Allen, Pam, NMENV

From: Maestas, Ricardo, NMENV
Sent: Thursday, June 26, 2014 8:05 AM
To: Allen, Pam, NMENV
Subject: FW: Radioactivity (dpm) vs. Radiation (mrem)
Attachments: ecrr2010.pdf

Email and att. for March

From: Smith, Coleman, NMENV
Sent: Wednesday, March 19, 2014 5:17 PM
To: Kliphuis, Trais, NMENV
Cc: Maestas, Ricardo, NMENV; Holmes, Steve, NMENV
Subject: RE: Radioactivity (dpm) vs. Radiation (mrem)

Trais,

I just added on to your e-mail. It is interesting how high our everyday cancer risk due to background radiation is about 1 in 5 chance, which is huge when compared to values in the E-4 to E-6 range. I didn't cover the actual conversion of DPM to rem because in both models, many factors combine to yield "effective" and "equivalent" doses, which would only add to the confusion. Let me know if this is what you had in mind. I can edit.

Cole

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From: Kliphuis, Trais, NMENV
Sent: Wednesday, March 19, 2014 9:49 AM
To: Smith, Coleman, NMENV
Subject: Radioactivity (dpm) vs. Radiation (mrem)
Importance: High

Cole,
Can you add what you said in my office to the text below? Try to keep it really clear – probably or posting on our web at some point. **Thanks.**

Here are some more basics that help explain the exposures:

Each radionuclide (e.g. Plutonium, Americium) represents a somewhat different health risk (mrem values). Currently, DOE health physisicts are estimating the relase to have created an exposure of 0.5 mrem. The health physicists' estimations are based on a model that converts activity (dpm) to radiation exposure (mrem). The existing model used in the US and some European Union countries is called the International Commission on Radiological Protection (ICRP) model. One of the major shortcomings of this model is that it fails to predict cancer rates among persons living near a

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nuclear facility and receiving a chronic low-level dose. In the late 1990's, when the low-level chronic radiation effects due to Chernobyl started to become apparent, the European Committee on Radiation Risk (ECRR) was formed. The main focus for the ECRR was (and is) to provide a new and updated model for the prediction of health risks associated with the inhalation of radionuclides. This model uses epidemiological data and scientific reasoning using historic absorbed dose data and known physico-chemical behaviour of elements. According to the ECRR model, the ICRP model underestimates internal dose health effects by one to two orders of magnitude.

In the United States, the National Regulatory Commission (NRC) and the Department of Energy (DOE) have adopted the ICRP model, and according to regulation, is the only model that can be applied for internal or external dose assessment. While the ICRP model accurately predicts a linear-no-threshold response for external high-level doses, it does not apply to low-level, chronic internal or external doses. For example, the following table summarizes the factors considered in each model:

The health effects of low-level radiation to be considered by ECRR compared with ICRP and other risk agencies.

Possible health effects	ICRP and risk agencies*	ECRR Committee
Fatal cancer	Yes	Yes
Non-fatal cancer	No	Yes
Benign neoplasm	No	Yes
Heritable damage	Yes	Yes
Infant mortality	No	Yes
Birthrate reduction	No	Yes
Low Birthweight	No	Yes
IQ retardation	Yes	Yes
Heart disease	No	Yes
General reduction in health and non-specific life shortening	No	Yes

**UNSCEAR, BEIR, NCRP, NRPB and EU member state agencies*

A comparison between the ICRP and ECRR models shows that ICRP assumes a threshold below which there is zero risk (100 mSv, 10 rem):

ICRP2007 and ECRR modified risk factors for whole population for whole body effects.

Outcome	ICRP risk factor (per Sievert)	ECRR risk factor (per Sievert ECRR)
Fatal cancer	Not specified	0.1
Non-fatal cancer	0.055	0.2
Benign neoplasm	Not considered	being assessed[b]
Heritable disease	0.02	0.04
Malformation after *in utero* exposure	100mSv threshold	No threshold
Heart disease	Not assumed	0.05

Cancer after *in utero* exposure	0.2[a]	50
IQ lowering after *in utero* exposure	30 IQ points; 100mSv threshold	30 IQ points; no threshold
Severe retardation after *in utero* exposure	0.4; 100mSv threshold	0.8; no threshold

Nominal probability coefficient expressed as Sv-1

[a] *This is the ICRP 1990 value, ICRP2007 avoids giving a value but states that the risk is the same as for exposure in early childhood and neglects to give that value.*

[b] *For radiogenic benign intracranial tumours see Schmitz Feuerhake et al 2009*

Note: Values for workers, where applicable, are slightly less than these owing to the different age distribution of workers. Refer to the ICRP publications for details.

The ECRR recommends that nuclear workers be limited to an annual total (internal + external) exposure ≤ 2 mSv (200 mrem).

The ECRR maximum permissible annual total dose to a member of the public is 0.1 mSv (0.001 mrem)

The NRC limits exposure (ICRP) for nuclear workers to an annual total effective dose equivalent (TEDE) of 5 rem (10 CFR 20.1201).

The NRC limit for the annual dose to a member of the public (ICRP model) is a TEDE of 0.1 mrem (10 CFR 20.1301).

The DOE exposure limit for workers is an annual TEDE of 5 rem (10 CFR 835.202, identical to the NRC limit).

The DOE exposure limit for an adult member of the public is 100 mrem annual TEDE (DOE Order 458.1).

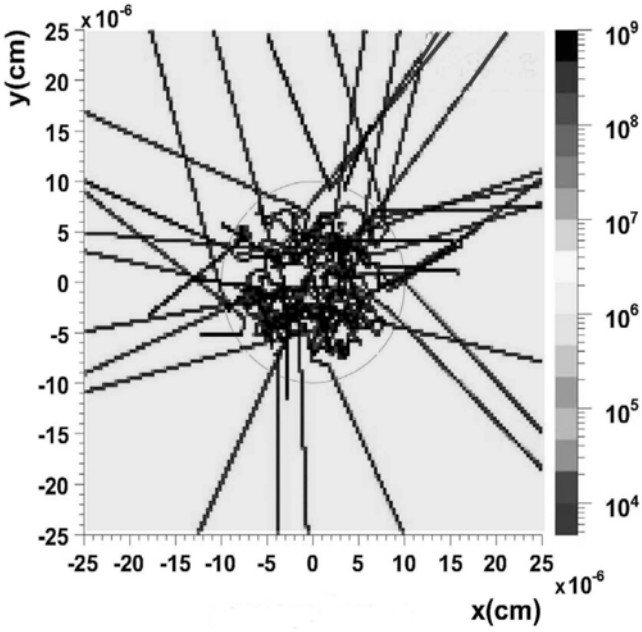
The salient point to remember is that both the NRC and the DOE use the ICRP model. This model was developed based largely on data from Hiroshima; health consequences from high external doses could be predicted from this data, but the ICRP model does not address more recent advances in cellular biology and anisotropic interaction of ionizing radiation with DNA.

In the United States and using the ICRP model, EPA health physicists currently estimate that overall, if each person in a group of 10,000 people exposed to 1 rem (1000 mrem) of ionizing radiation, in small doses over a life time, we would expect 5 or 6 more people to die of cancer than would otherwise (cancer risk = 6/10,000, or 6E-4). In this group of 10,000 people, we can expect about 2,000 to die of cancer from all non-radiation causes (cancer risk = 2,000/10,000, or 2E-1). The accumulated exposure to 1 rem of radiation, would increase that number to about 2005 or 2006. Most people receive about 3 tenths of a rem (300 mrem) every year from natural background sources of radiation (mostly radon).
[Adapted from: http://www.epa.gov/radiation/understand/health_effects.html]

As noted previously, the ICRP model assumes that risk is linearly proportional to dose.

ECRR

2010 Recommendations of the European Committee on Radiation Risk



The Health Effects of Exposure to Low Doses of Ionizing Radiation

Regulators' Edition: Brussels 2010

2010 Recommendations of the ECRR

The Health Effects of Exposure to Low Doses of Ionizing Radiation

Regulators' Edition

Edited by Chris Busby
with
Rosalie Bertell, Inge Schmitz-Feuerhake,
Molly Scott Cato and Alexey Yablokov

Published on behalf of the European Committee on Radiation Risk
Comité Européen sur le Risque de l'Irradiation

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European Committee on Radiation Risk
Comité Européen sur le Risque de l'Irradiation

Secretary: Grattan Healy
Scientific Secretary: C.C.Busby
Website: www.euradcom.org

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(Cover picture: XY projection of secondary photoelectron tracks induced in a 20nm diameter Uranium nanoparticle by 1000 natural background radiation photons of energy 100keV;in a water particle of the same size this exposure would produce 0.04 tracks in the same XY plane. FLUKA Monte Carlo code. Elsaessar et al. 2009)

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ECRR 2003 was dedicated to **Prof. Alice M Stewart**, the first scientist to demonstrate the exquisite sensitivity of the human organism to ionizing radiation. The Committee dedicates this present volume to the memory of:

Prof. Edward P Radford,
Physician and Epidemiologist
"There is no safe dose of radiation"

Radford was appointed Chair of the BEIR III committee of the US National Academy of Sciences. His BEIR report in 1979 drew attention to the inadequacies of the then-current radiation risk model. It was withdrawn and suppressed but he resigned and published a dissenting report. His career was destroyed.

In 2009 the ECRR awarded the Ed Radford Memorial Prize, donated by his widow Jennifer and the Radford family in the USA to

Prof. Yuri I Bandashevsky
Physician and Epidemiologist

Bandashevsky drew attention, through his scientific research and self publications in English, to the effects of internal radioactivity from Chernobyl on the health of the children of Belarus and was rewarded by arrest and imprisonment.

