The Christie NHS
NHS Foundation Trust

Christie Medical Physics and Engineering
Basic training day for Radiation Protection Supervisors
Pre-course reading

Radiation Dose and Risk in Perspective

1. Radiation dose and dose quantities

There are several ways in which radiation dose can be expressed and it is important to understand the differences between them.

1.1. Air kerma

Air kerma (Kinetic Energy Released per unit MAss) is the kinetic energy (measured in Joules, J) of all the charged ionising particles liberated by the x-ray beam in unit mass of air (measured in kilograms, kg). The units of air kerma are therefore Joules per kilogram (J/kg), which is given the special name of 'Gray' (Gy).

1 Gray (Gy) = 1 Joule per kilogram (J/kg).

Ionisation chamber instruments are usually calibrated in terms of air kerma. This is the quantity that we measure when we do our physics surveys on diagnostic x-ray systems.

1.2. Absorbed dose

This is defined as the amount of energy (J) that ionising radiation deposits in a standard mass of tissue (kg). The units of absorbed dose are also J/kg (Gy).

You may think this sounds exactly the same as air kerma but there is a subtle difference. When radiation interacts with tissue, as opposed to air, scattered radiation is absorbed closer to where it is produced. Absorbed dose must take this into account. In diagnostic radiology, air kerma is numerically equal to absorbed dose to air, but at energies above 200 keV the two quantities start to differ. At radiotherapy energies, it is necessary to convert from one to the other using an f-factor.

In diagnostic radiology and nuclear medicine, the doses received are of the order of some mGy (1 mGy = $^{1}/_{1000}$ Gy).
In radiotherapy doses to the target are generally tens of Gy.

Backscatter
At diagnostic energies, scattered radiation travels equally in all directions. If you placed a measuring device on the surface of a patient, you would measure the air kerma plus an amount of back-scattered radiation. This combined quantity is the entrance surface dose. To calculate entrance surface dose, the air kerma is multiplied by a correction for back-scattered radiation.

1.3. Equivalent dose

Different types of ionising radiation such as alphas, betas, gammas, neutrons and x-rays differ in the way they act with biological material so equal energy deposited (i.e. absorbed dose) does not mean equal biological damage. Alpha particles are more heavily charged and slower than x-rays and so lose their energy more densely along their path through tissue. Therefore, 1 Gy of alpha particles causes more biological damage than 1 Gy of x-rays. In order to account for this, we define a quantity known as equivalent dose. Equivalent dose is equal to the absorbed dose multiplied by a factor which reflects the relative effectiveness of the radiation in causing biological harm. For x-rays, gamma rays and beta particles this radiation weighting factor is 1. So the

absorbed dose and equivalent dose are numerically equal. However, equivalent dose has a different unit: the Sievert (Sv). Alpha particles have a weighting factor of 20 and neutrons have a weighting factor of 2.5 – 21. These are summarised in Table 2.

If you define dose in this way you can relate the dose from any form of ionising radiation to the risk of inducing cancer. For example, 1 Sv of alpha radiation to the lung would create the same risk of inducing fatal cancer as 1 Sv of beta radiation.

1.4. Effective dose

The risk to various parts of the body varies from organ to organ depending on how susceptible they are to the effects of radiation. This property is known as the 'radiosensitivity' of the organ. Other types of harm such as non-fatal cancer and hereditary damage caused by irradiation of testes and ovaries also need to be taken into account. In 1990, the ICRP (ICRP Report 60) published twelve organ weighting factors related to the risk associated with that tissue or organ. In 2007, the ICRP (ICRP Report 103) modified the weighting factors for some organs and also assigned a weighting factor to three organs which had previously been classed as 'remainder'. The majority of patient (but not staff) dose calculations now are based on ICRP103 weighting factors.

Table 1: Tissue weighting factors

Tissue or organ	Tissue weighting factor (ICRP 60, 1990)	Tissue weighting factor (ICRP 103, 2007)
Gonads	0.2	0.08
Red bone marrow, Colon, Lung, Stomach, Breast	0.12 Did not include breast	0.12
Bladder, Liver, Oesophagus, Thyroid	0.05 Included breast	0.04
Skin, Bone surface, Brain, Salivary glands	0.01 Did not include brain, salivary glands	0.01
Remainder	0.05	0.12
Whole Body Total	**1.00**	**1.00**

The equivalent dose in particular organs arising from an irradiation is multiplied by the relevant tissue weighting factor and the total is then summed to give the quantity 'effective dose'. Effective dose gives a broad indication of the detriment to health from any exposure to ionising radiation whether it was uniform body irradiation or whether only a section was irradiated. The unit is also the Sievert (Sv).

