to the dose from the ingestion of food, and that from cosmic radiation. Cosmic radiation originates from outside the atmosphere of the Earth and is composed of energetic particles. These particles pass through the atmosphere and irradiate the entire surface of the planet. A summary of the annual average dose to

a member of the UK population is presented in Table 1.3. From this table it is evident that exposure to radon provides the most significant contribution to the average dose.

Table 1.3 Summary of doses to the UK adult population from natural sources (from Watson et al, 2005)

	Average annual UK dose (μSv)	Range (μSv)
Cosmic radiation	330[a]	200 – 400[b]
Terrestrial gamma radiation	350	100 – 1000
Internal radionuclides	250	100 – 1000
Radon[c] (radon-222)	1,200	300 – 100,000
Thoron[c] (radon-220)	100	50 – 500
Total	2,230	1,000 – 100,000

(a) Including an additional 30 μSv from air travel. It should be noted that not all of the population is exposed to this source.

(b) Range does not include air travel.

(c) Including decay products.

1.5 Uranium-238 decay chain

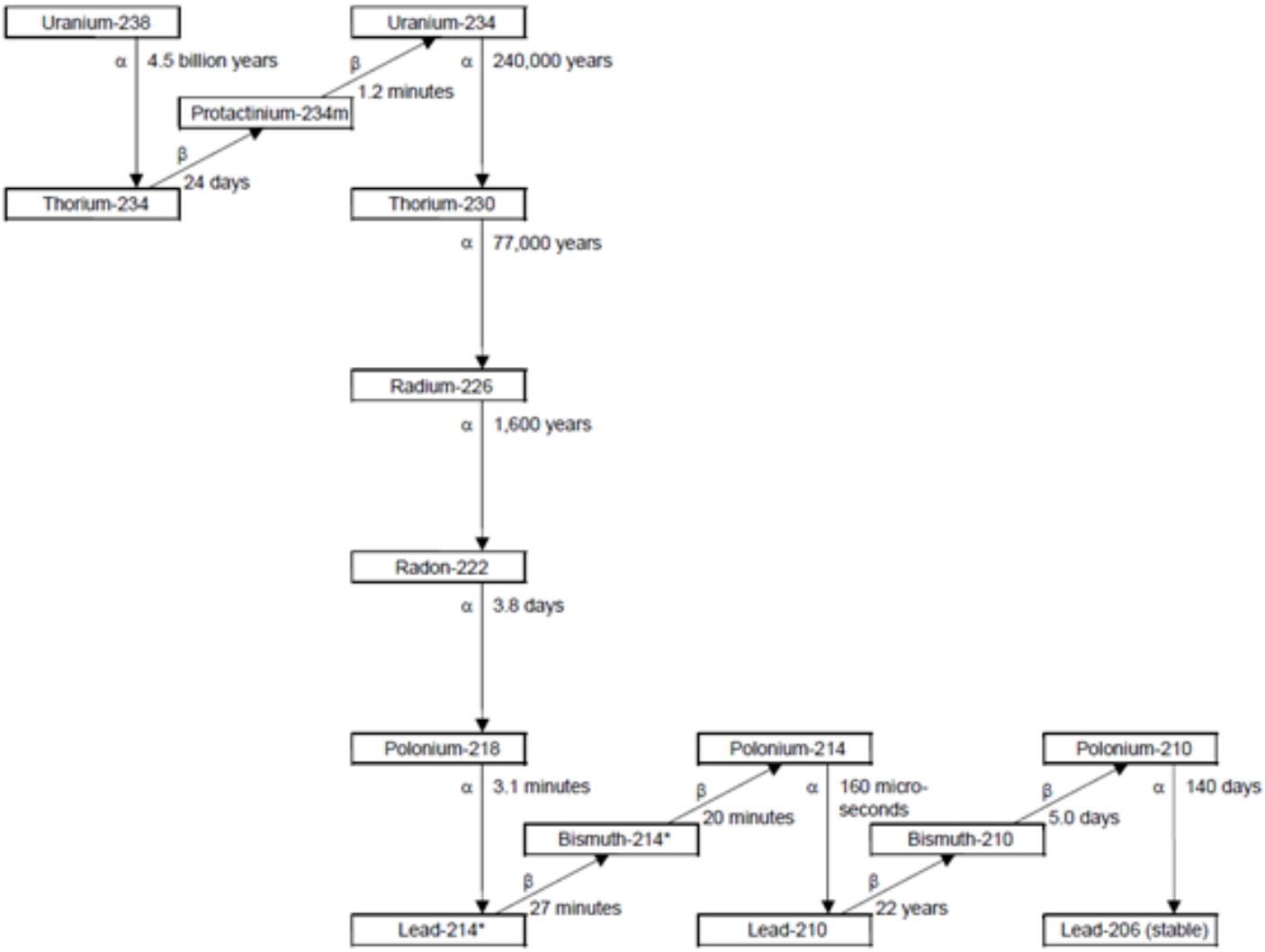

Figure 1.5 The uranium-238 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown for clarity.

Table 1.4 Radiological information about members of the uranium-238 decay chain

Radionuclide	Historic name	Half-life	Decay mode (MeV)† Intensity (%)*	Gamma-ray (keV) intensity (%)*	Product of decay
^{238}U	Uranium	4.5 10^{9} a	α 4.198 (79%) 4.151 (21%)		^{234}Th
^{234}Th	Uranium X1	24.1 d	β 0.199 (70%) 0.107 (19%) 0.106 (8%)	63.3 (5%) 92.4 (3%) 92.8 (3%)	^{234}Pa (0.2%) ^{234m}Pa (99.8%)
^{234}Pa	Uranium Z	6.7 h	β 0.472 (45%) 0.642 (19%) 0.413 (8%)	131.3 (18%) 946.0 (13%) 883.2 (10%)	^{234}U
^{234m}Pa	-	1.2 min	β 2.269 (98%)	1001.0 (1%)	^{234}U (99.87%) ^{234}Pa (0.13%)
^{234}U	Uranium two	2.5 10^{5} a	α 4.775 (71%) 4.722 (28%)		^{230}Th
^{230}Th	Ionium	7.5 10^{4} a	α 4.687 (76%) 4.621 (23%)		^{226}Ra
^{226}Ra	Radium	1. 6 10^{3} a	α 4.784 (94%) 4.601 (5%)	186.2 (4%)	^{222}Rn
^{222}Rn	Radon	3.8 d	α 5.490 (100%)		^{218}Po
^{218}Po	Radium A	3.1 min	α 6.002 (100%)		^{214}Pb (99.98%) ^{218}At (0.02%)
^{218}At	-	1.5 s	α 6.693 (90%) 6.653 (6%) 6.756 (4%)		^{214}Bi
^{214}Pb	Radium B	26.8 min	β 1.024 (6%) 0.729 (42%) 0.672 (49%)	351.9 (38%) 295.2 (19%) 242.0 (7%)	^{214}Bi
^{214}Bi	Radium C	19.9 min	α 5.516 (39%) 5.452 (54%) 5.273 (6%) β 3.272 (18%) 1.542 (18%) 1.507 (17%)	609.3 (46%) 1764.5 (15%) 1120.3 (15%)	^{214}Po
^{214}Po	Radium C'	164.3 μs	α 7.687 (100%)		^{210}Pb
^{210}Pb	Radium D	22.3 a	α 3.720 (100%) β 0.017 (84%) 0.063 (16%)	46.5 (4%)	^{210}Bi
^{210}Bi	Radium E	5.0 d	α 4.656 (60%) 4.694 (40%) β 1.162 (100%)		^{210}Po
^{210}Po	Radium F	138.4 d	α 5.304 (100%)		^{206}Pb

† For beta particles, maximum energy of the particle is given

* Only the 3 highest intensity decays or emissions are shown. Have rounded intensity to nearest significant figure and have only shown if 1% or greater.