Preface

The presentation in 2003 of the new radiation exposure model of the European Committee on Radiation Risk caused something of a revolution in the focus of scientists and politicians on the adequacy of previous scientific theories of the effects of radiation on living systems. This was long overdue, of course, since evidence has been available for more than 40 years that it was unsafe to use studies of external acute radiation to inform about risk from internal chronic exposures to evolutionarily novel radionuclides. Such a scientific paradigm shift is not easy: the course and direction of the nuclear, military, economic and political machine dedicated to the development of nuclear energy and its military applications is monolithic and has massive inertia. It was therefore surprising and encouraging that ECRR2003 received such attention, and effectively brought about a new and intense interest in the flaw in the then-current philosophy of radiation risk: the physics-based concept of absorbed dose. The support and encouragement for the new model, and its success in many court cases (where it was invariably set against the ICRP model) was perhaps assisted by the increasing evidence from Chernobyl fallout exposures and from examination of Depleted Uranium effects which were emerging at the time of ECRR2003. The success of the ECRR model is that it gives the *correct answer* to the question about the numbers of cancers or other illnesses that follow an exposure to internal fission products. This is immediately clear to anyone: to juries and judges as well as ordinary members of the public. It received powerful support from reports of increases in cancer in Belarus after Chernobyl and also from the epidemiological studies of Martin Tondel of cancer in northern Sweden published in 2004: Tondel's findings of a statistically significant 11% increase in cancer per 100kBq/m^2 of Cs-137 contamination from Chernobyl are almost exactly predicted by the ECRR2003 model.

There have also been developments in laboratory science that can be explained in the new model but are quite impossible to explain in the old ICRP model. One of these is the understanding that elements of high atomic number, like Uranium (but also non-radioactive elements like Platinum, Gold etc.) have the ability to alter the absorption characteristics of tissues in which they are embedded. Uranium is the central element around which the nuclear fuel cycle revolves, and huge quantities of the substance have been contaminating the biosphere since early in the last century. It is therefore necessary to update the ECRR risk model and include consideration of these 'phantom radiation effects'. The widespread dispersion of Uranium from weapons usage has made it necessary to add a chapter on Uranium weapons. Since its founding in Brussels in 1998, the ECRR has been joined by many eminent radiation scientists from many countries. It will be clear from this new revised edition that the pressure on politicians and scientists to change their understanding of the health effects of ionizing radiation is now too great to ignore.

1
The ECRR

1.1 *The background*

The European Committee on Radiation Risk is a spontaneous creation of Civil Society which was faced with clear and alarming evidence of the failure of its democratic institutions to protect it from the effects of radioactive pollution. Predictably, the engine which generated this development was the Green movement, the result of another and earlier Civil Society reassessment of the aims and ideologies behind the systematic exploitation and contamination of the planet. The ECRR was formed in 1997 following a resolution made at a conference in Brussels arranged by the Green Group in the European Parliament. The meeting was called specifically to discuss the details of the Directive Euratom 96/29, now known as the Basic Safety Standards Directive. This Directive has, since May 2000, been EU Law regulating exposure to radiation and to releases to the environment of radioactivity in most countries of the Union. The Euratom Treaty preceded the Treaty of Rome and so once the document had been passed by the Council of Ministers there was no legal requirement for the European Parliament to address it. It was thus cleared without significant amendment although, astonishingly, it contained a statutory framework for the *recycling of radioactive waste into consumer goods* so long as the concentrations of itemised radionuclides were below certain levels.

The Greens, who had attempted to amend the draft with only limited success, were concerned about the lack of democratic control over such a seemingly important issue and wished for some scientific advice regarding the health effects which might follow the recycling of man-made radioactivity. The feeling of the meeting was that there was considerable disagreement over the health effects of low-level radiation and that this issue should be explored on a formal level. To this end the meeting decided to set up a new body which they named the European Committee on Radiation Risk (ECRR). The remit of this group was to investigate and ultimately report on the issue in a way that considered all the available scientific evidence. In particular, the Committee's remit was to make no assumptions whatever about preceding science and to remain independent from the previous risk assessment committees such as the International Commission on Radiological Protection (ICRP), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the European Commission and risk agencies in any EU member State.

The ECRR's remit was and is:

1. To independently estimate, based on its own evaluation of all scientific sources, in as much detail as necessary, using the most appropriate scientific framework, all of the risks arising from exposure to radiation, taking a precautionary approach.

2. To develop its best scientific predictive model of detriment following exposure to radiation, presenting observations which appear to support or challenge this model, and highlighting areas of research which are needed to further complete the picture.
3. To develop an ethical analysis and philosophical framework to form the basis of its policy recommendations, related to the state of scientific knowledge, lived experience and the Precautionary Principle.
4. To present the risks and the detriment model, with the supporting analysis, in a manner to enable and assist transparent policy decisions to be made on radiation protection of the public and the wider environment.

Shortly after the ECRR was formalised, the Scientific Options Assessment (STOA) Unit of the European Parliament arranged (on the 5th Feb. 1998) a meeting in Brussels to consider criticisms of the 'Basic Safety Standards' for the public and workers from exposure to ionising radiation. At this meeting the eminent Canadian scientist Dr. Bertell argued that the ICRP, for historical reasons to do with the development of nuclear weapons and nuclear power during the Cold War period, were biased in favour of the nuclear industry and that their conclusions and advice in the area of low-level radiation and health were insecure.

Unfortunately, the STOA rapporteur, the late Prof. Assimakopoulos, did not accurately report the presentation of Dr Bertell, which was wide-ranging and extremely critical of the ICRP and its advice (Asssimakopoulos 1998). Responding for the ICRP, Dr.Valentin, its scientific secretary, told the workshop that the ICRP was an independent body which gave advice on radiation safety, *but that those who considered this advice unsafe or questionable were entirely free to consult any other group or organisation.* Members of the European Parliament who attended this meeting took note of this suggestion and agreed to support the preparation of a new report by the ECRR which would address the issue of the health effects of radiation exposure and could provide an alternative analysis to the one which underpins present legislation.

It was a widely held view, both at the initial meeting of the ECRR and at the STOA meeting, that enough evidence was available then showing that low-level exposure to man-made radioactive material caused ill health, and that the conventional models of the ICRP and other agencies, which used the same radiation risk models, entirely failed to predict these effects. A fresh approach to the problem was thus necessary, and in 2001 various members of the European Parliament together with two charitable Trusts supported the drafting of the 2003 report.

1.2 *Developments since 2003*

The presentation in Berlin in 2003 of the first ECRR recommendations *(ECRR2003: the 2003 Recommendations of the European Committee on*

Radiation Risk. The health effects of radiation exposure at low doses for radiation protection purposes) represented a watershed in the perception of the hazards of exposures to ionizing radiation. The ECRR published the new pragmatic risk model for calculating the effects of exposure to ionizing radiation. The application of this model, which was based on epidemiological data and scientific reasoning using historic absorbed dose data and known physico-chemical behaviour of elements, gave results which explained and predicted observations of exposed populations. It received significant attention. The report was reprinted three times and has been translated into Japanese, Russian, French and Spanish. A Czech edition is being prepared. It was addressed by the UK National Radiological Protection Board (NRPB), which dismissed it. At the same time the UK Environment Minister Michael Meacher founded an official government committee CERRIE to discuss the implications of the arguments and the evidence which supported them (CERRIE 2004, 2004a). These arguments were also addressed over the two years following the publication of ECRR2003 by the French IRSN which put a team of scientists to review the model. The resulting IRSN report (IRSN2005) concluded that the concerns of the ECRR regarding the scientific basis of the current ICRP model (and all similar models) *were well-founded*, although IRSN took issue with the scientific basis of the model itself. It was unlikely that ECRR's arguments would be accepted universally: this was and is a political issue, a matter which is discussed briefly in the present report.

In the period since 2003 and the CERRIE Committee, the radiation-risk landscape has altered totally. When the ECRR began, questions about internal exposures and their anisotropy of effect at the cellular target, the DNA, were largely new, or at least had been avoided by the ICRP. The epidemiological basis of the risk model at the time was solidly that of external exposures at high doses: the Japanese A-Bomb survivors study and its interpretation in ICRP1990. Since then, the health effects of the Chernobyl accident have become all too apparent, although these data seem to have been ignored by ICRP and the UNSCEAR which have shrilly continued to categorize such alarming reports as 'radiophobia'. Nevertheless, radiophobia cannot affect generations of bank voles, wheat plants, and other life forms whose genetic developments were described by eminent research scientists contributing to ECRR2006 and ECRR2009.

The results of real data on Chernobyl affected territories (both in the ex-Soviet Union countries and in European countries) bore out the predictions of the ECRR2003 model. Since then there have also been reports of anomalous effects of exposure to the element Uranium, in molecular and in particulate forms, as it exists in the fallout from the use of Uranium weapons, so-called Depleted Uranium. This has led to significant effort into research on the effects of internal exposures to Uranium. The questions raised by this research are also those posed by ECRR in 1997 and which have formed the basis of the ECRR2003 model, the development of weighting factors for internal exposures

to certain isotopes based on their chemical affinity for DNA and their mode of decay.

In 2004, Dr Okeanov of the Belarus cancer registry visited Switzerland and presented data on increased incidence rates which were in line with those predicted by ECRR2003. Also in 2004, a study of cancer in northern Sweden showed that there was a statistically significant 11% increase per 100kBq m^{-2} Caesium-137 contamination the 5 years following the Chernobyl fallout (Tondel *et al* 2004). This can be shown to demonstrate a 600-fold error in the ICRP model, and supports the evidence given in ECRR2003 that the weapons test fallout had a similar effect with a similar error factor. The data from Belarus and the findings in Sweden 2004 could therefore be seen as a confirmation of the new model.

In 2007, the latest of a long series of childhood leukemia studies was published: this one from the German Childhood Cancer Registry, showing a statistically significant effect on child cancer in those living within 5km of nuclear plants (KiKK 2007). The size of this study, and the affiliation of the authors, made it impossible to conclude that this was anything but proof of a causal relationship between childhood cancer and nuclear plant exposures to radioactive releases. This study thus added to those highlighted in ECRR2003 which collectively put the error in the ICRP model as about 500 to 1000-fold.