Effective dose can also be used to compare the risks of a nuclear medicine examination with the x-ray alternative, e.g. V/Q scan or CTPA. (For intakes of long-lived radioactive materials that remain in the body – not typically used in nuclear medicine, or regular intakes e.g. through food or water, the committed effective dose takes into account the extended period of exposure.)

1.5. Mean Glandular Dose (Mammography)

In mammography the dose of interest is the mean dose to the glandular tissues within the breast. The nature of the examination means that the equivalent dose to all organs in the body (apart from the breast) is an extremely small proportion of the total dose. Instead of quoting an effective dose for the examination, which relates to the risk of inducing cancer anywhere in the body, we quote a dose which relates to the risk of inducing cancer within the breast; this is the *Mean Glandular Dose*. The glandular tissues include the acinar and ductal epithelium and associated stroma, which together are believed to be the part of the breast most sensitive to radiation-induced carcinogenesis.

It is not possible to measure mean glandular tissue dose directly. Instead, the method adopted involves measuring the incident air kerma at the surface of the breast, and converting this figure into mean glandular dose using factors derived by mathematical methods. The incident air kerma (K), is measured using a suitable measuring instrument (ionisation chamber) on physics surveys. The conversion factors are given in IPEM Report 89 (IPEM, 2005), the physics report detailing how to perform QA in mammography. The formula is:

$$MGD = Kgcs$$

where the *g*-factor converts air kerma to mean glandular dose, the *c*-factor corrects for the composition (glandularity) of the breast, which varies with breast thickness, and the s factor corrects for the spectrum used i.e. Mo/Mo or Mo/Rh etc…

Questions

The following weighting factors convert absorbed dose (measured in Gy) to equivalent dose (measured in Sv). The more biologically damaging the radiation, the higher the weighting factor.

Table 2: Radiation weighting factors

Type of Radiation	Radiation weighting factor
X-rays	*1*
Gamma rays	*1*
Betas	*1*
Protons	*2*
Neutrons	*2.5 to ~21 (depending on energy)*
Alphas	*20*

1.1) What is the equivalent dose for: Don't forget the units!

a) 3 Gy of X-rays _____

b) 3 Gy of protons _____

c) 3 Gy of alpha particles _____

d) 3 Gy of gamma rays? _____

1.2) Which is the most biologically damaging type of radiation? _____

1.3) What is the equivalent dose for:

a) 2 Gy of alpha particles _____

b) 30 Gy of beta particles _____

c) Which of these gives a higher absorbed dose to the body? _____

d) Which of these would cause more biological damage to the body? _____

2. Radiobiology

In the targeted model of radiation damage, when ionising radiation passes through tissue, it ultimately causes changes to the DNA. This may occur directly or indirectly via free radicals, as shown in Figure 1.

Charged water molecules are produced when the radiation ejects one of the electrons in the molecule. These then break up into free radicals such as the free hydroxyl radical OH^{-} composed of an oxygen and a hydrogen atom. Free radicals are chemically highly reactive and can go on to alter DNA.

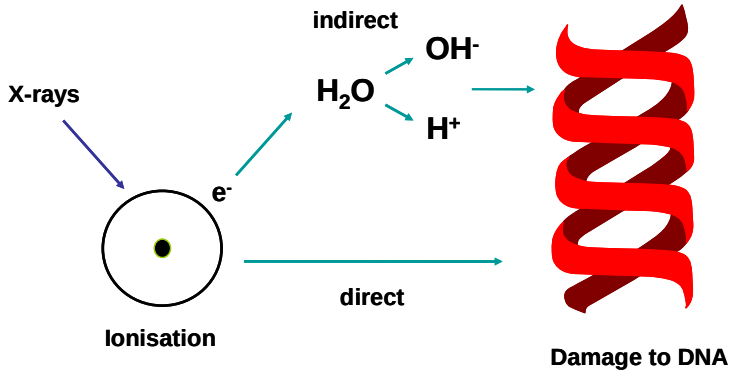

Figure 1: Ionising radiation causes damage to the DNA. This can be via an indirect or direct mechanism

Outcomes at the cell level are either:

- full repair
- cell death
- cell modification.