1.6 Thorium-232 decay chain

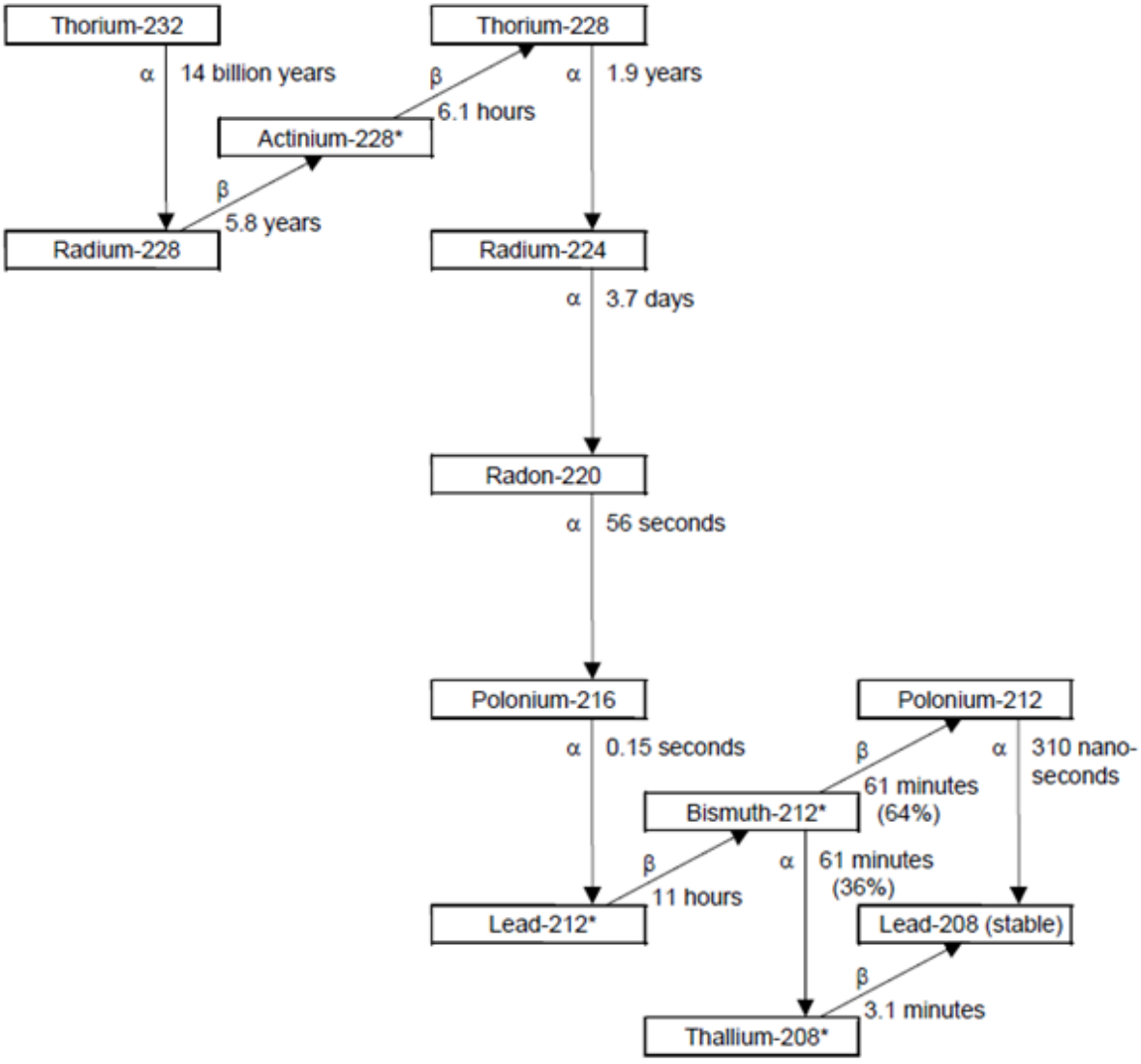

Figure 1.6 The thorium-232 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown for clarity.

Table 1.5 Radiological information about members of the thorium-232 decay chain

Radionuclide	Historic name	Half-life	Decay mode (MeV)† Intensity (%)*	Gamma-ray (keV) Intensity (%)*	Product of decay
^{232}Th	Thorium	1.4 10^{10} a	α 4.012 (78%) 3.947 (22%)	63.8 (26%) 140.9 (2%)	^{228}Ra
^{228}Ra	Mesothorium 1	5.8 a	β 0.039 (40%) 0.013 (30%) 0.026 (20%)	13.5 (2%)	^{228}Ac
^{228}Ac	Mesothorium 2	6.2 h	β 1.158 (30%) 1.731 (12%) 2.069 (8%)	911.2 (26%) 969.0 (16%) 338.3 (11%)	^{228}Th
^{228}Th	Radiothorium	1.9 a	α 5.423 (72%) 5.340 (27%)	84.4 (1%)	^{224}Ra
^{224}Ra	Thorium X	3.7 d	α 5.685 (95%) 5.449 (5%)	241.0 (4%)	^{220}Rn
^{220}Rn	Thoron	55.6 s	α 6.288 (100%)		^{216}Po
^{216}Po	Thorium A	0.1 s	α 6.778 (100%)		^{212}Pb
^{212}Pb	Thorium B	10.6 h	β 0.335 (83%) 0.574 (12%) 0.159 (5%)	238.6 (43%) 300.1 (3%)	^{212}Bi
^{212}Bi	Thorium C	60.6 min	α 6.051 (70%) 6.090 (27%) 5.768 (2%) β 2.254 (55%) 1.527 (4%) 0.633 (2%)		^{212}Po (64.06%) ^{208}Tl (35.94%)
^{212}Po	Thorium C'	0.3 μs	α 8.784 (100%)		^{208}Pb
^{208}Tl	Thorium C"	3.1 min	β 1.803 (49%) 1.293 (25%) 1.526 (22%)	2,614.5 (99%) 583.2 (85%) 510.8 (23%)	^{208}Pb

† For beta particles, maximum energy of the particle is given

* Only the 3 highest intensity decays or emissions are shown. The intensity is rounded to nearest significant figure and is only shown if 1% or greater.

1.7 Uranium-235 decay chain

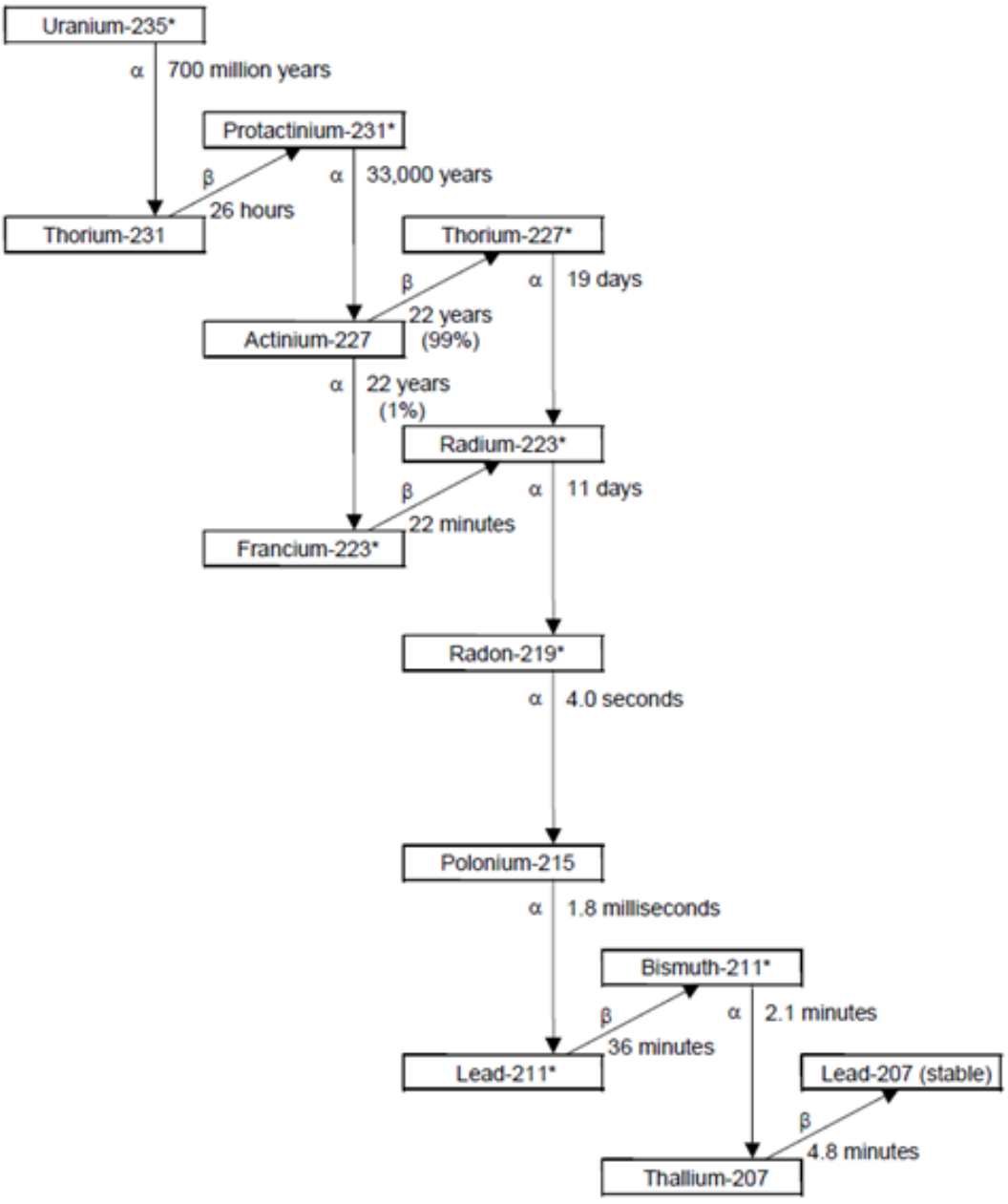

Figure 1.7 The uranium-235 decay chain. The symbols α and β indicate alpha and beta decay with the asterisk indicating if the radionuclide is also a significant gamma emitter. The times shown are the half-lives. Progeny that exist with less than 1% of their parent's activity, as a result of branching, are not shown for clarity.

Table 1.6 Radiological information about members of the uranium-235 decay chain

Radionuclide	Historic name	Half-life	Decay mode (MeV)† Intensity (%)*	Gamma-ray (keV) Intensity (%)*	Product of decay
^{235}U	Actin Uranium	7.0 10^{8} a	α 4.398 (55%) 4.366 (17%) 4.215 (6%)	185.7 (57%) 143.8 (11%) 163.4 (5%)	^{231}Th
^{231}Th	Uranium Y	25.5 h	β 0.288 (37%) 0.305 (35%) 0.206 (13%)	25.6 (15%) 84.2 (7%)	^{231}Pa
^{231}Pa		3.3 10^{4} a	α 5.014 (25%) 4.951 (23%) 5.028 (20%)	27.4 (10%) 300.1 (2%) 302.7 (2%)	^{227}Ac
^{227}Ac	Actinium	21.8 a	α 4.953 (48%) 4.941 (40%) 4.873 (6%) β 0.020 (10%) 0.036 (35%) 0.045 (54%)		^{227}Th (98.62%) ^{223}Fr (1.38%)
^{227}Th	Radioactinium	18.7 d	α 6.038 (24%) 5.978 (24%) 5.757 (20%)	236.0 (12%) 50.1 (8%) 256.3 (7%)	^{223}Ra
^{223}Fr	Actinium K	21.8 min	α 5.340 (100%) β 1.099 (67%) 1.069 (16%) 0.914 (10%)	50.1 (36%) 79.7 (9%) 234.8 (3%)	^{223}Ra
^{223}Ra	Actinium X	11.4 d	α 5.716 (53%) 5.607 (26%) 5.747 (9%)	269.5 (14%) 154.2 (6%) 323.9 (4%)	^{219}Rn
^{219}Rn	Actinon	4.0 s	α 6.819 (79%) 6.553 (13%) 6.425 (8%)	271.2 (11%) 401.8 (6%)	^{215}Po
^{215}Po	Actinium A	1.8 ms	α 7.386 (100%)		^{211}Pb
^{215}At		0.1 ms	α 8.026 (100%)		^{211}Bi
^{211}Pb	Actinium B	36.1 min	β 1.372 (91%) 0.540 (6%) 967.2 (2%)	404.9 (4%) 832.0 (4%) 427.1 (2%)	^{211}Bi
^{211}Bi	Actinium C	2.1 min	α 6.623 (84%) 6.278 (16%)	351.1 (13%)	^{207}Tl (99.72%) ^{211}Po (0.28%)
^{211}Po	Actinium C'	0.5 s	α 7.450 (99%)		^{207}Pb
^{207}Tl	Actinium C"	4.8 min	β 1.423 (100%		^{207}Pb

† For beta particles, maximum energy of the particle is given.