In 2009, in an update of the study reported in ECRR2003, a meta-analysis of data on the epidemiology of infant leukemia after Chernobyl, showed a statistically significant 43% excess in those children who were *in utero* at the time of the Chernobyl fallout: the error that this highlighted in comparing external and internal exposures was a 600-fold error (Busby 2009)

None of these issues were incorporated into the 2007 ICRP report which ignored all the evidence and cited a selection of research papers which supported its own model. The ICRP took its evidence from UNSCEAR 2006 which in turn failed to cite any evidence that showed that the ICRP risk model was falsified by data.

Further, it has been increasingly clear that the internal exposures to fission product fallout and to Uranium from atmospheric weapons tests has been the principle cause of the current cancer epidemic, a matter which was presented in ECRR2003. Legal cases and test veteran tribunals are now routinely won on the basis of ECRR2003 and its arguments (e.g. Dyson 2009) Government agencies increasingly employ the model to scope the outcomes of new practice, placing the outdated ICRP model at one extreme and the ECRR model at the other.

The embarrassment of the ICRP came to a head with the matter of Uranium photoelectron enhancement, a new development which is discussed in the present report. This idea, which considers the absorbing medium and its atomic variability, rather than assuming uniform tissue-equivalent material, shows Uranium to be hundreds of times more dangerous that is currently modelled by ICRP due to its high atomic number. ICRP and other satellite

agencies have been unable to respond credibly to this development yet nothing has changed and Uranium exposures continue to be sanctioned. Over the period many studies of epigenetic effects, such as bystander signalling and genomic instability have continued to falsify the scientific basis of the ICRP model, the clonal expansion theory of cancer. The model is now bankrupt.

In early 2009, the Scientific Secretary of ICRP, and editor of both its 1990 and 2007 reports, Dr Jack Valentin, resigned. At an open discussion in Stockholm between him and Prof Chris Busby of ECRR on April 21st 2009 he stated that the ICRP risk model could not be employed to predict or explain the health effects of exposures to human populations. This was, he continued, because the uncertainties for internal exposures were too great, a matter in some cases of two orders of magnitude. This has been the contention of ECRR since its formation, and is written down in ECRR2003. Valentin also stated (in this video interview) that *since he was no longer employed by ICRP he could say that he thought it was wrong for ICRP and UNSCEAR to ignore the Chernobyl and other effects raised by the literature reports and by ECRR.*.

In May 2009, ECRR held an international conference in Greece, Lesvos Island, attended by physicians and radiation specialists from eight countries. At this conference, the ECRR2003 risk model and its development were intensively discussed, including new evidence which has emerged since 2003, as well as incorporation of the phenomenon of photoelectron enhancement by elements of high atomic number and with a discussion of the effects of Uranium exposure. A concluding statement, the *Lesvos Declaration*, was formulated (see Appendix). The statement called for the urgent abandonment of the ICRP risk model by governments and, as an interim measure, the adoption of the ECRR2003 model. This model is updated here in 2010 with addition of new evidence which has emerged since 2003, and the incorporation of the phenomenon of photoelectron enhancement by elements of high atomic number, and with a discussion of the effects of Uranium exposure.

Since it is clear to the Committee that political and lobbying opposition to the adoption of new rules which have massive political, economic, military and legal implications is likely to be (and has been) significant, the area of the science-policy interface requires discussion. New approaches must be developed with a view to obtaining secure policy from scientific advice. Such a discussion has been added to Chapter 3. This is extremely relevant to the event which founded the ECRR. Although the Greens were unable to significantly affect the Basic Safety Standards Directive 96/29, they were able to amend it so that Article 6.2 required that:

Member States must review Justifications of all classes of practice involving exposures ***if new and important evidence emerges****.*

Such is now clearly the case on both epidemiological and theoretical grounds.

2
Basis and Scope of the 2010 Report

2.1 Objectivity

For reasons based on the principles outlined in the previous chapter, the Committee takes the view that its analysis should be based on all available information. The Committee believes that in the search for scientific objectivity it should 'look out of the window', rather than following the trend of increasing dependence on desktop mathematical modelling. Thus the Committee has considered the results of studies published in the peer-review literature and also reports, books and articles which have not been submitted for peer review. The Committee believes that the approach adopted by scientific risk committees of only accommodating evidence with accurate dose-response data published in peer-review scientific journals has resulted in the propagation of a model which is increasingly seen to be unsafe (Carson 1962, Bertell 1986, Nussbaum and Koehnlein 1994, Busby 1995, 2006, 2009, Sawada 2007). Furthermore, the Committee believes that discussions in the area of radiation risk must involve all groups in society. Therefore, although primarily consisting of scientists, the Committee and its advisors include those physicians and specialists who must deal with medical problems of exposed persons. For example, risk assessment should include physicians trained in public health, occupational health, oncology, pediatrics, and scientists trained in genetics, epidemiology and biochemistry. These disciplines are not represented in the Main Committee of the ICRP. The regulations on membership as posted by ICRP includes: physicists, medical regulators, radiologists, biophysicists, etc. Among those included as advisors to the ECRR are specialists such as ecologists, zoologists, botanists, risk sociologists, lawyers, politicians and members of non-governmental organizations and pressure groups.

2.2 Basis of the report

The present report, like the 2003 report, is intended to be accessible to and to inform decision makers who need to assess health risks to workers and members of the public who may be exposed as a result of practices which involve ionising radiation. The basis of the report is a perceived failure of the present radiation-risk model (referred to in this report as the ICRP model) to explain or predict real increases in ill health in a large number of groups exposed to ionising radiation at low doses and low dose rates. Most of the examples where this has occurred will be referred to in the body of the report but the position of the Committee has also been affected by much that cannot be included, for reasons of space.

This includes reports which have been published in the peer-review literature, and reports which have not, or which started life as television documentaries and ended as court cases. It includes consideration of those who

voted with their feet and left areas where there were nuclear sites, regions which slowly became wastelands where only the poorest people would live and where the beaches were deserted by holidaymakers and fish were increasingly difficult either to catch or sell. It includes the stories of ordinary people who have been affected by man-made radioactivity, in India, Namibia, Kazakhstan, Nevada, Australia, Belarus and the Pacific Islands. It includes the massive literature, both peer-reviewed and so-called grey literature, that surrounds the phenomena of exposure to Uranium weapons, from the Atomic Bomb test veterans to the populations of Iraq and the Balkans and veterans of those Uranium wars.

2.3 Scope of the report

The report will critically review the present methodology for assessing radiation risk. It will argue that its dependence on averaging, in the area of energy deposition in tissue in space as well as in time, and also its dependence on epidemiological studies involving external exposure, has resulted in major errors in its quantification of risk from internal irradiation. It is intended that the report should convey sufficient evidence that the present radiological safety models are largely accurate for external irradiation situations involving doses greater than 100 mSv so long as the exposures are well defined and uniform, but break down where calculations involving averaging methods are used to examine non-uniform doses in microscopic tissue volumes. It is the microscopic distribution of ionizing events in tissue, from the point of view both of the external field and of the medium of absorption, which is the critical factor in radiobiological damage and this has not been modelled by the physics-based ICRP model which largely ignores molecular interactions, dealing rather with average energy transfer.

The report will examine the historical origin of the ICRP model and will review epidemiological evidence for its successes and failures. The report will consider the philosophical and methodological aspects of the science of radiation risk and make a distinction between the inductive and deductive approaches to establishing objective risk estimates. It will discuss the current science-policy interface and the opportunity for (and evidence of) bias in the translation of scientific (experimental) knowledge into changes in policy. It will present evidence for quantitative ranges of error in the ICRP models as highlighted by various authors and studies and will assemble these into a set of hazard enhancement weighting factors which form the basis of a pragmatic approach to the problem of assessing radiation risk using the present units and quantities. It will extend radiological protection to non-cancer illnesses, lens destruction, neurological illnesses, diabetes, immunologies and several other radiogenic illnesses and will now specifically include a risk factor for heart disease.

Finally, the report will briefly outline some examples of the application of such a system for assessing radiation risk. A calculation of the mortality yield of the post-war nuclear age based upon ICRP and modified ICRP risk factors will also be presented. The approach is necessarily pragmatic. Data on radiation exposures and activities has historically been tabulated and recorded using units of *absorbed dose* devised from within the ICRP system: it is therefore necessary to provide factors which may be used with this system and this is what the Committee has striven to achieve. These factors are provided as central estimates of hazard-enhancement for certain types of exposure and may be used as multipliers of risk for the risk factors presently used by ICRP. However, the Committee believes that the use of the average energy dose units Gray and Sievert places too many constraints on the science of risk assessment for internal isotopes and that a different, more rational system of assessing such exposures is required. Some suggestions were made towards achieving such a system were made at the 2009 ECRR conference in Lesvos, Greece, but the consensus was that major difficulties existed in developing such a system and that the bases of such a system were best employed in developing semi-empirical weighting factors for the current system of absorbed doses.

2.4 References

In ECRR2003 the Committee carefully considered the question of whether the editors should attempt to reference every statement made in this Regulators' Edition. On the one hand, the ICRP, whose handbook ICRP90 the ECRR2003 volume was intended to supplant, provided no references. On the other hand, the more lengthy reviews of the United Nations (UNSCEAR) and the US Academy of Sciences (BEIR) carry selected references which support their statements whilst failing to cite work which either falsified or did not support their statements. The new 2007 ICRP Publication 103 contains 286 references. However, as the analysis in Chapter 5 shows, 90 of these are to non-peer-reviewed reports by ICRP itself, whilst only 120 are in peer-reviewed journals and these are reports mostly written by individuals associated with the risk organizations themselves. There are no references to any effects of Chernobyl or to childhood leukemia clusters near nuclear sites or to Uranium effects.

In ECRR 2003 the Committee considered the constraints that would be placed on the size of the edition if all statements were fully referenced, and the loss of flow of the argument which would follow the considerable expansion of the text. As a compromise, it decided to attach a list of the main works on which its beliefs are founded, without attaching each to some piece of the text. There was some criticism of the 2003 report on the matter of references and so in this 2010 report many references are now linked to text where it is felt that such a link would be valuable to the reader.