Cell death leads to tissue reactions (previously called) 'deterministic effects'. Cell modification leads to cancer and heritable effects (previously called 'stochastic effects'). These terms and others will now be explained in more detail.

2.1. Somatic effect

A somatic effect affects the exposed individuals.

2.2. Heritable effect

A heritable effect does not affect the exposed individuals but instead affects subsequent generations. The damage arises from the irradiation of reproductive organs or cells.

2.3. Tissue reactions

Tissue reactions are most often 'early' effects such as erythema (skin reddening), epilation (hair loss) and radiation sickness. They will only occur above a minimum radiation dose (a threshold dose) and result from the death of cells.

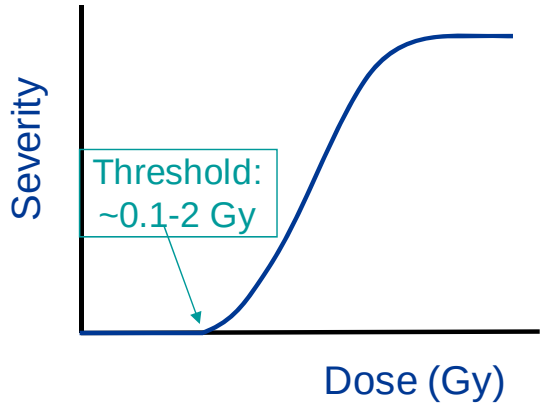

Figure 2: Tissue reactions: relationship between dose and severity of effect

Figure 2 shows that the severity of the effect increases with the dose received but that no effect will be observed below the threshold. The threshold dose is different for each effect as shown in Table 3 below.

Table 3: Threshold doses required to produce tissue reactions

Effect	Time to manifest	Threshold dose (mGy)
Bone Marrow Suppression	3-7 days	500
Skin: early erythema	2 – 24 hours	2 000
Skin: tissue necrosis	10 weeks	18 000
Eye lens: opacity	>5 years	500
Testes: permanent sterility	3 weeks	3 500
Ovaries: permanent sterility	<1 week	2 500

The doses measured for staff working in hospitals and for patients undergoing diagnostic x-ray examinations are of the order of mGy. Therefore deterministic effects are not usually observed. The exception is lens opacities. There is growing evidence that effects may be seen at lower doses than previously understood and that interventional radiologists and cardiologists may be developing cataracts at greater rates than the general population.

However, deterministic effects may be observed following interventional procedures and radiotherapy treatment. The aim of radiotherapy is to kill the cancer cells, which requires a dose of tens of Gy. Although the dose is localised to the tumour, the radiation passes through the skin so there is a real possibility of erythema. Interventional procedures are used to observe the flow of blood through the arteries (angiography) and often to treat narrowing and blockages. They have many advantages as the only alternative is invasive surgery. X-ray fluoroscopy (continuous application of low dose x-rays) is used to guide catheters through the arteries. Procedures which involve more than an hour of fluoroscopy with multiple digital images may approach the threshold levels for skin damage. However, these higher doses are often justified on the basis that the patient is very ill, often with a serious heart condition, and the risks associated with surgery are greater than those associated with the radiation dose.

2.4. Cancer and Heritable Effects

Unlike tissue reactions, which can only occur above a threshold dose, cancer and heritable effects are random and can occur at any dose. Since they are random, it is not possible to predict if the effect will occur in any one individual but we can use statistics to make a prediction of the number of people likely to be affected in a population.

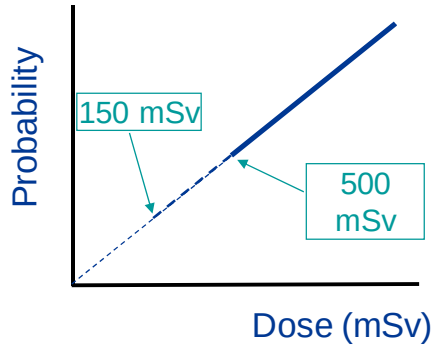

Figure 3: Cancer and heritable effects: relationship between dose and probability of effect

Figure 3 shows that the probability of an effect occurring ('risk') increases with the dose received. At the higher doses (solid line) we have good evidence for the relationship between dose and risk, but at lower doses, the data are less reliable because of the difficulty of isolating the effects of ionising radiation from all the other causes of cancer. This is explained further in sections 2.5 and 3.1/

Cancer and heritable effects are generally 'late' effects. They result from cell modification.