* Only the 3 highest intensity decays or emissions are shown. The intensity is rounded to nearest significant figure and is only shown if 1% or greater.

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2 PAPER 2: EXPOSURE PATHWAYS

WB OATWAY AND KA JONES

2.1 Introduction

Different routes by which people can be exposed to radiation are termed exposure pathways. Exposure pathways for radiation can be divided into two groups for convenience; those pathways where radionuclides exist outside the body and those pathways where exposure is to radionuclides that have been taken into the body. The relative importance of different exposure pathways depends on the nature of the radionuclide (including the type of radiation released and its half-life) plus its behaviour in the environment of interest. When assessing the dose to an individual the doses from each exposure pathway are assessed and then the dose is summed over all pathways in order to determine the total dose.

2.2 External irradiation

Radionuclides located outside the body are able to expose internal organs if the emitted radiation is able to first transverse the air between the radionuclide and the body and then pass through the skin. The radiation of concern with this pathway is gamma radiation, although high energy beta particles may also be important for exposure to the skin. The level of exposure a body receives from radionuclides located outside of the body is dependent on several parameters.

- Energy of the radiation: The higher the energy of the emitted radiation then the higher the dose received.
- Distance: The further away a source is located with respect to a body then the lower is the dose.
- Time: The shorter the time a body is exposed to a source of radioactivity then the lower is the dose received.

The first two factors can be combined to form a parameter termed a “dose rate”, which is the amount of dose received per unit time, usually expressed in µSv per hour. Generally this value would be measured and this measured value used in a dose assessment. However, where this is not possible then it can be estimated using models that take account of which radionuclides are present, how they are distributed and the location of the exposed individual. When the dose rate is combined with an exposure time, representing how long an individual was assumed to be present in an area where radionuclides are present, a total dose can be estimated. That is:

Dose (µSv) = Time (hours) * dose rate (µSv per hour)

For the assessment of doses at the University of Manchester, the exposure time is important as exposure would only occur whilst individuals were at work, and even then most of the exposure might only have occurred when the individual was located within a few rooms. This means that exposure was only likely to occur for a fraction of the total year.

External irradiation from radioactive materials located outside of the body is shown in Figure 2.1 for radioactive sources that emit gamma rays or beta particles.

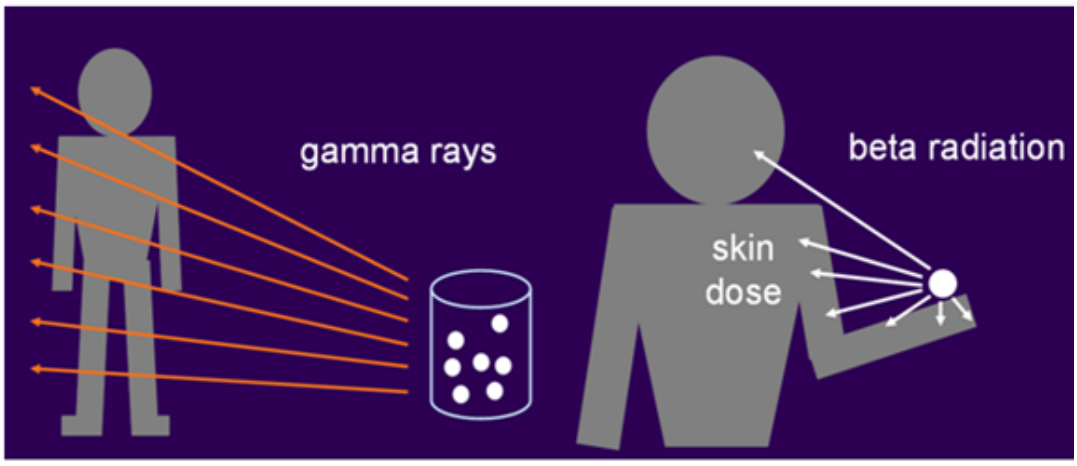

Figure 2.1 External irradiation by radionuclides located outside the body

2.3 Internal irradiation

Radionuclides will enter the body after they have been inhaled or ingested; this internal irradiation is shown in Figure 2.2. Inhalation occurs when radioactivity is present in the air. This could occur when the radionuclide is of a form that is able to become airborne, for example a gas such as radon, or when a radionuclide becomes associated with, for example, dust that then becomes airborne. An assessment of the dose from this pathway would consider how the radionuclide became available for inhalation, for example how dust particles become suspended, as well as what happens to the radionuclide once it entered the body.

Normally the most important pathway for radionuclide ingestion is when radionuclides get incorporated in food. If, for example, soil is contaminated with radioactivity then plant roots may take up the radioactivity and move it to parts of the plant that get ingested, such as fruit. However, at the University of Manchester, this would not be the case as no food is grown in the areas thought to be contaminated. Therefore the only mechanism for ingestion of radioactivity is when radionuclides become associated with dust or other material that may get onto food or other objects that get placed in the mouth, such as pens or fingers. For example, if food is placed on a table that has radionuclides on it then the radionuclides may become attached to the food and would then be subsequently ingested when the food is ingested.

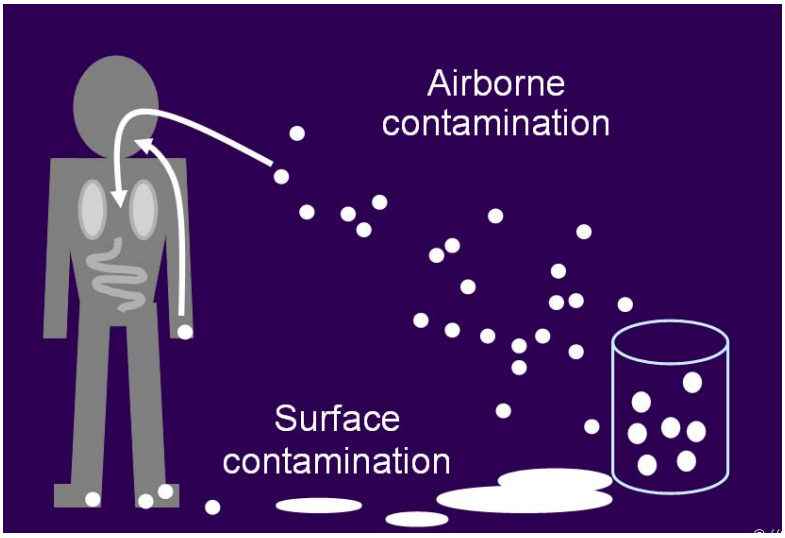

Figure 2.2 Internal irradiation by radionuclides taken into the body via inhalation and ingestion

The irradiation of the body and its various organs from incorporated radionuclides can be determined as the product of these intakes and an appropriate dose per unit intake conversion factor, termed a dose coefficient. The dose coefficients depend on the radionuclide, the exposure route (inhalation/ingestion), the chemical form of the radionuclide and the age of the person. Various compilations of dose coefficients are available with the ones recommended for use in the UK being from publications of the International Commission on Radiological Protection (ICRP, 1996). An overview of the models used to calculate these dose coefficients is provided in Paper 3.

Dose (Sv) = Activity taken in by the body (Bq) * dose coefficient (Sv Bq^{-1})

2.4 Radiologically significant members of the uranium and thorium decay chains

This section reviews the radioactivity of members of the decay chains which could have been used at Manchester. It includes the potentially important radionuclides, in terms of dose, that may have been present between 50 and 100 years after the time of Rutherford with respect to the pathways of external irradiation, ingestion and inhalation. This timeframe corresponds to approximately the year 1960 to the early years of the 21st century. This timeframe was selected to be representative of the time of interest in this study. The potential contribution to each of the exposure pathways (inhalation, ingestion and external irradiation) is given separately at this stage and is intended to give an indication of the relative importance of the given radionuclides. The potential contribution of each of the radionuclides will vary because their dose coefficients differ. The tables in the following sections also give an indication of when the radionuclide is likely to contribute significantly to the dose. For example it may take some time for a

radionuclide in the decay chain to grow in and therefore it will only contribute to the dose after several decades.

It should be noted that this paper only provides an indication of the potential radionuclides and exposure pathways of concern for a dose assessment of the period above. It is intended that Task 2 (Assessment of past risks from radioactive contamination) and Task 3 (Assessment of current and future risks from radioactive contamination) will give a more refined estimate of the potential risks from any particular radionuclide or pathway.

Given the large uncertainty in the fate of any radon gas created during the decay process (see Paper 1 of this report) it is cautiously assumed in the following discussion, which looks at whole decay chains, that no radioactivity is lost and that the effect on the decay chain activity would be minimal, that is, the effects of activity leaving the area because of radon gas is assumed to be zero. Lower members of the decay chain are therefore assumed to grow in, in the area where higher members of the decay chain are located. Given sufficient time, therefore, the entire decay chain will be able to reach a state of secular equilibrium. Given that the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc its relative importance is not given in the following sections. However the dose from radon will be considered in the detailed assessment done for Tasks 2 and 3.

2.4.1.1 Assuming uranium-238 was present at Manchester

If pure uranium-238 was used then within 100 years only uranium-238, thorium-234 and protactinium-234/protactinium-234m would be likely to be present with any significant activity. The next radionuclide in the decay chain, uranium-234, is likely to be present with an activity over 3000 times less than that for the higher decay chain members. However Rutherford and his colleagues would not have been able to separate uranium-238 from uranium-234 and therefore uranium-234 would also be present.

Over this timescale it is likely that the radionuclides from uranium-238 to protactinium will have reached a state of secular equilibrium. As the parent radionuclide, uranium-238, has a half-life of approximately 4.5 billion years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.1.