3
Scientific Principles

Since a wise man may be wrong, or a hundred men, or several Nations, and since even human nature, as we know it, goes wrong for several centuries on this matter or on that, how can we be certain that it occasionally stops going wrong and that in this century it is not mistaken?

Montaigne 1533-92, *The Essays*

3.1 Radiation Risk and Scientific Method

The Committee believes that it is instructive to examine the scientific basis of the method which has been historically developed to create the radiation risk models.

The classical exposition of the scientific, or inductive method (originally due to William of Occam) is what is now called Mill's Canons, the two most important of which are:

- The Canon of Agreement, which states that whatever there is in common between the antecedent conditions of a phenomenon can be supposed to be the cause, or related to the cause, of the phenomenon.
- The Canon of Difference, which states that the difference in the conditions under which an effect occurs and those under which it does not must be the cause or related to the cause of that effect.
- In addition, the method relies upon the Principle of Accumulation, which states that scientific knowledge grows additively by the discovery of independent laws, and the Principle of Instance Confirmation, that the degree of belief in the truth of a law is proportional to the number of favourable instances of the law.

Finally, to the methods of inductive reasoning we should add considerations of *plausibility of mechanism.*

These are the basic methods of science (Mill, 1879; Harre, 1985; Papineau, 1996)

The questions of interest here are:

- What are the health consequences of exposure to external radiation doses at levels below 2mSv, the approximate annual dose received from natural background?
- What are the health consequences of exposure to novel internal radioisotope exposures at whole organism and individual organ dose levels below 2mSv?
- Is the concept of dose applicable to internal radiation exposures?

Although risks from exposure to high levels of ionising radiation are generally accepted, since they are fairly immediate and visible, the situation with regard to low-level exposure is curious. There are now two mutually exclusive models describing the health consequences of such exposure. There

is the ICRP one, based on reductionist physics-based arguments and which is presently used to set legislation on exposure limits and argue that low-level radiation is safe, and one which is espoused by concerned independent public domain organisations and their associated scientists. These two models are shown schematically in Fig 3.1.

They arise from two different scientific methods. The conventional model is a physics-based one, developed by physicists prior to the discovery of DNA. Like all such models it is mathematical, reductionist and simplistic, and consequently has a powerful descriptive utility. Its quantities, dose, are average energy per unit mass or dE/dM and in its application, the masses used are greater than 1kg. Thus it would not distinguish between the average energy transferred to a man warming himself in front of a fire and a man eating a red hot coal. In its application to the problem at hand, the internal, low-level, isotopic or particulate exposure, it has been used entirely deductively. The basis of this application is that the cancer and leukemia yield per dose has been determined following the external acute high-dose irradiation by gamma rays of a large number of Japanese inhabitants of the towns of Hiroshima and Nagasaki. Together with this, other arguments based on averaging have been used to maintain that there is a simple linear relationship (in the low-dose region) between dose and cancer yield. This Linear No Threshold (LNT) assumption enables easy calculations to be made of the cancer yield of any given external irradiation.

By comparison, the mechanistic/epidemiological model shown at the bottom of Fig. 3.1 arises from an inductive process. There have been many observations of anomalously high levels of cancer and leukemia in populations living near nuclear sites, especially those where the measurements show that there is contamination from man-made radioisotopes, e.g. reprocessing plants. In addition there are populations who have been exposed to man-made radioisotopes from global weapons tests, downwinders living near nuclear weapon test sites, and those exposed to these materials because of accidents (like the Chernobyl infant leukemia cohort), or because of work in the nuclear industry or military. More recently, research has addressed those exposed to the fallout from the use of Uranium weapons: these have shown a wide range of genetic and neurological effects. A review of these findings is given later in this report. In contrast to the averaging approach of the conventional model, the biological model preferred by the ECRR considers each type of exposure according to its cellular radiation track structure in space and in time. Since ECRR2003 the effect of the absorbing element in the body has also become important. It is not easily possible to employ such a model to predict risks from unspecified ‘radiation dose’ to ‘populations’; rather it is concerned with microscopically described doses from specific isotopes or particles whose decay fractionations are considered to interact with cells which themselves respond biologically and biochemically to the insults and may be in various

stages of their biological development. The dose-response relationship following from this kind of analysis might be expected to be quite complex.

In examining radiation risk, the Committee finds that these philosophical models are mutually exclusive and has to decide which one is correct. In making such a decision the Committee has employed the basic rules of scientific method.

The Committee believes that the Linear No Threshold (LNT) model is fundamentally acceptable (with some reservations) in its application ***to acute, high dose, external irradiation,*** although it notes that the ICRP, UNSCEAR and BEIR Committees introduce a reduction of the modelled risk by a factor of 2 for low-dose-rate exposure, which breaks the assumption of linearity. The Committee believes that the extension of LNT to acute, external, low level radiation may be justified on the basis of theory, since the plausibility of the model rests on the idea of uniform density of radiation track events in microscopic tissue volumes. For ***chronic external irradiation***, the Committee does not believe that the scientific method has been properly used to show that there is either epidemiological or theoretical justification for assuming a linear response at low doses. This is because the complex ways in which the organism responds to low-dose radiation both at the cell and at the organism level have been overlooked. However, the Committee believes that the errors introduced by the assumption are unlikely to be more than an order of magnitude.

The Committee is also concerned that the assumption of linearity of dose response is used to inform epidemiological studies of trend. A number of epidemiological studies have shown decreasing health effects at the highest doses and this finding has been used to suggest that radiation exposure cannot be responsible for the effects studied, although several plausible reasons for such a result (e.g. high-dose cell killing) may exist. The range of error for external irradiation effects and the mechanisms involved will be addressed in Chapter 9.

With regard to ***internal radiation doses***, the Committee identifies a serious misuse of scientific method in the extension and application of the ICRP external model. Such a process involves deductive reasoning. It falsely uses data from one set of conditions— high-level, acute, external exposure— to model low-level, chronic, internal exposure. The procedure is scientifically bankrupt, and were it not for political considerations, would have been rejected long ago. On the other hand, it should be clear that the radical model shown in Fig 3.1, suggesting high risk, conforms to all the requirements of the scientific method listed at the beginning of this chapter. Man-made radioisotopes, often in the form of 'hot particles', are common contaminants of the areas near nuclear sites where there are cancer and leukemia clusters, and of nuclear site and test site downwinders, and of fallout-exposed populations. This satisfies the *Canon of Agreement*. The contingency analysis tables with control populations for such studies show that the *Canon of Difference* is also satisfied: people living in more remote regions than the downwinders show lower levels

of illness. The *Principle of Instance Confirmation* is fulfilled since so many studies have shown that increases in cancer and leukemia follow exposure regimes at low dose. We are left only with *Plausibility of Mechanism*, which will be addressed later in this report.

The Committee's position on the scientific applicability of the ICRP model to the yield of fatal cancer in a range of exposure types is outlined in Table 3.1.

It is important to note that science and scientific conclusions are not the same as conclusions based on legal styles of evidential analysis. Science is not a simple question of weighing evidence for and against a theory or model of reality as it might be in a court of law or in everyday decision-making. The rules are strict. If one single piece of experimental evidence cannot be explained or incorporated into a theory, the theory has to be discarded (Kuhn 1962, Popper 1962). Therefore the existence of the nuclear site child leukemia clusters alone is enough to falsify (to prove wrong) the ICRP risk model; yet nothing has been done despite these data emerging in the 1980s. The Committee feels that it may be illuminating to ask how such a state of affairs, once set up in ignorance, becomes crystallised and difficult to challenge, even when large numbers of sick and dying draw attention to the existence of an insecure model. The conservative nature of science and its systems was considered in the late 1950s by an eminent and past member of the British Royal Society, the Nobel-Prize winner, chemist and economist Michael Polanyi.

Table 3.1 Errors associated with ICRP extension of acute high dose external studies to other types of exposure

Type of exposure	**Is ICRP model applicable?**	**Uncertainty in error factor for fatal cancer identified by ECRR**
External acute >100mSv	Yes	0.5 to 25
External <100mSv	Very approximately but problems with cell and organism responses.	1 to 50
Internal <100mSv	No	1 to 2000
Internal High Z elements	No	1 to 2000

Polanyi, was interested in the scientific method, and in scientists: his writings pre-dated the Science War philosophers like Kuhn and Latour. He was aware that at any time, the scientific world view might be completely wrong. In

asking how we know anything at all and how we build up a picture of the 'real world' Polanyi saw many similarities between scientists and primitive witch-doctors like the Azande who had been studied in the 1930s by the anthropologist Evans Pritchard who wrote:

They reason excellently in the idiom of their beliefs, but they cannot reason outside, or against their beliefs, because they have no other idiom in which to express their thoughts. The contradiction between experience and one mystical notion is explained by reference to other mystical notions.

E. Evans Pritchard, *Witchcraft, Oracles and Magic among the Azande*, 1937

Addressing the supposedly scientific world view, Polanyi concluded:

[For] the stability of the naturalistic system we currently accept, instead, rests on the same logical structure as Azande witchcraft beliefs. Any contradiction between a particular scientific notion and the facts of experience will be explained by other scientific notions. There is a ready reserve of possible scientific hypotheses available to explain any conceivable event. Secured by its circularity and defended by its epicyclical reserves science may deny or at least cast aside as of no scientific interest, whole ranges of experience which to the unscientific mind appear both massive and vital.

M. Polanyi FRS, *Personal Knowledge*, 1958

The Committee has concluded that the ICRP scientists and risk models are good examples of such systems of closed scientific communities and epicyclical logic. Polanyi's comparisons with Azande witch-doctors are familiar territory to those who have registered the sequences of denials and implausible explanations which have followed discovery of the Sellafield (Seascale) child leukemia cluster and many other examples of the failure of the ICRP risk models. In the following chapter we examine the origin of the ICRP risk model and see how it has become the deductively based interpretative machine that rejects automatically and epicyclically any experience which to ordinary people seem both massive and vital.