There is always a minimum period of time between irradiation and the appearance of the radiation induced tumour known as the **latent period**. The latent period varies from one tumour to another. Some types of leukaemia have a latent period of only a few years. For other tumours of longer latency it is not yet clear how the cancer risk proceeds with time so projection models are used.

2.5. Dose versus risk models

There has been a debate amongst the scientific community over the past few years regarding the relationship between dose and risk. Risk models are based predominantly on data from the Japanese atomic bomb survivors but there are some limitations associated with using this data. These are discussed further in Section 3.1. One of the main issues is that the doses received

were of the order of hundreds of mSv. How do we know the risks associated with doses below this? The truth is that we don't know and we must make an educated guess (although this 'guess' is likely to be based on complex mathematical models!).

The currently accepted model is called the 'Linear No Threshold' or 'LNT' model and is shown in Figure 4a. At zero dose you have zero risk and then your risk increases linearly with dose. This implies that cancer induction can occur at any dose.

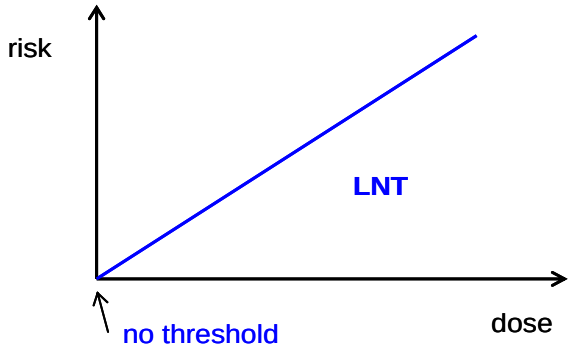

Figure 4a: LNT model

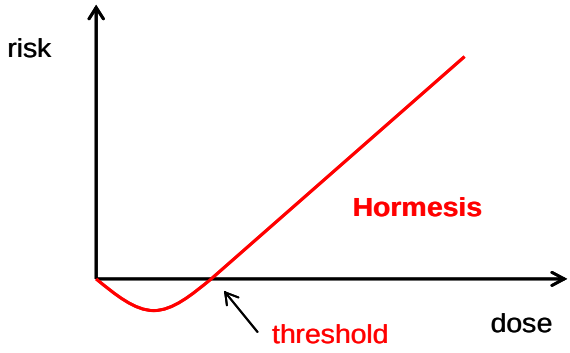

Figure 4b: Hormesis model

One alternative model is the 'hormesis' model and is shown in Figure 4b. At zero dose you have zero risk; but as dose increases you initially have a negative risk and then as dose increases further your risk increases linearly with dose. This model implies that low doses of radiation are actually beneficial and that there is a threshold dose for cancer induction. The theory behind this model is based on the idea that DNA repair genes can improve their defence mechanisms if they are exposed to low doses of radiation. It is a little bit like the idea of becoming immune to an illness. If you have an illness once (e.g. chicken pox), your body develops antibodies to prevent you getting the illness again.

The 'hormesis' model is considered credible but without more evidence it is unlikely to be adopted within the legislative framework for radiation protection.

Questions
Refer to Table 3 for threshold doses for deterministic effects.

2.1) Could a deterministic effect be observed in a patient who received the following doses?

a) 200 mGy _____

b) 2×10^3 mGy _____

c) 20 Gy _____

d) 0.4 Gy _____

2.2) A woman undergoes an interventional x-ray procedure. She receives a skin dose of 8 Gy during the procedure.

a) Will she experience a deterministic effect? YES / NO / MAYBE

b) If so, which effect do you think it would be (based on the table)?

_____

c) Will she experience a stochastic effect? YES / NO / MAYBE

3. Radiation dose and risk

The current probability for the lifetime risk of cancer in the UK is a little over 1 in 3. And approximately 1 in 4 people die from cancer. (The figure was 28% of deaths in 2010.) This is defined as the 'baseline' risk. However the risk of radiation-induced fatal cancer is much lower than the baseline risk and has been estimated as follows:

> A group exposed to known radiation doses is followed up and the cancer incidence is compared with the number of cancers expected in an age/sex matched control group which have not been exposed. The difference gives the **excess** cancer incidence. This raised risk of cancer per unit dose is called a risk factor. Large populations are required so as to obtain meaningful statistics.

The following populations have been used to obtain data:

- Atomic bomb survivors, whole body irradiation in Hiroshima and Nagasaki.
- Medical exposures, including radiotherapy treatments.
- Occupational exposure e.g. uranium miners, radiation workers and radium dial painters.