Table 2.1: Indication of the relative importance of radionuclide members of the decay chain headed by uranium-238 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	**^{238}U**	**All**	**100%**
Ingestion	**^{238}U**	**All**	**95%**
	^{234}Th	After 6 months	5%
External irradiation	^{238}U	All	25%
	^{234}Th	After a few days	15%
	^{234m}Pa[f]	**After a few days**	**60%**

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

(f) m = metastable. Most excited states in atoms are relived in less than 10^{-9}s or so by gamma emission. However some excited states may exist long enough to be readily measurable. A delayed release of energy represents a metastable state.

2.4.1.2 Assuming uranium-234 was present at Manchester

Within 100 years no radionuclide member of this decay chain is expected to be in secular equilibrium with the uranium-234. However, some decay chain members are radiologically significant when compared to the uranium-234 despite being present with an activity that could be up to a million times less.

As the parent radionuclide, uranium-234, has a half-life of approximately 25,000 years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.2.

Table 2.2: Indication of the relative importance of radionuclide members of the decay chain headed by uranium-234 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	**^{234}U**	**All**	**100%**
Ingestion	**^{234}U**	**All**	**100%**
External irradiation	**^{234}U**	**All**	**70%**
	^{214}Pb	After a few tens of years	5%
	^{214}Bi	After a few tens of years	25%

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.1.3 Assuming thorium-230 was present at Manchester

Within 100 years the progeny of thorium-230, radium-226, could potentially ingrow to around 2% of the activity of the parent radionuclide. The effect on the activity of lower decay chain members of radon-222 emanation is hard to assess and the cautious assumption to make is to ignore emanation. Therefore the progeny of radon-222, to lead-210, could be assumed to be in secular equilibrium with the radium-226. Lead-210, and its progeny of bismuth-210 and polonium-210, is likely to be present at a lower activity due to the relatively long half-life of lead-210 and in this case, after 100 years, is potentially present with an activity of about 30 times less than that of the thorium-230.

As the parent radionuclide, thorium-230, has a half-life of approximately 80 000 years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.3.

Table 2.3: Indication of the relative importance of radionuclide members of the decay chain headed by thorium-230 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	**^{230}Th**	**All**	**100%**
Ingestion	**^{230}Th**	**All**	**80%**
	^{210}Pb	After a few tens of years	5%
	^{210}Po	After a few tens of years	10%
External irradiation	^{214}Pb	After a year	10%
	^{214}Bi	**After a few days**	**90%**

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.1.4 Assuming radium-226 was present at Manchester

Within 100 years the progeny of radium-226, down to lead-210, could potentially grow in to be in secular equilibrium. As discussed in Section 2.4.1.3 the effect of radon-222 emanation is ignored. Therefore all members of this decay chain down to lead-210 could be assumed to be in secular equilibrium and are present at the same activity as radium-226. Lead-210, and its progeny of bismuth-210 and polonium-210, are likely to be present at a lower activity due to the relatively long half-life of lead-210 and in this case, after 100 years, are potentially present with an activity of about 97% of that of the radium-226.

As the parent radionuclide, radium-226, has a half-life of approximately 1600 years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.4.

Table 2.4: Indication of the relative importance of radionuclide members of the decay chain headed by radium-226 to the integrated dose over a period of 100 years for each exposure pathway [(a)]

Pathway	Important chain members over the first 100 years[(b)]	Significant exposure times[(c)]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[(d)]
Inhalation[(e)]	**^{226}Ra**	**All**	**50%**
	^{210}Pb	After a few tens of years	10%
	^{210}Po	After a few tens of years	40%
Ingestion	^{226}Ra	All	15%
	^{210}Pb	After a few tens of years	30%
	^{210}Po	**After a few tens of years**	**55%**
External irradiation	^{214}Pb	After a few days	10%
	^{214}Bi	**After a few days**	**90%**

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.1.5 Assuming lead-210 was present at Manchester

Lead-210 has a half-life of about 22.3 years and would therefore undergo significant decay over a period of 100 years. The potential activity present today would be about 22 times less than that present at the time of Rutherford. Both the radioactive progeny of lead-210 are likely to be in secular equilibrium with the lead-210. This information is summarised in Table 2.5.

Table 2.5: Indication of the relative importance of radionuclide members of the decay chain headed by lead-210 to the integrated dose over a period of 100 years for each exposure pathway [(a)]

Pathway	Important chain members over the first 100 years[(b)]	Significant exposure times[(c)]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[(d)]
Inhalation	^{210}Pb	All	25%
	^{210}Po	**After a few months**	**75%**
Ingestion	^{210}Pb	All	35%
	^{210}Po	**After a few months**	**65%**
External irradiation	**^{210}Pb**	**All**	**100%**

(a) During the period of 50 to 100 years after the original isolation of the source material the activity of all members of this decay chain are between 5 and 20 times less than the original material due to radioactive decay.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

2.4.2 Significant radionuclides within the thorium-232 decay chain

2.4.2.1 Assuming thorium-232 was present at Manchester

After a period of time of approximately 50 years members of this decay chain would be in secular equilibrium. The effect on the activity of lower decay chain members of radon-220 emanation is hard to assess and the cautious assumption to make is that there is no significant effect. Therefore the progeny of radon-220, polonium-216, and subsequent decay chain members to the stable form lead-208, could be assumed to be in secular equilibrium and are present at the same activity as thorium-232.

As the parent radionuclide, thorium-232, has a long half-life of approximately 1400 million years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.6.

Table 2.6: Indication of the relative importance of radionuclide members of the decay chain headed by thorium-232 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	^{232}Th	All	35%
	^{228}Ra	After a few years	5 %
	^{228}Th	**After a few years**	**55%**
	^{224}Ra	After a few tens of years	5 %
Ingestion	^{232}Th	All	20%
	^{228}Ra	**After a few years**	**65 %**
	^{228}Th	After a few years	5 %
	^{224}Ra	After a few tens of years	5 %
External irradiation	**^{228}Ac**	**After a few days**	**75%**
	^{212}Pb	After a few tens of years	10%
	^{212}Bi	After a few tens of years	15%

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.2.2 Assuming radium-228 was present at Manchester

Within this decay chain there are no radionuclides with a long half-life; the longest half-life being around 5 years for radium-228. This means that within 50 years radium-228 will have decayed so that its activity is lower by a factor of over 400. Over a period of 100 years decrease in activity has reached a value of around 200,000. Therefore there should be little trace of activity from any member of this decay chain present today, although exposure during the 1960s and 1970s may not have been trivial if the original source was very active. This information is summarised in Table 2.7.

Table 2.7: Indication of the relative importance of radionuclide members of the decay chain headed by radium-228 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	^{228}Ra	All	5 %
	^{228}Th	**After a few days**	**90%**
	^{224}Ra	After a few days	5 %
Ingestion	**^{228}Ra**	**All**	**75 %**
	^{228}Th	After a few days	10%
	^{224}Ra	After a few days	10%
External irradiation	**^{228}Ac**	**After a few days**	**70%**
	^{212}Pb	After a few days	10%
	^{212}Bi	After a few days	20%

(a) During the period of 50 to 100 years after the original isolation of the source material the activity of all members of this decay chain are approximately 400 less than the original material due to radioactive decay.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.3 Significant radionuclides within the uranium-235 decay chain

2.4.3.1 Assuming uranium-235 was present at Manchester

After a period of time of approximately 50 years the uranium-235 and its immediate progeny thorium-231 would be in secular equilibrium. The next radionuclide in the decay chain, protactinium-231, would be present with an activity approximately 1000 times less than the uranium-235 and thorium-231. However, the protactinium-231 and some of its progeny are significantly radiotoxic and therefore still contribute significantly to the total integrated decay chain dose despite being present with a relatively low activity.

As the parent radionuclide, uranium-235, has a long half-life of approximately 700 million years, there would not be any significant decrease in the activity present after 100 years when compared to the source material used during Rutherford's time. This information is summarised in Table 2.8.

Table 2.8: Indication of the relative importance of radionuclide members of the decay chain headed by uranium-235 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	**^{235}U**	**All**	**80%**
	^{231}Pa	After a few tens of years	5 %
	^{227}Ac	After a few tens of years	15%
Ingestion	**^{235}U**	**All**	**95%**
	^{231}Pa	After a few tens of years	2 %
	^{227}Ac	After a few tens of years	2%
External irradiation	**^{235}U**	**All**	**95%**
	^{231}Th	After a few tens of days	5%

(a) No significant loss of activity of the decay chain header will have occurred during the first 100 years so the total activity present is likely to have remained the same over this timescale.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.3.2 Assuming protactinium-231 was present at Manchester

If protactinium-231 was present then little decay of the original material would have occurred after 50 or 100 years. However, at present this radionuclide does not appear to have been present at Manchester and it is therefore not considered further.

2.4.3.3 Assuming actinium-227 was present at Manchester

After a period of time of approximately 50 years the actinium-227 and all members of its decay chain would be in secular equilibrium. Over a period of 50 years the activity of members of this decay chain would have decreased by a factor of about 5, and after 100 years this decrease would be around a factor of 25. This information is summarised in Table 2.9.

Table 2.9: Indication of the relative importance of radionuclide members of the decay chain headed by actinium-227 to the integrated dose over a period of 100 years for each exposure pathway [a]

Pathway	Important chain members over the first 100 years[b]	Significant exposure times[c]	Potential contribution to the integrated dose for each exposure pathway from the members of the decay chain after 50 to 100 years[d]
Inhalation[e]	**^{227}Ac**	**All**	**100%**
Ingestion	**^{227}Ac**	**All**	**90%**
	^{223}Ra	After a few years	10%
External irradiation	^{227}Th	All	25%
	^{223}Ra	**After a few days**	**30%**
	^{219}Rn	After a few days	15%
	^{211}Pb	After a few days	15%
	^{211}Bi	After a few days	15%

(a) During the period of 50 to 100 years after the original isolation of the source material the activity of all members of this decay chain are between 5 and 25 times less than the original material due to radioactive decay.

(b) Radionuclides given in bold represent those which contribute most to the integrated chain dose over the period 50 to 100 years.

(c) Represents the times that the radionuclides contribute more than a few percent to the total integrated chain dose for that pathway.

(d) These potential contributions to the total integrated dose are approximate and only intended for use as an illustration as many factors could alter the contribution any radionuclide makes, such as the relative activity of decay chain members in the original material and processes that change the natural process of radiological decay such as renovation of a room removing some radionuclides but not others.

