An Introduction to the Estimation of Risks Arising from Exposure to Low Doses of Ionising Radiation

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ABSTRACT

Exposure to ionising radiation leads to a radiation dose. The main health effect of low levels of radiation dose is an increase in the chance of developing cancer. The radiation risk factor quantifies the level of risk caused by a given amount of radiation dose, i.e. there will be an X% risk of getting cancer per unit of dose received. This document provides an introduction to the risks from exposure to low doses of radiation and explains the derivation of the radiation risk factors used in radiation protection. The examples are focused on radioactive waste management situations but the concepts can be applied to other situations.

The estimated values of risk factors are largely based on epidemiological studies of the Japanese atomic-bomb survivors, many of whom received medium to high doses of ionising radiation, and are supported by studies of other populations such as patients given medical exposures and workers receiving exposures at work. The International Commission on Radiation Protection (ICRP) used these estimates of radiation risk as the basis for their radiation protection system and made adjustments to allow for lower doses and lower dose rates, in order to estimate the risks of developing various cancers following exposure to ionising radiation from typical situations (e.g. medical, occupational or environmental exposures). Current ICRP recommendations (ICRP, 2007) for radiation protection assume an overall fatal cancer risk from low dose ionising radiation of about 5% per Sv.

The studies of the Japanese atomic-bomb survivors include many features of good epidemiological studies and the findings from studies of other populations are in reasonable agreement with them. Therefore HPA has confidence in the risk factors used by ICRP. Additionally, as a measure of their acceptance, the ICRP system of radiation protection and standards is applied internationally as well as in the UK. It is possible to estimate the accuracy of radiation risk models from the evidence provided by the epidemiology studies. HPA’s view is that, when considering the risk of all cancers in a population of all ages exposed to radiation at background dose levels and above, it is reasonable to assume that the estimates of risk used for protection purposes are accurate to within a factor of 3 either way for some radionuclides and for external exposure. For certain radionuclides the evidence suggests that the accuracy of risk estimates is likely to be around a factor of 10 either way.
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INTRODUCTION

1 INTRODUCTION

The aim of this document is to provide an introduction to the risks from exposure to low doses of radiation and to explain the derivation of radiation risk factors used in radiation protection. The examples are focused on radioactive waste management situations but the concepts can be applied to other situations.

Exposure to ionising radiation leads to a radiation dose (see Section 1.2 for more information on radiation dose). Radiation risk factors can be used to convert dose to risk to assess any potential health effects associated with a given dose. Risk factors quantify the level of risk caused by a given amount of radiation dose.

This report looks at the nature of health effects from exposure to ionising radiation; the differences arising from different types of exposure; the relationship between levels of dose and health effects; how risk factors are calculated; risk estimates in the context of other risks; and the uncertainties and confidence in risk factors. It also discusses clustering of health effects such as cases of leukaemia and whether such clusters could be linked to ionising radiation. More detailed descriptions of these topics can be found elsewhere (e.g. UNSCEAR 2008, ICRP 2007, COMARE 2006).

1.1 Health effects from exposure to ionising radiation

When ionising radiation passes through the organs and tissues of a living organism, some of the energy carried by the radiation is lost to the tissue’s cells and may cause damage to the DNA (genetic material) and other components within those cells. In most cases, damaged DNA is either successfully repaired by the body’s natural defence mechanisms, or the cell carrying the damage is eliminated. At high levels of dose there may be a substantial amount of cell killing, leading to obvious injury, e.g. skin reddening, organ damage and even death. At low levels of radiation dose there will be no obvious injury. However, although cells have very effective mechanisms for the repair of DNA damage resulting from radiation exposure and other causes, some DNA damage is more difficult to repair and sometimes mistakes occur, called mutations. Some mutations can result in changes in the characteristics of cells and set them on a path towards uncontrolled proliferation and cancer. Exposure to radiation is not the only way in which a cell can receive DNA damage or be triggered to become cancerous: DNA damage can occur spontaneously, or from exposure to chemicals, and some cancers are associated with specific infections. Hence, the body will carry some cells with these mutations from other causes and subsequent ionising radiation exposure may increase the number of these mutant cells. Therefore the main health effect of low levels of radiation dose is a small increase in the chance of cancer development (it should be remembered that cancer is a common disease, see Section 3). Usually, if cancer does subsequently develop, then it occurs many years after the exposure to ionising radiation. Because this health effect is related to the size of the radiation dose received, the additional risk of cancer resulting from very low doses is proportionately very low. As well as the possibility of causing cancer in the exposed individual, it is biologically feasible that mutations to genetic material could be passed on to future
generations (this is called a heritable effect). However, there is no direct evidence of radiation-induced heritable effects in humans (ICRP, 2007) and this genetic risk is judged to be considerably lower than that of cancer.