3.2 The Science Policy Interface and Bias: CERRIE

The Azande problem is what psychologists term *group-think.* (Janis and Mann 1977). It is not restricted to the ICRP and those supporting the ICRP approach e.g. UNSCEAR, NCRP, and BEIR. Since the UK Mad Cow Disease episode in the 1990s it has become increasingly clear that scientific advice on policy can be seriously biased in this way. In the Mad Cow episode, a scientific committee advised the government (against outside scientific advice and experimental evidence) that the agent could not cross the species barrier. They were wrong

and many members of the public have died because of their error. Interestingly, the Chair of the Committee which gave that wrong advice was at the same time the Chair of the UK National Radiological Protection Board, Sir Richard Southwood.

The Policy Information Network on Child Health and Environment, PINCHE, an EU-funded network of 30 or more eminent doctors and scientists from several countries of Europe, spent 4 years discussing this issue of the Science Policy Interface to produce a report which was commissioned by the EU. The PINCHE concluded that science advice from selected committees was regularly biased by the selection process of references to support the conclusions. These conclusions were generally biased by the affiliations of the committee members and secretariat and tended to support, in economic health as opposed to human health, those decisions which resulted in least damage to their affiliated institutions or industries (Van den Hazel *et al* 2006). The solution to this, PINCHE agreed, was to found oppositional committees where scientists were funded to support both sides of any argument which informed on environmental risk, an idea which was suggested by Scott Cato *et al* 2000. The *groupthink* concept is now widely accepted: the US Pentagon trains a *skeptics corps* to battle decisions and planning errors made through *groupthink*, a process called *Red Teaming.*

The Committee on Radiation Risk from Internal Emitters CERRIE was set up by the UK Environment Minister Michael Meacher in 2001 along just these lines. Its remit was to discuss the evidence for the failure of the ICRP model for internal emitters and present both evidence which supported and opposed such a belief. In the event, this process failed when the Minister was removed in 2003 before the final report was published and a new Environment Minister, Elliot Morley, was appointed by Tony Blair. Morley shut down the Committee before it could carry out the key research which had been agreed to decide the issue and legal threats were used to prevent the oppositional report being included (see endnote Morley 2010). The minority oppositional report (which was excluded by the legal treats) was separately published in 2004 (CERRIE 2004b).

3.3 The scientific basis of ICRP2007

Naturally, bias extends to ICRP 2007 which cites the main CERRIE report but not the oppositional report. With the results of PINCHE on biased selection of references in mind it is quite informative to examine the reference base for ICRP2007. There are 286 references: their general description is shown in Table 3.2.

Table 3.2 Distribution of references in ICRP2007

Number of references	Organisation being referenced	Peer reviewed
91	ICRP/ ICRU/IAEA	No
21	UNSCEAR/NCRP	No
52	Books and reports	No
103	Peer-reviewed journals	Yes
20	ICRP member's paper	Yes

Of the 123 references to peer-reviewed literature many of them are to personnel associated with the risk agencies in some way or other publishing in 'house journals' like the *Journal of Radiological Protection*, whose editor is Richard Wakeford, until recently Chief Scientist for British Nuclear Fuels. There are references to some bizarre journals like the *Central European Journal of Occupational and Environmental Medicine.* This latter reference is to work by a member of the ICRP Committee, A. Akleyev. One reference is to *The inheritance of pyloric stenosis* by *Carter C.O* published in the *British Medical Bulletin* in 1961. How can this be more valuable or relevant to radiological protection than the many references to Chernobyl effects available to ICRP or provided in the ICRP 2007 internet consultation by ECRR scientists?

The introduction to ICRP 2007 states: *We could not have done it without your help!* referring to an internet dialogue where ICRP canvassed comments on the draft recommendations. Many of the ECRR scientists communicated within this ICRP 'consultation' process, and their communications and references can still be read on the ICRP website. However, none of these suggestions or references made it into the final edition. Indeed there was one important and relevant paragraph which was in the 2005 ICRP draft on the internet.

It stated:

(50) For radiations emitted by radionuclides residing within the organ or tissue, so-called internal emitters, the absorbed dose distribution in the organ depends on the penetration and range of the radiations and the homogeneity of the activity distribution within the organs or tissues. The absorbed dose distribution for radionuclides emitting alpha particles, soft beta particles, low-energy photons, and Auger electrons may be highly heterogeneous. This heterogeneity is especially significant if radionuclides emitting low –range radiation are deposited in particular parts of organs or tissues, e.g. Plutonium on bone surface or Radon daughters in bronchial mucosa and epithelia. In such situations the organ-averaged absorbed dose may not be a good dose

quantity for estimating the stochastic damage. The applicability of the concept of average organ dose and effective dose may, therefore, need to be examined critically in such cases and sometimes empirical and pragmatic procedures must be applied.

But ICRP did nothing to change any of the dose coefficients for isotopes that caused such exposures or to apply such *empirical and pragmatic procedures.* and the embarrassing paragraph above was quietly dropped from the final ICRP 2007 report.

This brief review of the 2007 ICRP report demonstrates that there has essentially been no change in the model from that which was published in 1990, and that new evidence and arguments which scientifically falsify that model have been totally ignored. The ICRP continues to support the same risk factors for exposures to ionizing radiation and its model is still the basis for limits to releases to the environment. The ICRP 2007 model does not discuss the evidence: it is selective and partial and clearly does not conform to the philosophical requirements of science outlined in this chapter. As the Lesvos Declaration in the appendix demands, it must now be abandoned.

3.3 Peer review, research funding, and the Scientific Consensus

In responding to concerns about the clear failures of the ICRP model to predict or explain observations, politicians and regulators commonly refer to the concept of a *scientific consensus*. The Committee has made it very clear in this chapter and elsewhere that the political understanding of science as some kind of process which goes forward through the mechanisms of research, peer review of reports and acceptance of change by a 'scientific community' which represents a scientific consensus on any matter is dangerously inaccurate. One of the reasons for this which has not been discussed is the control of peer-review publication of research reports. In this model of science, before any research is believed to be 'scientific' it has to be published in a learned journal and subjected to peer-review. The reviewers are anonymous and may dismiss the contribution: in which case the editor usually rejects it. Thus the evidence is invisible from science and cannot become part of the *scientific consensus*. The problem is, of course, that reviewers will generally dismiss any research which contradicts their own beliefs; and if they do not, often the editor will. This process has kept many important results from being presented and therefore being incorporated into any scientific consensus. A good example of this bias is afforded by the *Journal of Radiological Protection* which appeared in the 1970s, in which those who believe in the ICRP system publish. Their papers are reviewed by each other, and therefore regularly appear: thus giving their beliefs and their model some spurious credibility; naturally, any contributions which disagree are sent out to referees and dismissed. The current editor of this journal, Richard Wakeford, of British Nuclear Fuels, Sellafield, is a champion

of the adequacy of the present approach. The editorial board reads like a list of those who work for the nuclear regulatory bodies, ICRP. UNSCEAR, IAEA and so on worldwide with a few nuclear energy workers thrown in. We see Jack Valentin of ICRP on this board. This process represents one way in which the evidence that the ICRP model is incorrect is excluded from 'science'. In a most recent example, the UK Health Protection Agency (Radiological Protection) withdrew from a stakeholder dialogue involving ECRR on the Secondary Photoelectron Amplification Effect (described below). The reason was that HPA's initial mathematical treatment was clearly absurd and contained many basic errors. When these were pointed out, the response was that there would be no further discussion on the issue: a paper was to be published in the peer review literature. The paper, by the Deputy Director of HPA(RP) John Harrison, is apparently to appear in the *Journal of Radiological Protection* where it will have been reviewed by those with a bias in favour of accepting it.

And there is another way in which the scientific consensus is skewed. Researchers who do manage to get dissident results into the scientific media lose their research funding and often their jobs (see Viel 1998). Politicians and decision makers should be cautious therefore in dismissing research which is not published in the peer review system (so-called grey literature). This was the conclusion (for exactly these reasons) of the science–policy group in the EU funded PINCHE report conclusions (van den Hazel *et al* 2005).

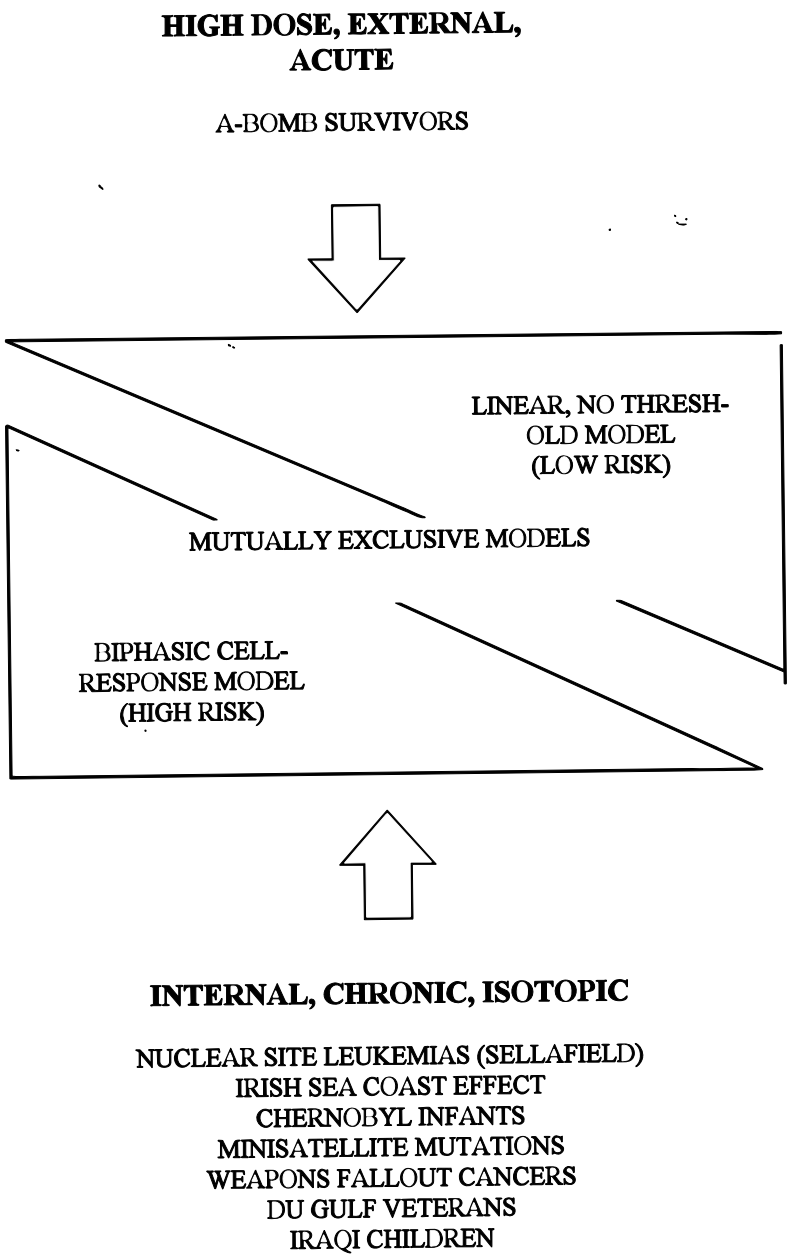

Fig. 3.1 Mutually exclusive models derive from induction versus deduction. The “high dose acute A-Bomb survivors” group had mean external doses of 200mSv but are strictly a “high dose rate” group.