(e) Radon is a member of this decay chain and is generally important with regard to the dose following the inhalation of radioactivity. However, the dose from radon is difficult to assess without site-specific parameters relating to the room size, ventilation, emanation rates etc so it is not included in this table as its relative importance is difficult to judge.

2.4.4 Other radionuclides within the uranium and thorium decay chains

There is evidence that several other radionuclide members of these decay chains, not identified in the preceding sections, were present during work at the University of Manchester in the early 20th century (Todd, 2008). In addition, some members of these decay chains would be chemically identical to those radionuclides that were known to have been present. For example, isolation of thorium-230 would also isolate thorium-231 if it was also present in the material used as a source of radioactivity. Therefore even though thorium-231 does not appear in any of the literature associated with the work at Manchester it must be assumed that this radionuclide could also be present. However, except for the radionuclides specifically mentioned in the above sections, the separation of all other members of these decay chains would result in no significant radioactivity being present after a few tens of years. This is because the radioactivity would have decayed away ie after a few tens of years the activity of these radionuclides would have decreased by a factor of at least 1 million. Therefore the dose from these radionuclides, which were separated out during Rutherford's time at Manchester, over the period of 50 to 100 years (representing approximately the time from the 1960s to the present day) would be negligible.

2.5 Possible importance of different exposure pathways for the uranium and thorium decay chains

As a detailed assessment of the doses has not yet been carried out the comments in this section are general and not specific to the situation at the University of Manchester.

Where radium or its parent radionuclides are present the decay products of radon typically make a significant contribution to the estimates of dose. The exposure pathway for radon gas is inhalation.

Within these decay chains there are several radionuclides that emit high-energy alpha particles. This means that, in terms of potential doses, exposure from radionuclides within the body will tend to be more important than external irradiation by radionuclides located outside of the body. For radionuclides taken into the body, those taken in via inhalation will generally result in a more significant dose than those taken in via ingestion. However, there are many factors that could affect this, such as the distribution of the activity around where someone spends a significant amount of time, the availability of any radioactivity present to become airborne or to be picked up on hands or on items that could then be placed in the mouth.

2.6 References

ICRP (1996). Age dependent doses to members of the public for intakes of radionuclides: Part 5 Compilation of ingestion and inhalation dose coefficients. ICRP Publication 72. *Ann ICRP*, **26** (1).

Todd N (2008). Historical and radio-archaeological perspectives on the use of radioactive substances by Ernest Rutherford. Interim version 1, December 2008. Personal communication.

3 PAPER 3: BEHAVIOUR OF RADIONUCLIDES AFTER INHALATION OR INGESTION

G ETHERINGTON AND J MARSH

3.1 Introduction

This section provides a brief description of the behaviour in the human body of selected radionuclides after inhalation or ingestion. The radionuclides are those identified to be of potential concern at specified locations on the campus of the University, ie, uranium and thorium isotopes and their radioactive progeny, radium-226, radon and its radioactive progeny, lead-210 and polonium-210. Radiation doses to the brain and pancreas that could result from intakes of these radionuclides are addressed specifically.

Radionuclides may be taken into the body via four main routes; inhalation, ingestion, wounds, or by absorption through intact skin. The latter pathway is not significant for the radionuclides of concern, and intake via wounds usually occurs as a result of accidents when industrial processes (eg, machining) are being carried out. Therefore, only inhalation and ingestion are considered here.

Biokinetics is the term used to describe the movement of radionuclides in the human body. There are three main aspects: biokinetics of radionuclides in the respiratory tract (RT) after inhalation; biokinetics of radionuclides in the gastro-intestinal (GI) tract after ingestion; and biokinetics of radionuclides in the other organs of the body after uptake from the RT and/or GI tract. The latter is sometimes called *systemic biokinetics*.

Most of the radionuclides of concern decay to produce a chain of radioactive progeny (see paper 1). This increases the complexity of any description of biokinetic behaviour. In some cases, radioactive progeny can be assumed to take on the biokinetic behaviour of the parent radionuclide (“shared biokinetics”); in others, the progeny adopt their own biokinetic behaviour (“independent biokinetics”).

3.2 Biokinetics in the respiratory tract (ICRP, 1994a)

Deposition of inhaled particles depends on physical factors such as breathing rate and particle size. Aerosols with particle sizes greater than about 0.5 μm deposit more in the extrathoracic airways (nasal airways, larynyx, etc) than the lungs; below 0.5 μm, deposition in the lungs dominates until particle sizes approach the nanometre size range. The ambient atmospheric aerosol is characterised by a distribution with a mass median close to 1 μm; close to the source of an aerosol produced by industrial processes, mass median values tend to be higher, and are often assumed to be 5 μm.

Particles are cleared from the lungs by two processes, particle transport to the GI tract and the lymphatic system (the former by mucociliary clearance), and absorption into blood. Particle transport is assumed to be the same for all materials, whereas absorption is dependent on chemical form. Compounds are assigned to one of three

default *Absorption Types*, Types F (fast), M (moderate) or S (slow). For instance, uranium hexafluoride (UF_6) is assigned to Type F, whereas uranium dioxide (UO_2) is assigned to Type S. Decay products of radionuclides deposited in the lung are generally assumed to adopt the absorption type of the parent radionuclide.

Sites of deposition in, and corresponding rates of clearance from, the RT are important because they are among the main factors determining radiation dose to the RT. The longer a radionuclide remains in the RT, the higher the radiation dose will be. The same factors determine uptake of radionuclides from the RT, and hence radiation doses to other organs.

3.3 Biokinetics in the gastro-intestinal (GI) tract (ICRP, 1979, 2007)

Ingested radionuclides pass through the GI tract and are excreted in faeces. As in the RT, absorption into blood can occur, and is again dependent on chemical form. Compounds are assigned a GI uptake factor, f_1 (ICRP, 1979) or absorption factor, f_A (ICRP, 2001) between 0 and 1 (ICRP, 1994b). For a particular radionuclide, lower values result in higher radiation doses to the GI tract (because more of the radionuclide remains in the GI tract), and lower doses to other organs.

3.4 Systemic biokinetics

Distribution, retention and excretion following uptake from the RT or GI tract is dependent on the element, rather than the radionuclide or the chemical form of the radionuclide at the time of the intake. The International Commission on Radiological Protection * (ICRP, www.icrp.org) has developed and published biokinetic models for each of the elements of interest, and these are described below. In some cases, features of the models depend on age at time of intake; the descriptions below refer to adults. These models often group organs other than bone together and refer to them as “soft tissues”. The brain and pancreas are generally included in this grouping.

The models for the radionuclides of concern are rather complex. The main features of these models are described here but reference should be made to the ICRP source documents for a full account.

* Members of ICRP are acknowledged international experts within their areas of specialisation in radiation protection. An ICRP Task Group either develops a biokinetic model itself, or validates and adopts models published in the open scientific literature. All ICRP models are subject to normal scientific peer review, and are generally acknowledged to reflect the “state of the art”. The recommendations of ICRP form the scientific basis of radiation protection legislation in all countries of the European Union.

3.4.1 Uranium (ICRP, 1995, 1997)

Uranium entering the blood after uptake from the RT or GI tract is retained by bone and soft tissues (including the liver), or excreted in urine and faeces. Uranium is unusual among the heavy metals in that approximately 75% of uranium entering the blood is excreted *via* urine rapidly (ie within a few days). A substantial proportion of the uranium excreted in urine is retained temporarily in kidneys, with the result that damage to the kidney can result if uranium concentrations in that organ are high enough. This is the origin of the chemical toxicity of uranium.

Uranium deposited in soft tissues is generally retained with biological half-times of a few days, although a small fraction of the activity deposited in liver is retained with a half time of 10 years. A biological half-time is the time required by a body or organ to process and eliminate half the amount of a substance introduced into it. Uptake and retention of uranium in the skeleton is qualitatively similar to the behaviour of calcium. About 10% of uranium entering the blood deposits in bone, initially on bone surfaces. From here, it either re-circulates into blood or moves into the bone volume. Some of the uranium in the bone volume is retained there for very long times (with a half-time of around ten years) and is removed only as a result of bone resorption. The location of deposition within the bone is important because cells on inner bone surfaces are the target tissues for induction of bone cancer. Furthermore, red bone marrow (the target tissue for leukaemia induction) is irradiated to a greater extent by radionuclides deposited on bone surfaces as compared with radionuclides in bone volume.

The main path of the decay chain for uranium-238 includes radionuclides of the elements thorium, protactinium, uranium, radium, radon, polonium, lead, and bismuth (see paper 1). The biokinetics of these decay products is described below.

3.4.2 Thorium (ICRP, 1995, 1997)

Like uranium, thorium is retained by bone and soft tissues (including the liver), or excreted in urine and faeces. Compared with uranium, a lesser amount (~10%) is excreted rapidly in urine. A significant fraction of the thorium deposited in soft tissues is retained for very long times (half-times of 2–100 y). About 50% of thorium entering the blood initially deposits on bone surfaces, and accumulates there further over following years. It then translocates slowly (over decades) to bone volume and bone marrow.

The main path of the decay chain for thorium-232 includes radionuclides of the elements radium, actinium, thorium, radon, polonium, lead, bismuth and thallium (see paper 1). The biokinetics of these decay products is described below.

3.4.3 Radium (ICRP, 1993, 1997)

The biokinetic behaviour of radium is qualitatively similar to that of uranium. In fact the models used for the two elements are structurally identical, differences arising from the different rates with which radium moves from one organ or tissue to another. One of the main differences in biokinetic behaviour is that radium does not exhibit the high initial excretion in urine that is found with uranium.

The main path of the decay chain for radium-226 includes radionuclides of the elements radon, polonium, lead, and bismuth (see paper 1). The biokinetics of these decay products is described below.

3.4.4 Progeny of uranium, thorium, and radium (ICRP, 1993, 1995)

Except for isotopes of radon, thorium and actinium, radioactive progeny produced in bone volume adopt shared biokinetics until removed from the bone volume. Outside the bone volume, decay products adopt independent biokinetics.