Radium dial painters used to lick their brushes so they could get them to a fine point before painting the faces of watches and instrument dials. Unfortunately they had no idea that they were ingesting a radioactive substance. It is only relatively recently that the dangers of radiation have become fully understood. In the 1920s radioactive toothpaste was available! It made your teeth glow in the dark and encouraged children to brush their teeth. It led to many incidences of oral, tongue, oesophagus and stomach cancers. Even in the 1950s, x-ray fluoroscopy was used in shoe shops to make sure that your shoes fit properly.

The early pioneers of radiation work such as the Curies and Roentgen's workers also died of radiation induced illness. Pierre and Marie Curie discovered Radium and pioneered its use for cancer therapy. Roentgen discovered X-rays, and their importance as a method of imaging inside the body.

3.1. Risk factors

The current risk factors are based on the Japanese atomic bomb survivors. However, there are some limitations associated with using this data. Firstly, the bomb data is obtained at high dose (hundreds of mSv) and high doserate and has to be extrapolated to low dose and low doserate for radiation protection purposes. Secondly, it is not possible to observe all survivors for their lifetime; therefore prediction models must be used. Thirdly, there will also be uncertainty when transferring risks across populations (e.g. Japanese to Western population) where the baseline risk is very different.

The uncertainty in the cancer risk estimates could be a factor of two.

Table 4: Lifetime risk of fatal cancer induction

Lifetime risk of fatal cancer induction	Whole population	Working population
ICRP 60 (1990)	5×10^{-5} per mSv (1 in 20,000 per mSv)	4×10^{-5} per mSv (1 in 23,000 per mSv)
ICRP 103 (2007)	4.4×10^{-5} per mSv (1 in 25,000 per mSv)	
NRPB* 1993 (UK)	5.9×10^{-5} per mSv (1 in 17,000 per mSv)	5×10^{-5} per mSv (1 in 20,000 per mSv)

**National Radiological Protection Board, now known as Public Health England (PHE)*

The ICRP recommends the use of a whole-population, risk factor of 5% per Sv for radiation detriment. In reality, the risk factor varies significantly with age – it is lower for the working population (18-65 years) because workers are, on average, healthier than non-workers and do not include children and young people.

In the UK, Public Health England (formerly the Health Protection Agency) has produced a report that looks at the risks of x-ray exposures for patients of both sexes and ages 0-99. (Report HPA-CRCE-028, available from

http://www.hpa.org.uk/Publications/Radiation/CRCEScientificAndTechnicalReportSeries/HPACRCE028/)

The risk factors presented in this report range from 20% per Sv for the youngest patients, to <0.1% per Sv for those aged 90+.

3.2. Staff risk: occupational exposure

Some typical annual doses for staff working in diagnostic radiology are shown in the table below with the corresponding level of risk.

Table 5: Typical annual doses received by staff working with ionising radiation

Occupation (Average annual dose)	Risk of death per annum
Diagnostic Radiology Radiographer (0.06 mSv)	1 in 333,000
Interventional Radiologist (0.35 mSv)	1 in 57,000
Cardiologist (0.20 mSv)	1 in 100,000
Nuclear medicine nurses, radiographers, technicians (0.71 mSv)	1 in 28,000
Radiotherapy source technicians (0.34 mSv)	1 in 59,000

For a radiographer receiving an annual effective dose of 0.06 mSv the risk of death per annum from cancer is 1 in 333,000. (Doses from: Ionising Radiation Exposure of the UK population - 2005 review, HPA-RPD-001)

Comparing these risks to those of other professions, it can be seen that the occupational risk of working in diagnostic radiology is comparable to or less than most other occupations.

Table 6: Comparison of risk of death in UK professions

Average annual risk of death in the UK from industrial accidents and from cancers due to radiation work	
Coal mining	1 in 9 200
Construction	1 in 17 000
Radiation work (0.5 mSv/yr)	1 in 40 000
Manufacturing industry	1 in 77 000
Service industry	1 in 333 000
Manufacture of electrical / optical equipment	1 in 500 000

3.3. Patient risk: medical exposures

Given that the baseline cancer risk is 1 in 3 and the baseline fatal cancer risk is 1 in 4, an x-ray examination will merely serve to increase this relative large underlying risk by a very small amount. For low dose examinations such as those of the chest, the increased risk of cancer is about one in a million. The risk from typical examinations is shown in the table below.