4
Radiation risk and ethical principles

4.1 Problem to be addressed

The release of radioactive materials into the environment results in the contamination of living organisms. This internal radiation exposure and external radiation from the same radioactive materials in the environment cause damage to cells. Recent research into genomic instability and bystander signalling suggests that such exposure results in death or mutation in roughly a third of all somatic or genetic stem cells intercepted by a radiation track. One high-impact consequence of this is that a small proportion of the descendants of these irradiated cells may become cancerous and kill the individual. Other consequences are that the general loss of cells to the organism may result in both specific and general impairment of health. Third, these effects in germline cells are not restricted to the exposed individual and may be passed on to the next generation.

The question to be answered is: is it ethically acceptable to sanction the operation of an industry for which this circumstance is an inevitable outcome? Two other questions may be asked:

- First, is such a sanction a case of political decision making after consent has been given by an electorate, and if so, has it followed adequate debate and full access to accurate information?
- Second, is the answer to the ethical question subject to a *de minimis* threshold such that small amounts of harm may be sanctioned if the outcome is justifiable in terms of a greater good? This latter question seems to have been implicitly asked and answered, but as the Committee will argue (para. 4.4.7), the basis of the answer is philosophically questionable and should be revisited.

4.2 Human chauvinism

Before embarking on an exploration of the case to be made for and against radioactive emissions from the perspective of different ethical theories the Committee acknowledges that the major ethical theories presented here—particularly those of rights and utilitarianism—are anthropocentric. That is to say, they agree about the scope of moral decision making, and that it should include only one species: our own. Routley and Routley have challenged what they refer to as ‘the inevitability of human chauvinism’ in the following terms.

In our enlightened times, when most forms of chauvinism have been abandoned, at least in theory, by those who consider themselves progressive, Western ethics still appears to retain, at its very heart, a fundamental form of chauvinism, namely, human chauvinism. For both popular Western thought and most Western ethical theories assume that both value and morality can ultimately be reduced to matters of interest or concern to the class of humans
(Routley and Routley, 1979).

The drawing up of guidelines for exposure to ionising radiation as a result of the civilian nuclear power programme is a typical example of such human chauvinism. All the models are designed to determine doses to people, despite the obvious fact that all wild and most domestic animals spend more time outside, and are thus more subject to radiation, than most people.

The ethical problems with routine contamination of people by radionuclides presented in this chapter are fairly compelling in themselves, but a serious consideration of animal rights would suggest a vast inflation in the level of harm caused. The Committee welcomes the efforts that various agencies (e.g. IAEA 2002, ICRP 2002) have made in exploring various ethical approaches to protection of the environment as distinct from protection of people. The Committee does not set out to rehearse them, but notes that there is a general tendency to recognise that the environment has its own *moral standing* - i.e. towards recognising the validity of protecting the environment for its own sake, rather than for the sake of its human utility.

The positions thus taken may be far more rational than they at first seem to western minds. Major eastern philosophical/religious systems which are frequently cited (e.g. by IAEA 2002) as the sources of non-anthropocentric views of environmental protection hold to the law of *action, motive and result* - the idea that harm deliberately done inevitably returns to the perpetrator, almost always in a future life. The fact that this is seen as an obstacle to the preeminent goal of achieving enlightenment throws a fresh light on the supposed non-anthropocentrism of eastern attitudes to the environment. *Action, motive and result* also raises questions about the long-term interests of anyone engaged in radiation protection to the extent that they were deliberately to ignore relevant evidence. Ironically, hoping that those responsible might suffer as a result of the harm their actions caused would itself be a further obstacle to enlightenment.

In view of the difficulties of identifying and quantifying detriment to the environment at the low levels of dose commonly found and the consequent issue of whether such doses matter, it may be helpful to bear in mind an important insight from the debate between environmental ethicists. Mary Midgley (1983) identifies a problem commonly associated with certain environmentally and socially destructive processes; that although they may be greeted with general moral repugnance it is often difficult to substantiate the

objections to them. To illustrate her point she offers the following entry from the diary of Robinson Crusoe:

19 Sept. 1685. This day I set aside to devastate my island. My pinnace being now ready on the shore, and all things prepared for my departure, Friday's people also expecting me, and the wind blowing fresh away from my little harbour, I had a mind to see how all would burn. So then, setting sparks and powder craftily among certain dry spinneys which I had chosen, I soon had it ablaze, nor was there left, by the next dawn, any green stick among the ruins . .

(Midgley, 1983: 89).

Midgley identifies that it is the moral tradition of the (western) Enlightenment that has made our objections to such activity unstatable. In her words:

Today this intellectualist bias is often expressed by calling the insights of common morality mere 'intuitions'. This is quite misleading, since it gives the impression that they have been reached without thought, and that there is, by contrast, a scientific solution somewhere else to which they ought to bow—as there might be if we were contrasting common-sense 'intuitions' about the physical world with physics or astronomy.

(Midgley, 1983: 90).

Interestingly in view of our subject, she sees the model as drawn from atomic physics.

4.3 The Ethical Basis of the Civilian Nuclear Power Programme

4.3.1 Introduction

Para. 101 of the ICRP 1990 Recommendations represents the closest that the international nuclear community has come to providing an ethical basis for its activities. The paragraph states:

Most decisions about human activities are based on an implicit form of balancing benefits against costs and disadvantages, lead*ing to the conclusion that a particular course of action or practice either is, or is not, worthwhile. Less commonly, it is also recognised that the conduct of a practice should be adjusted to maximise the net benefit to the individual or society . . .When the benefits and detriments do not have the same distribution through the population, there is bound to be some inequity. Serious inequity can be avoided by the attention paid to the protection of individuals. It must also be recognised that many current practices give rise to doses that will be received in the future, sometimes the far future. These future doses should be taken into account in the protection of both populations and individuals.*

The ICRP, their satellite committees and those political decision makers that have come after them appear not to have addressed explicitly the philosophical

and ethical basis of their recommendations or indeed the moral justification for the health consequences that are an inevitable result of the radioactive emissions of civilian nuclear power programmes. However, para. 101, cited above, does identify implicitly the source of the ethical thinking of the Committee, and it appears to be planted firmly in the utilitarian tradition. The method of decision making that results from such a philosophical foundation is inevitably that of cost-benefit analysis. The members of the ICRP clearly assumed that such a moral position was universally accepted, and perhaps the only source of ethical guidance. This chapter, which outlines the position of the ECRR, takes a broader view, addressing the issue of the health consequences of nuclear power and other sources of radioactive contamination from the perspective of a variety of ethical theories, as well as providing a critique of the utilitarian position, especially as applied to nuclear power. It proceeds to address specific aspects of decision making that would need to be resolved for civilian nuclear power to have a firm ethical foundation.

Civilian nuclear power is an interesting case of policy making, since it appears to have never faced ethical or democratic scrutiny. Although this is not stated, it can only be deduced that such justification was felt unnecessary, due to the close link between the civilian and military nuclear industries and the origin of both in the period of the Cold War. In an era when it was believed that we would be better off dead than red, a few extra deaths as a result of nuclear processes may have seemed a small price to pay in exchange for our place at the big table of international diplomacy. Given the changed political situation an evaluation of the ethical foundation of nuclear power is long overdue.

4.3.2 The Health Consequences of Nuclear Power as Seen from Alternative Ethical Perspectives

Utilitarianism

Utilitarianism is well known as the moral philosophy that assesses the ethical rightness of an act or policy on the basis of its ability to maximise the total aggregated happiness of all the members of society. As one environmental ethicist expresses it, ‘utilitarians consider an action or a decision to have the moral quality of rightness to the extent that it leads to: . . . the maximisation of good consequences, conceived in terms of social welfare or utility, over the long run (Sagoff: 1988: 171). In other words, the central tenets of utilitarianism are that results are the key to a moral evaluation of actions and that to assess their moral rightness we should compare these results in terms of the happiness or unhappiness that they cause (Shaw, 1999).

The objective of this ethical position is the maximisation of total utility, or happiness. It is important to grasp that it has nothing to say about the distribution of that happiness (Shaw, 1999). In fact, one of the original criticisms of utilitarianism is that it would be quite consistent with a slave society. Its concern is to maximise well-being on average. This is interesting in

the context of a discussion of the ethics of nuclear pollution, where doses to the public are also considered on average, which leads to many of the inaccuracies of health-risk models identified elsewhere in this report. The policy mechanism whereby these 'average well-being' calculations are translated into policy, the cost-benefit analysis, thus has basic philosophical problems as well as the practical difficulties explored later in this chapter.