Biokinetic models for polonium and lead are described below. Other decay products are assumed to behave as follows:

Protactinium and *actinium* are similar to thorium (described above).

Radon produced in soft tissues or on bone surfaces translocates to blood on a timescale of minutes, whereas radon produced in bone volume translocates to blood on a timescale of days. Radon entering blood is assumed to be removed by exhalation on a timescale of minutes.

Bismuth produced in the body is assumed to translocate to blood on timescales of tens of days. It is then rapidly distributed between urine, the GI tract (and then to faeces), kidneys, liver and other tissues.

Thallium isotopes occurring in these decay chains have half-lives less than 4.2 minutes and so are assumed to decay at their point of origin.

3.4.5 Lead-210 (ICRP, 1993)

The biokinetic behaviour of lead is broadly similar to that of radium. Uptake to liver is higher at early times after intake, although this component of liver retention is associated with a relatively short retention half-time (tens of days).

Lead-210 decays to polonium-210. When in bone volume, polonium-210 adopts shared biokinetics. Outside bone volume, it adopts independent biokinetics.

3.4.6 Polonium-210 (ICRP, 1993)

Polonium is an unusual heavy metal in that it deposits predominantly in soft tissues after uptake to blood. In ICRP's biokinetic model for polonium, fractions of 0.1, 0.1, 0.1 and 0.7 go to liver, kidneys, spleen and all other tissues, and are retained with a half-time of 50 days.

3.4.7 Radon-222 and its radioactive progeny

An intake by inhalation of radon gas usually involves intake of its accompanying radioactive progeny. These are relatively short-lived isotopes of polonium, lead and bismuth, which finally decay to the 22 year half-life radionuclide lead-210 (see paper 1).

Radon and its progeny are generally in a partial state of equilibrium[+]; equilibrium is not usually complete because progeny radionuclides may be removed from the air as a result of deposition onto surfaces. Radon and each of its progeny radionuclides will decay within the body, where equilibrium conditions are likely to be different to those characterising the intake. A description of the biokinetics of radon and its progeny is thus rather complex.

After inhalation, radon gas may be exhaled, or it may dissolve in body fluids and distribute among body organs, where it can decay to form polonium, lead and bismuth. Radon is known to concentrate preferentially in fatty tissues such as bone marrow. The progeny of inhaled polonium, lead and bismuth are assumed to adopt shared biokinetics while in the respiratory tract. After uptake to blood, progeny of lead are assumed to adopt independent biokinetics, whereas progeny of polonium and bismuth are assumed to adopt shared biokinetics.

3.5 Doses to brain and pancreas

Committed equivalent doses to the brain and the pancreas resulting from intakes by inhalation of selected radionuclides have been determined. The equivalent dose to an organ is the sum of the absorbed doses from each type of radiation (alpha, beta, gamma, etc), each weighted by the appropriate radiation weighting factor. The absorbed dose is the energy deposited by ionising radiation per unit mass of the organ. These radiation weighting factors reflect the effectiveness of the particular radiation type in causing cancer. For instance, alpha radiation is know to be more effective in causing cancer than beta and gamma radiation, so alpha radiation has a radiation weighting factor of 20, whereas beta and gamma radiation have a radiation weighting factor of one. The equivalent dose to an organ can be used to make an estimate of the risk of a cancer occurring in that organ. The radionuclides for which doses have been determined are: uranium-238, uranium-234, thorium-232, thorium-228, radium-226, lead-210 and polonium-210.

Doses from intakes of radionuclides are protracted over time, so committed doses are used. For adults, this is the dose received over 50 years following the intake.

Equivalent doses are taken from the ICRP database of dose coefficients (ICRP, 2009). It is assumed that any intakes arise from exposure to ubiquitous low-level contamination that (with the exception of radon) has been present for many years. As a result, it is probable that it would be present in a form that is attached to the ambient atmospheric aerosol. The particle size distribution for such an aerosol is usually characterised using the ICRP default value for activity median aerodynamic diameter (AMAD) of 1 μm. This is the default for public exposure. For consistency, other ICRP default parameter values for public exposure have also been used. Dosimetric calculations could be made using many different combinations of model parameter values, but given the current lack of information on possible exposure conditions, this is considered to be unjustified.

[+] When equilibrium is complete, all of the radioactive progeny in the decay chain have the same activity (measured in Becquerel, Bq), as the parent radionuclide.

The ICRP database does not contain dosimetric data for intakes for radon. These results have been calculated by the authors. For simplicity, shared kinetics was assumed in these calculations for the progeny of lead, as well as for the progeny of polonium and bismuth.

These results, presented in Tables 3.1 and 3.2, show that equivalent doses to the brain and pancreas are significantly less than the highest organ doses (to lungs or bone surfaces) for the radionuclides considered.

Table 3.1 Committed equivalent doses for intake by inhalation for selected radionuclides

Radionuclide (Absorption Type)	Most exposed organ (T_M)	Equivalent dose (Sv per Bq intake)		
		T_M	Brain	Pancreas
^{238}U (M)	Lungs	$2.2\ 10^{-5}$	$1.2\ 10^{-7}$	$1.2\ 10^{-7}$
^{234}U (M)	Lungs	$2.7\ 10^{-5}$	$1.4\ 10^{-7}$	$1.4\ 10^{-7}$
^{232}Th (S)	Bone surfaces	$2.9\ 10^{-4}$	$8.2\ 10^{-7}$	$8.3\ 10^{-7}$
^{228}Th (S)	Lungs	$3.3\ 10^{-4}$	$6.7\ 10^{-8}$	$7.2\ 10^{-8}$
^{226}Ra (M)	Lungs	$2.8\ 10^{-5}$	$2.4\ 10^{-8}$	$2.4\ 10^{-8}$
^{210}Pb (M)	Bone surfaces	$1.3\ 10^{-5}$	$6.5\ 10^{-8}$	$6.5\ 10^{-8}$
^{210}Po (M)	Lungs	$2.6\ 10^{-5}$	$4.9\ 10^{-8}$	$4.9\ 10^{-8}$

Table 3.2 Committed equivalent doses for intake by inhalation of radon-222 and progeny

Organ	Committed equivalent dose[(a)]		
	from intake of radon	from intake of associated radon progeny	Total
Lungs[(b)]	$9.6\ 10^{-3}$	$9.9\ 10^{-1}$	1
Brain	$4.7\ 10^{-4}$	$7.1\ 10^{-5}$	$5.4\ 10^{-4}$
Pancreas[(c)]	n/a	$8.3\ 10^{-5}$	n/a

(a) Doses normalised to the total committed equivalent dose to the lungs

(b) The most exposed organ is the lungs

(c) n/a – not available. The current radon intake model does not allow calculation of this quantity

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4 PAPER 4: BACKGROUND INFORMATION ON RISKS OF PANCREATIC AND BRAIN CANCER FROM IONISING RADIATION

RGE HAYLOCK AND CR MUIRHEAD

4.1 Introduction

The effects of ionising radiation exposure on the long term risks of cancer have been studied for many years. Large acute exposures have been shown to raise the risks of some, but not all, solid cancers and leukaemias. However, evidence of the magnitude of these risks at low doses and for chronic exposures and of how factors such as age at exposure and time since exposure might modify risk is far less clear.

Current risk models for cancer induction at low doses and low dose rates are based on information from the atomic bomb survivors in Hiroshima and Nagasaki (specifically from the Life Span Study) and people exposed to ionising radiation for medical reasons, both diagnostic and therapeutic. However, information from occupationally-exposed populations, eg, nuclear workers and underground uranium miners, is also beginning to be used as studies of these groups become more informative.

The latest data from all these sources has been comprehensively reviewed by the United Nations Scientific Committee on the Effects of Atomic Radiation in the UNSCEAR 2006 report, published in 2008 (UNSCEAR 2008). This report found that data for the Japanese atomic bomb survivors are consistent with a linear relationship between cumulative dose and risk for all solid cancers taken as a group; however, it concluded that for a number of individual solid cancer types the evidence for a linear relationship is far less certain.

The current evidence regarding the association of pancreatic cancer and brain cancer with radiation exposure is presented below together with information on the risk of cancer following exposure to certain radionuclides. The evidence concerning the possibility of interactions between radiation and other agents such as mercury in inducing cancer and information on radiation and diseases other than cancer are also discussed.

4.2 Pancreatic cancer

4.2.1 Background

Pancreatic cancer is the sixth most common incident cancer in the UK. The annual incidence rate among males has fallen slightly over the last three decades to an age adjusted value of around 10 cases per 100,000 in 2005. For females the rate has remained steady over this period at a slightly lower value of 8 cases per 100,000 (Cancer Research UK: http://www.cancerresearchuk.org/). Incidence rates are very low at young ages in both sexes and only begin to increase after age 45 with 75% occurring

in those aged over 65. The prognosis for pancreatic cancer is poor with survival rates of five years among the lowest of any cancers at around 2-3%.

The most consistent risk factor for pancreatic cancer is smoking. The International Agency for Research on Cancer has concluded (IARC 2004) that this cancer is causally associated with smoking and that the risk increases both with the duration of smoking and the number of cigarettes smoked daily.

4.2.2 Ionising radiation

A cohort of 64,209 underground miners was examined in a collaborative analysis of 11 separate studies (Darby et al, 1995).The total cohort was followed up for an average of 16.9 years and individuals had an average cumulative exposure of 155 Working Level Months (WLM)*. A statistically significant trend in risk with cumulative radon dose was observed (based on a total of 85 pancreatic cancer deaths). There was also an overall excess of pancreatic cancer but it was small and not statistically significant. However, the authors discounted the statistically significant trend result, suggesting that it may have been either a chance finding (since they looked for associations between radon and many cancer types) or the result of disease misclassification (where a pancreatic cancer is listed as the primary disease but is in fact a secondary cancer).

A more recent study of underground uranium miners in Germany (Kreuzer et al, 2008) based on a similar size cohort to that of Darby et al (1995) but with both a longer average follow-up (of 35 years) and a higher average individual cumulative radon exposure (of 279 WLM) did not find any excess of pancreatic cancer nor an increasing trend with cumulative exposure (based on a total of 223 pancreatic cancer deaths).