1.2 Different types of radiation and dose

There are different types of ionising radiation. The principal types are known as alpha, beta and gamma radiation. Different types of radiation have different properties. Alpha radiation* does not travel very far, and is easily stopped, by for example a sheet of paper. Beta radiation† travels further and is stopped for example by a sheet of aluminium, while gamma radiation‡ is more penetrating and will pass through dense material. For example a few centimetres of lead will typically be required to stop gamma radiation. The distance that radiation travels in material depends on the rate at which it loses energy along its path: the faster it loses energy, the shorter the distance it travels. There is no fundamental difference between the alpha, beta or gamma radiation that comes from naturally occurring materials and the alpha, beta or gamma radiation that comes from man made materials, although the particular energy associated with the radiation may differ.

A radiation dose is a measure of the energy deposited in the body tissue. Strictly this is referred to as the absorbed dose and it is a physically measurable quantity defined as the energy deposited per unit mass and is expressed in gray (Gy). However, the relationship between the radiation dose and any subsequent health effects is complicated and therefore two other units of dose have been defined, by the International Commission on Radiological Protection (ICRP) (ICRP 1991, 2007), to take account of this. Firstly, there is equivalent dose which is expressed in sieverts (Sv). This is based on the absorbed dose and takes account of the fact that types of radiation that lose energy more rapidly are more damaging to cells. Hence alpha radiation is more damaging per unit absorbed dose than gamma radiation. However, the results of such damage in terms of the health effect (e.g. cancer) will also depend on the organ and tissue being irradiated. This is taken into account in the second quantity effective dose, also expressed in sieverts (Sv). This is based on equivalent dose and takes account of the fact that some cells, tissues and organs are more sensitive to radiation than others. Hence effective dose is a measure of the energy deposited in the body tissue and the associated damage in terms of health effects.

Both equivalent dose and effective dose are defined by ICRP taking account of information on the health effects of different types of radiation. They provide a

* Alpha radiation is the name given to alpha particles that are emitted from the nucleus of an atom as it undergoes radioactive decay. An alpha particle is made up of two neutrons and two protons.
† Beta radiation is the name given to beta particles that are emitted from the nucleus of an atom as it undergoes radioactive decay. A beta particle is the same as an electron.
‡ Gamma radiation is the name given to high energy radiation waves emitted by an atom as it undergoes radioactive decay. It is basically the same type of radiation as light but with a much higher energy.
convenient method for the addition of doses received as a result of different types of exposure, see Section 2.

1.3 Different types of exposure

Since different types of radiation can travel different distances through body tissue the distribution of dose and effects will depend on the type of radiation. It will also depend on whether it is received from a source outside the body (external exposure) or from a source inside the body (internal exposure). In the case of gamma radiation which can travel through several centimetres of body tissue, the dose from a wide beam of radiation from a source outside a person will be the same as that from the same source distributed in the body. However, for alpha and beta radiation which travel much smaller distances, doses from external and internal sources will be different. For example, an alpha source is not a hazard if it is outside the body because the radiation cannot penetrate the layer of dead cells on the outside of the skin. However, it is hazardous if ingested or inhaled as it could become incorporated in tissues and cells and cause localised damage. Even from an internal source, alpha or beta radiation will not travel as far as gamma radiation in body tissue. Therefore the dose will be delivered in a smaller area of the body tissue, and the damage will depend on the sensitivity of the irradiated cells. Dose models are used to calculate doses to different body organs and tissues from external and internal sources.