Utilitarianism has always had an immediate appeal, and especially to policy makers. Shaw (1999:2) considers that 'utilitarian goals have shaped public decision-making in the twentieth century'. An important explanation for the appeal of utilitarianism is its simplicity. It reduces hard moral cases to simple mathematical equations, which allows policy makers to believe both that they are in control of a desperately complicated situation, and also that they can come up with an answer that is easy to defend.

The downside of the utilitarian calculation is that it yields outcomes that are morally repugnant to many citizens (see Shaw, 1999). For example, most citizens believe it would be intolerably callous to allow premature babies to die, although the costs expended on such a small number of members of the population are immense. By any rational utilitarian calculation these costs would increase the sum of human happiness more if they were spent finding improved means of pain relief or a cure for cancer. Yet the importance that people place on individual moral worth in contrast to the sum of human happiness is illustrated by the public horror that was displayed when children died during surgery by incompetent doctors in Bristol. Although the absolute number of deaths was small by comparison with the total number of heart operations carried out annually, the moral outrage was vast. Thus citizens appear to agree with the conclusion of Anne Maclean, who in a discussion of the field of bioethics, claims that 'pure utilitarianism eliminates the essential ingredients of moral thinking' (1993).

Perusal of government documents makes it clear that considerations of average well-being do tend to take precedence over individual rights. For example, a recent report into the deleterious health consequences of living near landfill sites was played down by its authors on the basis that the number of children actually born with the defects that had been shown to be related to proximity to a landfill was small. While this follows the logic of the utilitarian calculus it is unacceptable to our moral sentiments, so that the cluster of congenital malformations near the Nant-y-Gwyddon tip in South Wales caused a national outcry.

Utilitarianism allows the death from leukemia of children who live near nuclear sites to be balanced by the societal gain from the energy source, or the Plutonium for weapons to defend the country; it can balance the warmth delivered by electric fires in millions of homes against breast cancer of those women living downwind of the nuclear power station. It may have an appeal to the policy maker, but it does not follow the moral sentiments of the citizen.

This may be part of the explanation for the growing gulf of trust between politicians and the citizens they are elected to represent.

Rights-based theories

It appears that, implicitly or explicitly, utilitarianism has ruled the ethical roost and provided the philosophical basis for policy making for over a century in the UK and elsewhere. Its popularity in the United States has been undermined by the growth in popularity of a new ethical system based on the concept of rights. If utilitarianism may be characterised as making the right subservient to the good, then rights-based theories may, by contrast, be considered to hold that the good is always subservient to the right. This has far-reaching implications for policy making in general and for the civilian nuclear power programme in particular.

The starting-point for such theories is a rejection of the averaging principle of utilitarianism, which would sacrifice the well-being of any given individual for the sake of the greater good of the whole community. Rights-based theories argue that each human being has inviolable rights as an individual and that the state may only override these with the express permission of the individual.

Ronald Dworkin, who offers a strong legal defence of rights, argues for their fundamental importance in *Taking Rights Seriously* (1977): 'the invasion of a relatively important right must be a very serious matter. It means treating a man [*sic*] as less than a man'. In terms of the conflict between utilitarianism and rights-based moral theories he argues that the state 'must not define citizens' rights so that these are cut off for supposed reasons of the general good'.

So, how might we apply rights-based ethical theories to the activities of the nuclear industry? While debate continues about the level of harmfulness of emissions, it is accepted by all sides that the production of energy from nuclear sources will create a fixed amount of radioactive pollutants which will be released to the environment and will inevitably contaminate the bodies of those who live in that environment. Such activity, carried out without the full knowledge of the citizenry, and certainly without their informed consent, represents an infringement of the most fundamental natural right: the right to the inviolability of the body. This right is considered basic in rights theory and is used to justify, for example, the use of violence in self-defence if one's body is under attack.

We may find a more specific statement of the individual's right not to be contaminated in the UN Declaration of Human Rights where Article 3 states: 'Everyone has the right to life, liberty and the security of the person'. Although it has yet to be tested in court, there seems a strong *prima facie* case that contamination of citizens' bodies with nuclear waste represents an unacceptable threat to the security of the person, and is therefore illegal under international law. From a rights perspective, in order for the nuclear industry to

continue to operate legally, all those who might potentially be contaminated would have to be accurately informed about the true risks to their health from such nuclear processes, and would have to agree that the processes should continue.

Rawls's Theory of Justice

An influential contribution to moral and political philosophy was made by John Rawls, with the publication of his *A Theory of Justice* in 1971. While this is not a rights theory as such, Rawls is often discussed in connection with such theories since his aim was to determine principles of justice that would ensure ethically justifiable distributions. His concern was primarily the distribution of wealth but we may extend his theory to consider the distribution of 'illth' associated with nuclear processes. Rawls's central intellectual tool is the 'veil of ignorance': he suggests that a distribution is fair if a citizen would choose it from a range of alternatives without knowing which position in the distribution she or he would find her- or himself in. Hence the theory stands in contradiction to utilitarianism which only maximises total welfare and hence could easily admit a small number of very unpleasant situations so long as they were balanced by pleasant ones. In Rawls's system, by contrast, an individual would protect her- or himself against the worst possible outcome. In such a moral universe the question facing the citizen would be, should the nuclear industry be allowed to continue emitting radioactive waste which will cause a small number of deaths. The citizen would be behind the 'veil of ignorance' and hence would not know whether it would be his or her child or grandchild that might be the one to develop leukemia. The chance would be small, but would it still be a situation they would potentially accept?

For Rawls, such questions are, in any case, of second order. The overriding commitment of his moral theory, as with those discussed in the previous section, is with the absolute right of the person. As he expresses this point:

Each person possesses an inviolability founded on justice that even the welfare of society as a whole cannot override. (Rawls, 1971: 3).

This 'inviolability' may be considered to include bodily inviolability, hence the contamination of citizens with radioactive emissions without their knowledge or consent would not be possible within a just state, no matter how much the process that produced the emissions benefited society as a whole. Since the citizens of modern nations have never given their consent to the contamination of their bodies by the routine emissions of nuclear wastes (and are unlikely even to be aware that such a process occurs on a daily basis) such emissions are, according to rights-based theories, simply immoral.

Virtue ethics

The strand of moral philosophy identified as virtue ethics provides an alternative view of how we might judge behaviour to be ethical. Rather than being based on a technique involving measurement and calculation, or on a claim to the fundamental inviolability of rights, it proposes instead that ethically sound behaviour is behaviour that could be considered virtuous. Theorists of this school may at first appear vulnerable to the suggestion that they are not actually providing useful guidance, since there can be no objective agreement about which types of behaviour are virtuous. However, a little consideration makes it clear that in fact such subjectivity problems afflict the other theories also. For example, utilitarianism rests on a no less subjective judgment about what 'happiness' or 'utility' may be. And similarly there can be no absolute agreement about which rights are fundamental and inviolable when two rights come into conflict. Virtue ethics, by contrast, makes no claim to objectivity. According to Rosalind Hursthouse (1999), ethics cannot be given a foundation from a neutral point of view; rather, we all have an acquired and subjective ethical outlook.

This is a philosophical position with little appeal for policy-makers, since it does not provide them with water-tight answers to hard cases. However, we may consider that it is a more accurate reflection of the complex reality of moral decisions. Although virtue ethics is a system that begins with the behaviour of the individual, it has important lessons for policy-makers. First, we may conclude that any system that constrains individual virtue is morally damaging to the individual. Thus a generalised acceptance of a form of vicious behaviour, for example lying, will encourage a general cultural response in the direction of greater dishonesty, encouraging a general decline in the standard of virtue. By contrast, behaviour that is generally recognised to be virtuous operates as a kind of moral education to others.

In terms of the nuclear industry we may draw some important lessons from a virtue ethics approach. The operation of the civilian nuclear power programme has been founded on some highly dubious moral decisions. Perhaps most important is that of secrecy. Initially because of the relationship with nuclear weapons, and now because of the threat of terrorism, it is clear that the nuclear industry has tended to operate in an atmosphere of secrecy and dishonesty. One example is the secrecy over the full extent of and possible consequences of the radioactive releases following the Windscale reactor fire in 1957. There are many others. From the perspective of virtue ethics this may be considered to undermine a virtuous society. The justification of pollution and health detriment, and the minimisation of the risks involved, has also appeared to demonstrate a callousness that is not conducive to a morally sound society.

4.4 Ethical Considerations for Policy-Makers
4.4.1 Problems with cost-benefit analysis

Cost-benefit analysis is a methodology now favoured by policy-makers in attempting to decide whether a given process should be allowed to be initiated. It is the method used for deciding whether to grant a licence to build a new nuclear power station, for example. However, there are considerable problems with this method as an aid to policy-making.

In the first case it relies on the ability to measure costs and benefits accurately. It is notoriously difficult to measure environmental costs (see e.g. Pearce, 1993; Funtowicz and Ravetz, 1994). As is demonstrated elsewhere in this report, in the case of nuclear power, measurement of the negative health consequences is equally intractable. Similarly, the benefits of any process may often be assessed and given a monetary value in a way which views the process from within an existing paradigm. For example, the value of energy is assessed within a policy framework which plots an inevitable increase in our need for energy, ignoring the possibilities of energy-saving and demand management. Behind the assumption that we will always and inevitably need more energy lies the further assumption that economic growth will continue, an assumption which has long been the subject of fierce debate (see e.g. Daly, 1973). Within such a set of assumptions the benefits of extra energy are likely to be overstated.

Cost-benefit analysis has been identified as having its origin in the utilitarian philosophy and this explains its second major flaw: the question of the equitable distribution of costs and benefits. We have seen that utilitarianism is based on an averaging process, and cost-benefit analysis similarly averages costs and benefits across all members of society, considering what it calls the ‘social utility function’, which represents the simple addition of all individuals’ utility functions. But the reality of industrial processes is that some segments of society bear a disproportionate share of the cost. This is acknowledged explicitly in ICRP's para. 101 above, although ICRP ignores the need to justify it on ethical grounds.