Table 7: Risk from typical x-ray examinations

Examination	Average effective dose (mSv)	Lifetime risk of fatal cancer per examination
Chest PA	0.017	1 in 980,000
Pelvis AP	0.66	1 in 25,000
Abdomen AP	0.7	1 in 25,000
Lumbar spine AP	0.69	1 in 25,000
Barium meal	2.6	1 in 6,400
Barium enema	7.2	1 in 2,300
CT Head	1.8	1 in 9,300
CT Chest	8.3	1 in 2,000
Mammogram	0.6*	1 in 33,000

* Note that the average Mean Glandular Dose from a two view mammogram is about 5 mGy. For women of screening age attending for two-view screening, the lifetime risk of radiation induced breast cancer is thought to be approximately 1 in 100,000 per mGy which equals 1 in 20,000.

Risk is strongly dependent on age at exposure. As it may take years or decades for a cancer to develop after exposure to radiation, the risks are reduced for people who are elderly at the time of exposure. Children however may have twice the lifetime risk of a middle aged person from the same examination.

This difference in risk is even more significant in mammography. The risk is approximately 7 times greater for a woman having an x-ray mammogram at age 35 compared to age 55.

3.4. Background radiation

The information in this section comes from the report *Ionising Radiation Exposure of the UK population - 2005 review, HPA-RPD-001.* We are all exposed to natural and artificial background radiation in our everyday lives. This comes from a number of sources.

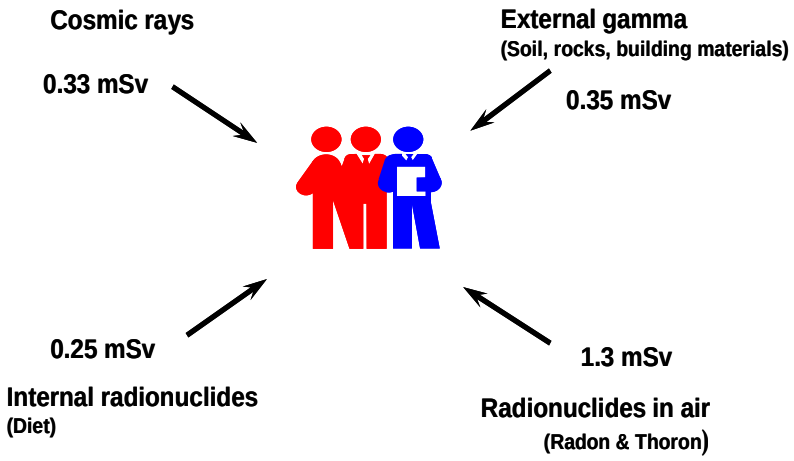

Figure 5: Sources of natural background radiation

Cosmic rays are produced when high energy particles from outer space interact with the atoms of the atmosphere and doserates increase with altitude. The difference between sea level and the top of a mountain is insignificant but the difference between ground level and flying altitudes is significant. A small contribution to our annual total is assumed to come from flying (we receive about 4 μSv per hour).

Some naturally occurring radionuclides in rocks, soil and consequently building materials emit gamma rays.

The greatest contribution comes from radionuclides in the air, mainly radon gas which contributes 1.2 mSv, with the remaining 0.1 mSv coming from thoron. Radon gas comes from uranium that occurs naturally in the ground and we all breathe it in. Levels vary across the country. Outdoors it disperses but can build up indoors because of the nature of the ground and level of ventilation. Radon decays producing alpha-particles that have potential to cause damage.

A certain amount of radionuclides are introduced through our diet including mineral water (and to a much smaller extent, tap water) and crops which take up uranium and thorium.

The average annual dose in the UK from all natural sources is 2.23 mSv. Due to the variation in radon levels, this ranges from a maximum of 6 mSv in Cornwall to about 1.5 mSv in the Isle of Wight.

We are also subject to a small amount of artificial radiation each year. The following figures are for the average person but would obviously increase for someone who had received several x-ray exposures or had a higher dose occupation, such as a pilot (more exposure to cosmic rays).

Artificial sources	**Average annual dose (mSv)**
Consumer products e.g. smoke alarms	0.0001
Nuclear fallout	0.006
Nuclear discharges	0.0009
Medical exposures	0.41
Occupational	0.006
Total (All sources)	2.65

The average annual dose in the UK from all background sources is 2.65 mSv. The average risk of death in one year associated with this is 1 in 7,500.

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Revised by Lorna Sweetman
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If you have any queries about this topic, please get in touch!

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