1.4 Relationship between dose and health effects

High doses of radiation, ie doses of the order of several tens of sieverts, can kill a large number of cells and lead to serious injury or, in the worst cases, rapid death. However, for these types of injury there are dose thresholds, of the order of a few sieverts or higher, below which they do not occur or cannot be detected. At levels of dose below these threshold levels, the main health effect is an increased risk of getting cancer (see Section 1.1) and the increased risk depends on the dose received. At very low levels of dose (for example, a few tens of microsieverts) the assumed increase in the risk is very small and impossible to detect in epidemiological studies (studies of patterns and causes of diseases, see Section 4), so it is not possible to determine whether there is a dose level below which no effects occur at all. However, for protection purposes, it is commonly assumed that there is no ‘safe dose’ threshold, so that any level of exposure, however small, may cause harm, and that the relationship between risk and dose is linear, with the increased risk being proportional to the dose received. This is known as the linear no-threshold model (LNT) and the estimates of health risks from radiation in the ICRP system of radiation protection are based on this model. It is likely that dose-response relationships are different for different cancer types but linearity is regarded as a good overall assumption for radiological protection purposes. For example it is suggested by some that the relationship between risk and dose is linear but only above a threshold, or that the relationship is supra-linear, or that at low doses ionising radiation has a protective effect on cells. The CERRIE (Committee Examining Radiation Risks of Internal Emitters) (CERRIE, 2004) report discusses a number of possible dose/response
relationships. HPA’s view is that the LNT model is a scientifically defendable assumption for radiation protection purposes.

A key aim of radiation protection is to reduce the increased risk of getting cancer from all controllable sources of radiation as far as possible, without incurring disproportionate cost. One of the consequences of adopting the LNT model is that, however much money is spent, the increased risk could never be reduced to zero.

2 ESTIMATING RISKS FROM IONISING RADIATION

On the basis of the assumption of a linear no-threshold relationship between risk and dose, the risk that a health effect will occur can be determined from the dose, ie there will be an X% risk of getting cancer per unit of dose received. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has regularly reviewed information on radiation risks, most recently in a report published in 2008 (UNSCEAR, 2008). This estimation is largely based on epidemiological studies of the Japanese atomic bomb survivors (Preston et al, 2007), many of whom received medium to high doses of ionising radiation, and is supported by studies of other populations such as patients given medical exposures and workers receiving exposures while at work (occupational exposures). ICRP (2007) used the available UNSCEAR estimates of radiation risk, together with data from Preston et al (2007), as the basis for the risk estimates used in their radiation protection system. They made adjustments to allow for lower doses and lower dose rates, in order to make estimates of the risks of getting various cancers following exposure to ionising radiation from typical situations such as medical diagnostic, occupational or environmental exposures. ICRP estimated the risks of getting various cancers per unit dose received for males and females separately, and for adults and children at different ages and then combined them to provide one overall estimate of the risk of cancer per unit dose received. They also made adjustments to allow for the fact that the total risk of getting cancer depends on the underlying average risk of getting cancer and that differs from country to country. The ICRP risk estimates are therefore designed to give the likelihood of cancer occurring in an exposed population, for health protection purposes, rather than giving the risk to a specific individual. One of the reasons for combining the risks for females and males and for adults and children was so that the risk estimates do not appear to be more accurate than they actually are. Another important point is that control of exposure is applied to all workers irrespective of gender, and all members of the public from infants to adults. Hence the risk factors are essentially estimates for a ‘reference’ person.

The basis for the ICRP risk factors was reviewed and generally supported by the independent UK CERRIE report (CERRIE, 2004). CERRIE also concluded that advances in understanding the detailed interactions of radiation with tissue (see Section 5) may ultimately provide a complementary approach and called for further research.

Current ICRP recommendations (ICRP, 2007) for radiation protection indicate an overall fatal cancer risk from ionising radiation of about 5% per Sv for a population of all ages. Other views do exist, however, particularly regarding the validity of the adjustments
made when taking risk estimates that are based on exposures to high doses of external irradiation and then applying them to low doses or to internal exposure. This has led for example to the independent European Committee on Radiation Risk (ECRR) disagreeing with the ICRP risk factors and suggesting that there are large underestimates for some radionuclides (ECRR, 2003). HPA disagrees with the views expressed by the ECRR and has confidence that the ICRP risk factors are suitable for radiation protection purposes because they are supported by other studies that do consider low doses and internal exposure (HPA, 2009b). Equally, other reports claim much lower risks at low doses and even no risk at all because of an assumed low-dose threshold for the process of cancer induction (Tubiana et al, 2005). HPA thinks that there is not enough evidence to support these views.