The Life Span Study (Preston et al, 2003; Preston et al, 2007) found no statistically significant excess of pancreatic cancer mortality or incidence among the Japanese A-bomb survivors. A range of studies of people who were medically exposed also did not provide reliable evidence for an association with radiation exposure (UNSCEAR 2008). Two recent studies of patients exposed to Thorotrast, an X-ray contrast medium, provide some indication of raised pancreatic cancer risk (Travis et al, 2003; Becker et al, 2008), although these analyses are limited by the generally small numbers of exposed cases and the potential for bias when considering specific cancer types; further details are given in section 4.4.6.

Pancreatic cancer has been studied in several studies of radiation workers. A study of mortality among 8,116 workers at a nuclear materials production plant (Loomis and Wolf, 1996) found an excess of pancreatic cancer over the general population. However, this excess was not statistically significant and no adjustment was made for the known

* A working level month is a unit of exposure used to express the accumulated human exposure to radon decay products. A working level month is equivalent to the exposure at an average concentration of 1 working level (WL) for 170 working hours. The concentration of radon progeny is measured in units of working level (WL) which is a measure of the potential alpha particles energy per litre of air. One WL of radon daughters corresponds to approximately 8000 Bq m^{-3} of radon in a typical indoor environment.

effects of smoking on pancreatic cancer risk which may have resulted in a biased value. Furthermore, the authors did not investigate whether the risk of pancreatic cancer was related to dose.

In contrast, a recent dose-response analysis (Muirhead et al, 2009) of cancer risk based on about 175,000 radiation workers on the UK National Registry for Radiation Workers (NRRW) did not identify any statistically significant association between pancreatic cancer mortality or incidence and cumulative radiation dose. In addition, a large study of 407,391 nuclear workers in 15 countries (Cardis et al, 2007) also did not find a statistically significant association between pancreatic cancer mortality and cumulative dose.

4.2.3 Summary

Current evidence (except the latest NRRW analysis and the Thorotrast analysis of Becker et al) was reviewed in the UNSCEAR 2006 report (UNSCEAR 2008) which concluded that '*there is little, if any, evidence for associations between pancreatic cancer and radiation dose*'.

4.3 Brain cancer

4.3.1 Background

Brain and central nervous system (CNS) tumours account for less than 2% of all newly-diagnosed primary tumours each year. The age adjusted annual incidence rate per 100,000 persons was 9 cases for males and 6 cases for females in 2005 (Cancer Research UK: http://www.cancerresearchuk.org/). This overall rate has increased slightly in both sexes in the last three decades but it is not clear if this is a real increase or a result of improvements in diagnostic techniques. Brain tumours can occur at any ages but incidence rates are generally low up to the end of teenage years then build gradually to a peak between 70 and 80 years. An additional complication is that different tumour subtypes tend to predominate at different ages.

The prognosis for brain cancer is quite poor with 5-year overall survival rates only increasing slightly over the last three decades in England and Wales to around 10% for males and about 12% for females. There is considerable variability in survival with age at diagnosis (around 40% during early adulthood to only a few percent late in life) and also between types of tumours; in particular, between malignant and benign tumours.

Investigation of the causes of brain cancer is complicated by the fact that there are many different types, differentiated mainly by the areas of the brain affected and by whether they are classed as benign or malignant. The heritability of a number of types of brain tumours has been investigated in a number of studies and a few hereditary syndromes have been characterised both clinically and now genetically. However, these syndromes are rare and thought to account for less than 5% of brain tumours (Preston-Martin, 1996).

Many common environmental and lifestyle factors have been investigated (Preston-Martin et al, 2006) with research on childhood brain cancers focusing on factors related

to *in utero* exposures. No clear evidence has emerged to support any factors as causative. In particular, parental smoking has not been found to be related to childhood brain cancer, neither has smoking in later life been associated with adult onset brain cancer. The same is true for alcohol consumption.

4.3.2 Ionising radiation

Exposure to high doses of ionising radiation, in particular therapeutic irradiation of the head and neck during childhood, has been found to be associated with increased brain cancer risk (Sadetzki et al, 2005). There is also some evidence of raised risks among patients exposed to Thorotrast (Travis et al, 2003; Becker et al, 2008), as described in section 4.4.6. However, taken overall, current data on risks from exposure in adulthood and/or at low dose exposure are limited and equivocal (UNSCEAR, 2008).

Investigations of the Life Span Study of Japanese A-bomb survivors (Preston et al, 2002) have found a borderline statistically significantly raised risk in the incidence of glioma and meningioma and a more statistically significantly increased risk for schwannoma. A linear dose response relationship has been found to fit the data well, even if these data are restricted to low doses. No evidence was found to support the hypothesis that risk decreases with time since exposure, indicating risks may persist to the end of life.

A number of large occupational studies of radiation workers (exposed to low doses) that have looked at brain cancer risks did not find any raised risk (eg, Cardis et al, 2007). In particular the recent analysis of the UK National Registry for Radiation Workers did not find any increasing trend with radiation dose in either incidence of mortality (Muirhead et al, 2009). Two studies of underground uranium miners (Darby et al, 1995; Kreuzer et al, 2008) also failed to find a statistically significant increase in brain cancer risk.

A specific risk model for all malignant brain cancers, as a group, based on the latest LSS incidence data has been developed by UNSCEAR (UNSCEAR 2008). Under this model the relative increase in risk is predicted to vary linearly as a function of dose and to increase with decreasing age at exposure. This model predicts that if a group of people representative of the UK population were exposed to a single acute dose of 0.1 Sv, then this exposure would increase the incidence of brain cancer by 0.035% over and above the number expected to occur in a similar but unexposed group from the UK population (UNSCEAR 2008).

4.3.3 Summary

There is evidence that high doses of radiation can cause brain cancer. A risk model for predicting that risk has been developed. The form of this model supposes that the risk relative to baseline increases linearly with radiation dose and also increases with decreasing age at exposure.

4.4 Radiation-induced health effects in relation to specific radionuclides

Risks from internal exposure to the alpha particle emitting nuclides of uranium and thorium isotopes and the decay chain of radium (ie, radium-226, radon and its daughters, polonium-210, lead-210) are of particular interest for this risk assessment. While alpha particles pose almost no risk outside the body (due to their very limited range in air) they can be highly damaging if inhaled or ingested. Evidence for the effects of specific radionuclides on cancer risks is highly nuclide-dependent. A good summary of the evidence is provided by the International Agency for Research on Cancer (IARC, 2001).

4.4.1 Radium

Occupational exposure to radium in the first half of the 20th century arose when it was used as a luminising agent for watch and instrument dials. Many dial painters received high doses and cohort studies of these painters have found a statistically significant dose-response relationship between estimated internal doses from radium and the incidence of bone cancer (Rowland et al, 1978). Patients who were treated with radium injections for ankylosing spondylitis received lower doses than the dial painters. Studies of these patients have shown a possible raised risk of leukaemia but due to small numbers of cancers it is difficult to draw reliable conclusions (Wick et al, 1999).

4.4.2 Radon and progeny

There is very good evidence from many studies of underground hard-rock miners and from residential radon studies linking indoor exposure to radon and its progeny to an increased risk of lung cancer (Darby et al, 2005; Grosche et al, 2006*;* Krewski et al, 2006). In contrast, there is generally little evidence of associations with other types of cancer (Darby et al, 1995, Kreuzer et al, 2008).

4.4.3 Polonium-210

There is no direct epidemiological evidence linking exposures from polonium to an increased incidence of specific cancer types.

4.4.4 Lead-210

There is no direct epidemiological evidence linking exposures from lead-210 to an increased incidence of specific cancer types.

4.4.5 Uranium isotopes

Studies of occupational exposure to uranium dust have not found consistent effects on specific cancer rates. A study of workers at a nuclear materials fabrication plant found an overall excess of lung cancer incidence but it was not possible to identify any positive dose-response relationship (Richardson and Wing, 2006).

4.4.6 Thorium isotopes

Thorium was used in an X-ray contrast medium, known as Thorotrast, between about 1930 and 1950. Studies have found that it is retained in the body for many years. The long-term effects of Thorotrast have been studied in a number of cohorts.

A study of two cohorts of patients exposed to Thorotrast (Travis et al, 2003), the first of patients in Denmark and Sweden and the second of patients in the USA, found a large and statistically significant excess of liver cancer and a smaller, but still statistically significant, excess of leukaemia excluding chronic lymphatic leukaemia.

This study also found a statistically significant raised relative risk of 3.8 for pancreatic cancer incidence among the Danish and Swedish patients compared to a matched control group (ie the risk of pancreatic cancer among Thorotrast-exposed patients was 3.8 times that among unexposed patients). However, this finding was imprecise – being based on a total of 11 cases in the Thorotrast group – and the 95% confidence interval (representing the level of statistical precision) for the relative risk ranged from 1.3 to 12.3. There was some evidence that the risk of pancreatic cancer in this cohort increased with increasing time since injection of Thorotrast and with a surrogate measure of cumulative radiation dose, but again this was based on small numbers of cases. Statistically significant raised risks were also seen for a range of other cancer types, including brain and other central nervous system tumours (relative risk 2.5) and statistically significant trends in incidence with the cumulative dose surrogate were found for gallbladder, peritoneum and other digestive cancers. However, Travis et al (2003) highlighted caution in interpreting the findings for specific types of cancer because of the potential for bias associated with the selection of cohort participants, possible lack of comparability between the exposed and control groups and confounding by the reason(s) for which Thorotrast was administered.

Travis et al (2003) found no excess of pancreatic cancer mortality among US Thorotrast patients; here the 95% confidence interval (CI) for the relative risk (0.9) ranged from 0.1 to 4.4. The corresponding relative risk for brain and other central nervous system tumours was 1.3, with a 95% CI ranging from 0.6 to 3.7, ie, there was no statistically significant excess. A study of Thorotrast patients in Portugal reported a relative risk for brain cancer mortality of 2.9 (95% CI 0.9 to 11.0) but did not report results specifically for pancreatic cancer (dos Santos Silva et al, 2003).