ICRP also introduce the concept of radiation detriment. This takes into account the fact that some cancers are fatal, some are curable, and some have longer periods of illness leading to a reduced quality of life. It also takes into account the possibility of heritable effects. Therefore detriment is a more comprehensive measure of the health effects of exposure to radiation than the risk of dying from cancer on its own. Taking all this into account, ICRP calculates (ICRP, 2007) a risk of detriment (serious health effects) of 5.7% per Sv. To avoid the appearance of undue accuracy, HPA has recommended (HPA, 2009a) that a risk of 6% per Sv is used when performing assessments of radioactive waste disposal. The risk of detriment is therefore slightly greater than the risk of fatal cancer of 5% per Sv.

3 RISK ESTIMATES IN CONTEXT

When considering a risk in terms of a measurable value, it is important to be aware of what that actually means. A risk is a chance that something might happen; usually that something undesirable will occur as a result of a particular hazard. In this case a source of radioactive material, such as radioactive waste, is the hazard which leads to a risk that people, who may be workers or members of the public, may develop cancer sometime in the future. It is important to remember that it is only the people (and their future children) who actually receive a radiation dose that are subjected to this risk.

Also, the risk associated with the dose that a person receives is actually the risk for a ‘reference’ person; the models used for radiation protection purposes for the calculation of equivalent and effective dose (see Section 1.2) and the estimation of radiation risk factors are based on reference persons and do not take account of individual characteristics as it would be too complex and hence not practicable to do so (Harrison and Day, 2008). Similarly, the dose limits that are set for health protection purposes do not take account of individual characteristics but apply generally, e.g. to all members of the public. Nevertheless, more specific risk factors (e.g. those appropriate to the person’s age or sex) can be used in some detailed dose reconstruction studies for particular individuals and particular circumstances.

It is also important to remember that risk is a random process so that even if a person does receive a radiation dose it does not mean that they will develop cancer as a result;
they just have an increased chance of developing cancer sometime in the future. Therefore, if the doses and risks to a small population are low, then it is possible that no-one will actually develop cancer as a result of the exposure. This is why reliable estimates of risks can only be obtained from well conducted studies of large populations.

It is also important to consider the level of the risk in comparison to other risks, to help give an idea of exactly how dangerous the hazard is. For example, applying the ICRP recommendation that the risk of fatal cancer from ionising radiation is 5% per Sv to a dose of 20 μSv per year, which is one of the higher doses expected to be received by landfill workers handling Very Low Level (VLLW) radioactive waste from the non-nuclear industry (SNIFFER, 2007), gives an estimated risk of $10^{-6}$ or one in a million (0.0001%). This means that each worker who receives this dose has an additional risk of one in a million of dying from cancer as a result of receiving this radiation dose. The current average risk of dying from cancer in the UK is one in four (25%) (Cancer Research UK, 2008). Therefore the total risk of dying of cancer rises from around 25% to around 25.0001% for each worker who is exposed to this level of radiation dose. Expressed differently, this indicates that if 1 million workers handled this waste and each received this dose, then only one would be expected to die of a corresponding radiation induced cancer while about 250000 naturally occurring cancer deaths would be expected. Such an increase is not detectable because of the normal variations in cancer incidence within populations and because radiation-induced cancers cannot be distinguished clinically from other cancers.

The Ionising Radiations Regulations (IRR99) (UK Parliament, 2000) specify dose limits for people at work. Currently, this means that a dose limit of 20 mSv (effective dose) per year applies to employees of 18 years of age and above, and that workers who are likely to receive more than 6 mSv per year should be designated as classified workers. It should be noted that currently HSE would consider workers at non-purpose built disposal facilities for radioactive waste, eg a landfill site that also receives hazardous wastes, to be occupationally exposed to radiation. However, it is unlikely that they would need to be classified workers.

UK government regulations (UK Parliament, 2000) specify that doses to members of the public should not exceed a value of 1mSv (one thousandth of a sievert) per year and any management or disposal options for radioactive waste management would have to comply with this. A dose of 1 mSv per year is equivalent to an additional risk of fatal cancer of one in twenty thousand (0.005%) per year. This would also not be detectable among normal background levels of cancer risk. The 20 μSv per year dose discussed above is a factor of 50 lower than this public limit.

The Health and Safety Executive (HSE) have looked at levels of risk, and in particular have assessed tolerability and acceptability of different levels of risk. As a risk increases the tolerability of that risk can be considered to move from being acceptable, to being tolerable, to being unacceptable (HSE, 2001). HSE describe an “acceptable” risk as one

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* A term used to cover a range of industries, hospitals, research establishments and other establishments that use radioactive materials but are not part of the nuclear power industry.
“that for purposes of life or work, we are prepared to take pretty well as it is”, while a “tolerable” one is not regarded as negligible or something we might ignore, but rather as something we need to keep under review and reduce still further if and as we can (HSE, 1988). An individual risk level of 1 in a million (1 in 1000000, or 0.0001%) per year is generally considered to be acceptable, while 1 in a hundred thousand (1 in 100000 or 0.001%) per year is considered to be tolerable. The regulators base the requirements that they impose on operators on these levels of acceptability and tolerability, while still requiring that doses should be reduced below these levels as far as possible, allowing for social and economic factors. This process of keeping doses as low as reasonably achievable without incurring disproportionate cost is called optimisation and is a key principle of radiation protection.

Other levels of risk that are often found helpful for comparison purposes are the projected annual risk of fatal cancer from the average natural radiation dose in the UK (roughly 1 in ten thousand) and the annual risk of death from lightning (1 in 18.7 million).

A dose can also be put into context by considering the average annual dose of radiation received by people in the UK. The average annual dose of 2.7 mSv is made up of doses from naturally occurring and artificial (man made) radiation sources, as shown in Figure 1 (Watson et al, 2005). The greatest contribution comes from naturally occurring sources, giving an average annual dose of 2.2mSv. Thus the 20 μSv per year discussed above equates to 0.75% of the UK average annual dose from natural and artificial sources of 2.7 mSv.

![Figure 1 Breakdown of the average annual dose of ionising radiation received in the UK](image)

**Figure 1** Breakdown of the average annual dose of ionising radiation received in the UK

4 **EPIDEMIOLOGY**

Estimates of health risks from radiation are based mainly on information from studies of human populations exposed to radiation. Epidemiology (the study of the causes,
distribution, and control of disease in populations) has played a vital role in identifying and quantifying the health risks of cigarette smoking and exposure to agents such as asbestos, as well as radiation.

Not all epidemiological studies are of equal value. A good study for the purposes of deriving estimates of radiation risks will include

- high statistical power, which will require
  - a large number of individuals, ideally including both male and female subjects, covering a range of ages;
  - long-term follow-up for mortality and cancer incidence;
  - a range of doses within the exposed population;
- low potential for any bias (bias may be introduced for example if members of the study population are selected because of suspected ill-health);
- care to take account of confounding factors, such as the effects of smoking when considering lung cancer;
- reasonably accurate and precise estimates of individual exposures;
- analysis by appropriate statistical methods.

In general, epidemiologists take considerable care in the design, conduct and analysis of studies to minimise the potential for bias and confounding and to maximise statistical power. However, it is difficult to eliminate all potential flaws from a study and therefore, when evaluating epidemiological findings, it is important to consider how each study was formulated and carried out (Elwood, 2007), rather than to accept the results at face value. Guidelines proposed by Sir Austin Bradford Hill (Hill, 1965) are often used to this end.

The next step in evaluating the results of an epidemiological study is to compare the results with existing knowledge, including biological and mechanistic information, whilst at the same time recognising that the causes of disease are not always fully understood. As some individual studies may not be powerful enough on their own, it may be necessary to look at the results of many studies in order to get a better understanding of the overall findings.

5 UNCERTAINTIES IN ESTIMATES OF RADIATION RISK FACTORS

The primary basis of radiation risk factors lies in epidemiological studies. Any epidemiological study involves statistical analyses, with the end result that - however good the study - there will always be some uncertainty in the findings because of the inherent statistical variation in the data. Calculation of radiation risk factors also relies on knowing the dose received by the exposed population, and any estimation or
measurement of this will also be subject to some degree of uncertainty. In addition to this, further uncertainties are introduced if the risk factor is to be applied to a different exposure situation than that in the study. For example, application of the estimated risk factor from high gamma doses and dose rates to low dose and/or low dose rate exposures, or to internal exposures, or to non-gamma radiation, or to a different population. These differences are taken into account on the basis of comparison with studies involving other exposure situations and also experimental work, but there are uncertainties involved in these calculations.