A study of German Thorotrast exposed patients (Becker et al, 2008) identified a large excess of liver cancer mortality compared to an unexposed control group, and that relative risk increased with increasing time since first exposure. It also found statistically significant excesses in mortality from cancers of the liver, gallbladder, pancreas, testis, prostate, brain, unspecified sites and haematopoietic malignancies (among males); and liver, brain and unspecified sites (among females). For pancreatic cancer, the relative risk was 5.5 (95% CI 1.7 to 23) in males and 1.3 (95% CI 0.2 to 9.6) in females, based on 15 and 3 deaths respectively among exposed patients. For brain tumours, the relative risk was 3.3 (95% CI 1.3 to 9.2) in males and 17.0 (95% CI 2.7-711) in females, based on 19 and 17 deaths respectively among exposed patients. This study did not examine whether there was a relationship between cancer mortality and surrogate measures of radiation dose.

In summary, Thorotrast patients in several countries have a large and statistically significantly raised risk of liver cancer and there is evidence of a raised risk of leukaemia excluding chronic lymphatic leukaemia. There is also some evidence of raised risks for other cancers, including pancreatic cancer and brain tumours. However, the strength of this evidence is limited by the generally small numbers of exposed cases and the potential for bias.

4.4.7 Summary

Current evidence suggests that exposure to the nuclides listed here has been associated with raised risks of bone cancer from radium exposure, lung cancer from radon exposure and liver cancer from thorium exposure. The evidence for associations with other cancers is weaker.

4.5 Interactions between radiation and other Factors

This topic has been considered by UNSCEAR (UNSCEAR 2000). This committee concluded that with the exception of radon and smoking (for which there is an increased combined effect on the risk of lung cancer), there was no evidence that low-level exposures to radiation and other cancer-causing agents yielded combined risks that differed greatly from the sum of the individual risks.

Specific evidence from epidemiological studies as to whether exposure to mercury can modify the effects of exposure to ionising radiation is very sparse. A study of 8318 male radiation workers at the Oak Ridge National Laboratory in the USA (Wing et al, 1993) adjusted for exposure to a number of chemical exposures - including mercury - when analysing cancer mortality in relation to low-dose external penetrating ionising radiation. Taking account of occupational exposure to mercury did not significantly affect the cancer risk estimates. However, the authors acknowledged that the power of the study to detect any changes was low.

Thus, there is no epidemiological evidence to suggest that mercury exposure can alter the risk of cancer from ionising radiation exposure.

4.6 Radiation and diseases other than cancer

4.6.1 Non-cancer diseases

Studies of the Japanese A-bomb survivors and of patients treated with radiation (eg for breast cancer) have shown raised risks of heart disease, as well as of respiratory and digestive diseases in the case of the A-bomb survivors (Preston et al, 2003; UNSCEAR, 2008). However, it is not clear whether risks are raised at doses from low-LET radiation below 0.5 Gy, nor what mechanisms might underlie such a diverse set of diseases.

There is very little epidemiological evidence as to whether ionising radiation is associated with motor neurone disease (MND). A study in Japan that examined whether MND mortality in the 1960s was related to mechanical injuries (Kondo et al, 1981) also looked at whether participants were present in Hiroshima or Nagasaki at the

time of the atomic bombings, as well as at occupational radiation exposure. In both instances there was insufficient data to enable risk estimates to be calculated.

Variations in Japanese MND rates between 1950 and 1900 have also been examined in relation to patterns in radioactive fallout from atmospheric weapons testing (Neilson et al, 1995). Although an association was found, no account was taken of confounding factors (of which there are many) and issues regarding the validity of the study methodology mean that this association cannot be considered to be causal.

Another correlation study by the same authors (Neilson et al, 1996) found that MND rates between 1981 and 1989 in 55 counties of England and Wales were positively associated with county averaged indoor radon dose rates but negatively associated with Gamma–ray dose rates. However, considerable variation in radon exposures to individuals within each county (Kondo, 1997) means that these results cannot be used as evidence that the same association would be found an individual level.

In summary, no reliable epidemiological evidence exists to support the hypothesis that MND is caused or accelerated by exposure to ionising radiation.

4.7 Heritable disease

Hereditary effects of radiation exposure have been extensively reviewed by UNSCEAR (UNSCEAR, 2001). The Committee found that epidemiological studies of human populations have not so far demonstrated that exposure to ionising radiation leads to a measurable increase in the risk of disease in offspring. In particular, no effects have been identified in the children of the survivors of the Japanese atomic bombings (Izumi et al, 2003b; Neel et al, 1990; Izumi et al, 2003a). Furthermore, studies of the offspring of radiation workers and of medically-exposed groups have generally not demonstrated raised risks of cancer or adverse pregnancy outcome following parental preconception irradiation (COMARE, 2002, 2003). However, the epidemiological data cannot rule out the possibility of small raised risks of heritable diseases. Furthermore, experimental data and understanding of the induction of heritable diseases suggest that ionising radiation can increase the risk of these diseases.

UNSCEAR (UNSCEAR, 2001) estimated that for a population exposed to radiation in one generation only, the risks to the progeny of the first post radiation generation are estimated to be 3,000 to 4,700 cases per Gy per one million progeny; this constitutes 0.4 to 0.6 per cent of the baseline frequency of those disorders in the human population.

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5 GLOSSARY

absorbed dose

The quantity of energy imparted by **ionising radiation** to a unit mass of matter such as tissue. Absorbed dose has the unit joules per kilogram (J kg^{-1}) which has the special name **Gray** (Gy).

alpha activity

The alpha activity is the number of alpha particles emitted per unit time. An alpha particle is identical to the nucleus of a helium atom, consisting of two protons plus two neutrons. An alpha particle has low penetrating power but high **linear energy transfer** (LET). The unit of activity is the **Becquerel** (Bq).

atoms

The simplest unit into which an element can be broken down whilst retaining its unique identity and properties. They consist of a central nucleus with a net positive electrical charge, orbiting around which are small lightweight negatively charged particles called **electrons**.

Becquerel (Bq)

The international (SI) unit for the number of nuclear disintegrations occurring per unit time, in a quantity of radioactive material. 1 Bq = 1 radioactive disintegration per second. This is an extremely small unit and levels are often prefixed with mega (10^6 Bq – MBq), giga (10^9 Bq – GBq) and tera (10^{12} Bq – TBq) particularly in the context of discharges of activity into the environment. Conversely, under normal circumstances, activity concentrations in environmental materials are generally low and so prefixes such as milli (10^{-3} Bq – mBq) and micro (10^{-6} Bq - μBq) are used.

beta radiation/beta particle

Beta radiation is a form of radioactivity in which beta particles are emitted from an atom. It has greater penetrative power than an alpha particle, but has a low **linear energy transfer** (LET). A beta particle has a mass and charge identical to that of an electron. Beta particles can be either positively (called a positron) or negatively charged (called an electron).

decay

The process of spontaneous transformation of a **radionuclide**. The decrease in the activity of a radioactive substance.

decay product

A nuclide or **radionuclide** produced by **decay**.

dose

General term for quantity of **ionising radiation**. See **absorbed dose**, **effective dose**, **equivalent dose** and **collective effective dose**. Frequently used for effective dose.

dose coefficient

Dose coefficients are values recommended by the International Committee on Radiological Protection that allow the activity taken into the body, either by inhalation or ingestion, to be converted into an **effective dose**. These values have been calculated by modelling the movement of a radionuclide within the body and determining the resulting tissue doses.

effective dose

The effective dose is the sum of the weighted **equivalent doses** in all the tissues and organs of the body. It takes account of the susceptibility of organs and tissues to radiation damage. Unit **Sievert** (Sv).

electron

An elementary particle with a low mass, of 5 10^{-4} that of a **proton**, and unit negative electric charge. Positive charged electrons, called positrons, also exist (see **beta particle**).

equivalent dose

The quantity obtained by multiplying the **absorbed dose** by a factor to allow for the different effectiveness of the various **ionising radiations** in causing harm to tissue. Unit **sievert**, symbol Sv. Usually the factor for **gamma rays**, **X-rays** and **beta** particles is 1 but for **alpha** particles it is 20.

fission (products)

The spontaneous or induced disintegration of a heavy atomic nucleus into two or more lighter fragments (nuclei). The energy released in the process is referred to as nuclear energy.

gamma rays

High energy photons, without mass or charge, emitted from the nucleus of a **radionuclide** following radioactive **decay**, as an electromagnetic wave. They are very penetrating but have a low **linear energy transfer** (LET).

Gray (Gy)

The international (SI) unit of **absorbed dose**. 1 Gy is equivalent to 1 joule of energy absorbed per kilogram of matter such as body tissue. Can also be used with prefixes such as nano to make units such as nanogray (nGy - 10^{-9} Gy).

half-life

The time taken for the activity of a **radionuclide** to lose half its value by **decay**. During each subsequent half-life its activity is halved again so its activity decays exponentially.

ionising radiation

Radiation which is sufficiently energetic to remove electrons from atoms in its path. In human or animal exposures, ionising radiation can result in the formation of highly reactive particles in the body which can cause damage to individual components of living cells and tissues. The term includes radiation at least as energetic as **X-ray**; **gamma rays** and charged particles such as **alpha** and **beta particles** are also forms of ionising radiation.

isotopes

Nuclides containing the same number of **protons** (ie, same atomic number) but a different number of **neutrons**.

linear energy transfer (LET)

This property of radiation relates to how much energy is lost by the radiation when travelling a given distance. High LET radiation loses most of its energy quickly and in a short distance. For example an alpha particle may not travel far but it may cause much damage to a cell it is travelling through compared to a radiation that loses energy over a larger distance (for example, a **beta particle**).

microsievert – see **Sievert.**

millisievert – see **Sievert.**

neutron

The uncharged particle in an atomic nucleus; its mass is similar to the mass of a hydrogen atom.

photon

The basic unit of light or other electromagnetic energy.

proton

The positively charged particle in an atomic nucleus; its mass is similar to the mass of a hydrogen atom.

radionuclide

A type of atomic nucleus which is unstable and which may undergo spontaneous **decay** to another atom by emission of **ionising radiation** (usually **alpha**, **beta** or **gamma** radiation).

Sievert (Sv)

The international (SI) unit of effective and equivalent dose. Because the Sievert is a large unit, effective dose is commonly expressed in milliSieverts (10^{-3} Sv, or mSv) and microSieverts (10^{-6} Sv, or μSv).