The precision and accuracy with which risk can be estimated is also limited by current knowledge of the biological effects of radiation. Some areas are more fully understood than others. Some effects, such as radiation-induced instability* and bystander effects† are less well understood, particularly in relation to their contribution to cancer incidence at low doses. Radiation research continues to investigate, using a wide range of biological methods, the mechanisms of how tissue reacts to ionising radiation in order to increase understanding of these effects. While current knowledge is not sufficient to fully understand these mechanisms, the estimation of radiation risk factors is based upon direct human epidemiological data, and therefore these biological mechanisms will be built into the risk estimates already. However, a common observation of these effects is that they saturate with increasing dose, so that the effect may be more significant at lower doses than it is at higher doses, (reviewed by Prise et al 2003, Smith et al 2003), and hence high dose epidemiological studies may not fully account for them. In general, it can be said that uncertainties in assessments of radiation doses from internal exposures are larger than those from external exposures, and the degree of uncertainty differs between various radionuclides.

It is important to acknowledge that there may be interactions - whether synergistic or antagonistic - between radiation and other toxic insults, and this is not explicitly addressed in current radiation protection models. However, very few reports of interactions between ionising radiation and other agents are available. One notable exception is the epidemiological study of lung cancer in those exposed to radon gas which demonstrates a strong synergy between tobacco smoke and radon in the development of lung cancer (Darby et al 2005). It should of course be noted that in good epidemiological studies of radiation risk, the exposed and unexposed populations will both have received exposures to other environmental agents. In this respect interactions between radiation and other agents are implicitly taken into account in the ICRP radiation risk estimates. Limited experimental evidence of interaction is also available. For example, Lord et al (1998) explored the possibility that an earlier radiation dose predisposes an organism to the development of cancer following subsequent exposure to a conventional carcinogen. In other words, radiation is unlikely to be the only causative agent in low dose exposure circumstances, but it may play an important role in the development of diseases of environmental origin.

Despite all these sources of uncertainty, there is actually a lot of information available on the effects of radiation on human tissues and this has been used by international groups

* Persistent increase in rate of genetic changes in cell populations
† Cellular effects seen in un-irradiated cells neighbouring the cells exposed to radiation
of scientists to gain a good basic understanding of the risks from radiation. Research into the mechanisms of how tissue reacts to radiation is also continuing (e.g. Department of Health, 2008). In summary, there are a number of unavoidable uncertainties in the estimates of the radiation risk (UNSCEAR, 2008; CERRIE, 2004, Baverstock, 2008) and this is an area of ongoing research and critical analysis.

6 REASONS FOR CONFIDENCE IN THE CURRENT RADIATION RISK FACTORS

The studies of the Japanese atomic-bomb (A-bomb) survivors, on which estimates of radiation risk factors are primarily based, include many features of good epidemiological studies. They cover a large population, of both sexes, and a wide range of ages. Because the population was not selected on the basis of disease, or other similar factors, there is little potential for the population studied to be biased towards a particular type of person. There has been a long period of follow up, during which the population has been assessed for both mortality and cancer incidence. In addition, individuals received a wide range of doses, and because of the extensive effort spent in the decades since the bombings to develop estimates of radiation doses (Cullings et al, 2006) there is confidence in the accuracy of the estimated doses.

Studies of other populations exposed occupationally, medically or environmentally give reasonable agreement with the A-bomb studies, providing reassurance that the risk factors are about right. Examples are populations exposed to the naturally occurring radioactive gas, radon (both domestic exposure and uranium miners) and workers in the nuclear industry (UNSCEAR, 2008). Some of these studies involved populations who received relatively low dose or dose rate radiation, or internal exposure, or alpha emitters, thus giving reassurance that the risk factors may be applied to a range of exposure situations. One of the studies is based on the actual (low) doses received by workers in the UK over the last 50-60 years, and provides good consistency with risk factors derived from high doses (Muirhead et al, 2009). This study also shows a decrease in the risk of cancers with a decrease in dose.

The reliability of the estimates of radiation risk factor depends on the level of dose being considered because the level of dose determines whether it is possible to see any health effects in an epidemiology study. Uncertainties are lowest at the levels of dose at which health effects have been observed (doses of about 10mSv or greater). At lower doses where the actual risk is very low and has not been detected in epidemiological studies, the proportional level of uncertainty is greater. However, it is possible to estimate an upper bound on the uncertainty at these low doses because otherwise some health effects would actually have been detected. As discussed in Section 3 people receive average doses of a few mSv from natural background radiation, but the estimated cancer risk associated with such exposures cannot be demonstrated by epidemiological studies.

Despite the many uncertainties involved in estimates of radiation risks, it is possible to estimate the accuracy of the radiation risk models from the evidence provided by the
epidemiology studies. HPA’s view is that, when considering the risk of all cancers in a population of all ages, it is reasonable to assume that the estimates of risk provided by these models are accurate to within a factor of 3 either way for some radionuclides and for external exposure. For certain radionuclides the evidence suggests that the accuracy of the risk estimates is likely to be around a factor of 10 either way. For example, for external irradiation and all cancers they are accurate to within a factor of 4 or less (Muirhead et al, 2009); for certain internal exposures and specific cancers they are within a factor of 10 (Harrison and Day, 2008); for external exposure and leukaemia they are within a factor of 3 (Muirhead et al, 2009). There is no reason to believe that risk estimates are underestimated by a factor of 100 or more as has been suggested following observations of clustering of health effects as discussed in Section 7.

Additionally, as a measure of their acceptance, the ICRP (ICRP, 1991) system of radiation protection and standards is applied internationally as well as in the UK.

7 CLUSTERING OF HEALTH EFFECTS SUCH AS LEUKAEMIAS

Over the past 25 years clusters of childhood cancers, mainly leukaemia, have been reported around some nuclear sites in the UK. Such reports have prompted suggestions that ionising radiation could be responsible for these cancer clusters. However, measured radiation levels around these sites are too low, by more than a factor of a hundred, to account for the clusters, using radiation risk estimates accepted by ICRP and other international bodies (UNSCEAR, WHO). Uncertainties in these risk estimates do not account for this discrepancy. Furthermore, doses due to radioactive releases from nuclear sites are lower than those from natural sources (see the piechart in section 3). As a result of the reports of clusters, there have been numerous epidemiological studies concerning childhood leukaemia and other cancers in relation to nuclear sites, and also suggestions that the radiation risk factors may be wrong. Several studies have been instigated and co-ordinated by the Committee on Medical Aspects of Radiation in the Environment (COMARE) (COMARE 1986, 1988, 1989, 1996, 2002, 2005, 2006). One of the main problems is to determine whether or not any cluster has just arisen by chance. In 2005 COMARE published analyses (COMARE, 2005) carried out on a national data set of childhood cancer cases which found no evidence of excess numbers of cases of childhood cancer around any of the nuclear power stations in Great Britain, although some clusters were found around other nuclear installations eg Sellafield and Dounreay. With approximately 32,000 cases in the time period 1969–1993, this is the largest data set of childhood cancer cases ever compiled anywhere in the world, giving considerable confidence in the results. Although the clusters around Sellafield and Dounreay are statistically significant, the diseases are rare: there were 10 cases of leukaemia or Hodgkin’s Disease in Seascale, near Sellafield, between 1954 and 2001, a period of 47 years (COMARE, 2002).

The 11th COMARE report (COMARE, 2006) examined the general pattern of childhood leukaemia and other childhood cancers within Great Britain. It reported that childhood leukaemia and many other types of childhood cancers do not occur evenly within the population of Great Britain, but show more variation than would be expected from simple
random or chance variations. In other words, childhood cancers tend to cluster ‘normally’ and some clusters have been seen around non-nuclear industrial features eg railway lines. It is not known why childhood cancers tend to cluster like this, but it is recommended that epidemiological studies should allow for the fact that such ‘normal’ clustering does occur since this affects whether any clustering can be considered to be ‘exceptional’ (COMARE, 2006). Other analyses conducted in Great Britain (McNally et al, 2009a, 2009b), various parts of Europe (Alexander et al, 1998) and the United States (Wartenberg et al, 2004) have also reported a tendency for childhood leukaemia to cluster normally.

In their 10th report COMARE give some advice on interpretation of apparent clusters: “The clusters that become a cause of public or media concern tend, however, to come to attention either through a chance observation or through a non-systematic collection of information that results in an apparently striking aggregation of cases. Such observations are impossible to evaluate in a precise statistical way and the process of assessing them is rather complex. A suspected cluster could be an indication of a causative factor in the local environment. However, it could also be the result of chance, or due to a misclassification of cancer cases, or a miscalculation of the number of cases (for instance, because some cases have been counted twice, are in the wrong age group, or were outside the area of interest at the time of their diagnosis)”. In other words, many reports of clusters are not real clusters but are only identified as a result of incomplete or poor analysis of the data. When these suspected clusters are subsequently investigated using rigorous statistical techniques, with careful attention to details of when and where the individual cases arose, the clusters are found not to exist.

A recent study (Spix et al, 2008; Kaatsch et al, 2008) in Germany reported an increased risk of cancer amongst children less than 5 years of age living within 5 km proximity of nuclear power plants in Germany. The authors also noted that the clusters were not explained by the levels of radiation from routine nuclear power plant operation and that the risk of cancer increased the closer the child lived to the power plant. This study has been interpreted by some as demonstrating that the risks from radiation have been underestimated. However, HPA strongly disagrees with this interpretation because, amongst other reasons, it is a study that is based on location not on the dose received and therefore it is not valid to infer a dose/risk relationship. A similar conclusion has been reached by the German Commission on Radiological Protection (SSK, 2008). The study also did not take account of the ‘normal’ clustering of childhood cancer, as recommended by COMARE, and hence it is difficult to draw conclusions from it. Nevertheless, it is still viewed by some as a key study because it reported an increased risk with proximity to the power plant. However, the evidence for this trend was restricted to within 5km of the nuclear power plant and no evidence of raised risk, or indeed of this trend with distance from the power plant, was found at greater distances (SSK, 2008). Similar studies in France (Laurier et al, 2008) and UK (Bithell et al, 2008) have not replicated the findings of the German study, and hence it is not possible to draw any conclusions regarding the risks from radiation from the German study.

Some clusters of childhood cancer, including leukaemia, have been identified near nuclear installations (not power stations) in the UK (COMARE, 2005) but the levels of radiation are not high enough, by factors of hundreds, to account for them. Furthermore, the doses from releases of radionuclides from these installations are less than the dose
from natural levels of radiation. Therefore, if ionising radiation were to be responsible for these clusters then the dose or risk estimates would have to be wrong by factors of hundreds. However, if radiation risk estimates were factors of 100 wrong, there would be evidence of higher incidence of the disease in other populations than is seen. Also, if there was another significant (unknown) source of radiation at these sites then there would be a general increase in adult cancer in the area as well, and this is not seen.

Based on current risk models, it has been estimated that about 20% of all childhood leukaemias might be due to natural background radiation (Wakeford et al, 2009). However, epidemiological studies of childhood cancer have generally not shown associations with levels of natural radiation (eg. Richardson et al, 1995; UK Childhood Cancer Study Investigators 2002a, 2002b), although there have been some reports suggesting a link between childhood leukaemia and exposure to radon in homes (eg. Raaschou-Nielsen et al, 2008).

HPA is therefore confident that the radiation risk estimates are not far enough out to account for the reported clusters, and concludes that there must be some other cause for these cancers.

Unfortunately it is very difficult for non experts to understand the importance of particular epidemiological studies and therefore HPA suggests that the best approach is to rely on the peer review process and to put greater reliance on publications that are in the peer reviewed literature than on those that are not.

8 CONCLUSIONS

To summarise, a low dose of radiation is one of many factors that can lead to an increased risk of cancer, but there are other possible factors, e.g exposure to particular chemicals or infections. Based on the body of evidence that has been collected over a large number of years, including detailed, regular and recent reviews of biological and epidemiological data, the HPA has confidence that the radiation risk factors used by ICRP provide a sound basis for a radiological protection system. HPA considers that the COMARE study of clusters of childhood leukaemias in the UK provides evidence that the risks from radiation have not been substantially underestimated.

The radiological protection system recommended by ICRP is applied internationally and in the UK.

There are a number of unavoidable uncertainties in the estimates of the radiation risk and this is an area of ongoing research and critical analysis.

9 REFERENCES

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