Tietenberg (2000) offers an example from the United States. In 1979 a sociologist from Texas wrote a report about a campaign by African-Americans in Houston to oppose the siting of a hazardous waste site in their community. They lost the campaign. He suggested that race and not just income was a factor in the land-use decision. A fuller study in 1983 found that 3 of the 4 commercial hazardous facilities were in African-American communities; the fourth was in a poor community. A study by the Centre for Policy Alternatives in 1994 found that the situation had worsened.

One can easily draw parallels with the situation in the UK, where all the nuclear power-stations were sited in areas of high unemployment. The reason cited was the attempt to spread the benefits of the technological revolution, yet it can easily be seen that the costs have also been borne unduly

by these people, as evidenced by the Sellafield leukemia cluster. This policy has since been enshrined in a planning directive which zones areas of high unemployment and allows lower standards of environmental protection there in order to attract job creators.

The costs of any potentially hazardous industrial process are always minimised by siting the facility in a poor area for several reasons:

- Land costs are lower in these areas;
- Future legal liabilities would be minimised, since the poor will be less able to fight legal actions;
- Poor communities will require lower compensation, since their potential future earnings lost through early death are lower.

So the averaging methodology used during cost-benefit analyses ensures that the costs of the process under consideration will fall unduly on the poor. But what about the benefits? Wealthier households have a higher level of consumption and therefore make greater demands for the processes that generate environmental pollutants. For example, a home with a dishwashing machine and central heating will demand more electricity, and hence will be responsible for a larger share of the pollutants resulting from the production of energy. It will have received more of the benefits of energy production, but is likely to have paid less of the costs.

4.4.2 Problems with discounting

A key problem with environmental decision-making, as identified earlier, is that present actions have effects long into the future; this is a particular concern in the case of nuclear power, whose waste products will be hazardous for a future longer than we can reasonably include in our policy-making. In order to make choices when the benefits and costs may occur at different points in time policy-makers use a method known as calculating the present value, which they achieve by discounting future values using a discount factor based on the monetary interest rate.

In other words, £1 invested today yields £1.10 in a year's time if the interest rate is 10%. So the present value of £1.10 received a year from now is £1. We can find the present value of any amount of money x received one year from now by computing:

$$x/(1+r)$$

where r is the current rate of interest, now referred to as the 'discount rate'.

What would be the value of your £1 in two year's time at an interest rate of r? Because of compound interest its value would be:

$$£1(1+r)(1+r) = £1(1+r)^2$$

Hence the present value of *x* received two years from now is:

$$x/(1+r)^2$$

If we follow the same pattern we find that the present value of a one-time net benefit received *n* years from now is:

$$PV[B_n] = \underline{B_n}\,(1+r)^n$$

The present value of a stream of net benefits $[B_0, \ldots, B_n]$ received over a period of years is computed as:

$$PV[B_0 \ldots\ldots B_n = \sum_{i=0}^{n} \frac{B_i}{(1+r)^i}$$

Where *r* is the interest rate and B_0 is the amount of net benefits received immediately.

This method is used to gain a clearer idea of the present value of something which will yield future costs and benefits as well as present ones. The values of benefits and costs to future generations are greatly affected by the process of discounting, which has a fairly limited time horizon so that the present value of a benefit or cost tends to converge to its lower limit of zero within a finite, and quite short time. The process of discounting itself reduces costs and benefits occurring in the far distant future to virtually zero within a finite time. As Hussen (2000: 329) states for the benefits:

When the time horizon of a project under consideration is fairly long, as is the case for many environmental projects, the difference between private and social discount rates that are within the range 3 to 5 percent is irrelevant. This is because discounting reduces benefits coming in the far distant future to virtually zero within a finite time, as long as the discounting rate is positive. What matters is the very fact that a positive discount rate is used.

The same applies to costs, so that the process of discounting radically reduces the importance of long-lasting costs, thus most of the costs of the nuclear industry, which will be paid thousands of years into the future, will be mathematically removed from cost-benefit analysis.

The whole process of discounting implies that gains and losses to society are valued less the more distant they are in the future. So long as the discount rate, however small, is positive (implying that jam today is always

preferable to jam tomorrow) then discounting will always imply unequal weighting of costs and benefits over time. Can we justify this ethically when we are enforcing costs on future generations? Taking seriously the calls for intergenerational equity would require us to use a discount rate of zero.

4.4.3. The precautionary principle

The precautionary principle suggests that when we are unsure about the risks of a certain industrial process or its pollutants we should not allow it to proceed until we can be sure that it is safe. Such a principle has never been applied to the civilian nuclear power industry. The primary reason for the lack of precaution was that, in spite of the novelty of the procedures they were engaged in, nuclear physicists were convinced that they were not a risk to public health, and they convinced policy-makers of this also. However, it is clear from the scientific findings presented elsewhere in this report that there is considerable doubt about the health effects of radionuclides. Certain areas of scientific discovery, particularly cell biology and the study of the immune system, have made tremendous progress since the inception of the nuclear power programme. This is illustrated in particular by the fact that the risk model within which the nuclear programme currently operates was drawn up before the discovery of DNA. Given this level of scientific insecurity it would seem advisable in the interests of public health to apply the precautionary principle to the operation of nuclear stations and to prevent them from releasing further radioactive emissions until they can prove conclusively, and in accordance with the most recent physiological discoveries, that they are safe.

4.4.4. Who bears the cost?

In response to a challenge to the ethical foundation of civilian nuclear power and the cancers caused by licensed emissions, nuclear industry apologists have offered comparisons between the number of miners killed as part of the life-cycle of energy production in coal-fired power stations with the number of citizens killed by cancers consequent on nuclear releases. However, this is an ethically flawed position. The miners are well informed about the risky nature of their employment and accept it in return for direct pecuniary gain. Their situation is not the same as that of the adult or child who breathes in radioactive particles released from Sellafield without knowing they are in the air, or without benefiting directly from their production. Such people are in effect by-standers and thus have a morally distinct status from those who are engaged in producing the pollutants. The situation is more analogous to that of the people in London who died in the smogs caused by coal-fired power stations and industrial plant. Once the facts about the health risks of such unregulated burning of coal in cities were known these deaths were considered morally unacceptable, leading to the introduction of smokeless zones. A similarly strict moral position needs to be adopted with regard to the nuclear industry and

would be if the true levels of emission and their real effects of health were more widely known.

4.4.5 Accounting for different levels of radiosensitivity

It is accepted as scientific fact that not all human systems respond similarly to radiation; there are variations in levels of radiosensitivity. About 6% of the population are heterozygous for the ATM gene which confers inefficiency in the system which identifies DNA damage and enables repair to take place: these people are significantly more sensitive to radiation. A number of other genetic defects have been identified which make sub-groups exquisitely sensitive to radiation carcinogenesis. This means that a fixed level of exposure to ionising radiation represents a much greater risk to some people than others, or, put another way, that a level of licensed discharge that may be considered safe for one citizen has a fairly high probability of causing another, more radiosensitive, citizen to develop cancer.

This presents a very particular ethical problem. In the case of many genetic susceptibilities, say nut allergy or *xeroderma pigmentosum*, we can reasonably expect people suffering from such conditions to avoid nuts or stay out of the sun. However, radiosensitive citizens in a modern society face two insurmountable problems in terms of such self-protection. First, they are unaware of the condition, since there is no medical test. Secondly, even if they were aware of the condition, they could do nothing to avoid the emissions from power stations, which are released without warning and spread through the air and water. The only message to the radiosensitive would be John Gofman's: 'If you can't stand the radiation, you'd better stay out of the environment'. Again, we are faced with the result of a risk-modelling system that relies on averaging. In this case, the average radiosensitivity of the human system is used as the basis of the model. This will inevitably lead to some particularly radiosensitive members of the population facing very large risks of developing cancer and other radiogenic illnesses. By some accounts the proportion of radiosensitive individuals is roughly 20%. In addition it seems that different races may have different radiosensitivities, making the basis for radioprotection on the Japanese LSS study inapplicable to different racial groups. Once we take into account varying radiosensitivity in the population it is difficult to think of a morally acceptable alternative to developing risk models that are based on the health risks of the most susceptible citizens. The question is revisited in Chapter 9.

4.4.6 Cross-border problems

The cost-benefit procedure and the utilitarian philosophy that underpins it are both based on calculations of human satisfaction within a given community. Thus, for example, all calculations of doses to the UK public from the production of nuclear power are based on the population of the UK. However,

it is clear that environmental pollution does not recognise national boundaries. Pollution from Sellafield has been discovered throughout the North Sea, leading to complaints from Scandinavian governments. A research institute in St. Petersburg found evidence that the main source of pollution in the Barents Sea was from Sellafield, rather than the nuclear submarine Kursk which sank there. It has also been found as far away as northern Canada. The country most heavily contaminated with pollution from the UK civilian nuclear programme is the Republic of Ireland. This has led to furious political activity in a country which has no nuclear power of its own. The Irish government rightly argues that a cost-benefit analysis of the Sellafield operation may confer benefits on the population of the UK but costs are also borne by citizens of the Irish Republic who receive no benefits.

Thus the methodology of justification of UK nuclear power takes no consideration of its effect outside the borders of the UK and for it to have a sound ethical basis the deleterious consequences for citizens of other countries need also to be considered. Other examples of serious transboundary problems are satellite catastrophes like the event of 21 April 1964, when US satellite "Transit-5 BN-3" dispersed 950 g. Pu-238 (about 17 000 Ci) which tripled the amount in the worlds atmosphere.

4.4.7 De minimis *and Justification by comparison with natural background*

The Committee has considered two justifications for permitting exposures, namely the *de minimis* argument and the ‘natural background' argument. The *de minimis* argument is based on the legal principle that the ‘law does not concern itself with trifles’. Thus, an exposure which is assumed to carry a risk of say one death in 100,000 persons exposed is often advanced as a trivial risk and compared with the much larger risk of being killed in a car accident or dying of cancer following a life of cigarette smoking. Whilst these arguments may be used to minimise access to the law for compensation for trivial harms the Committee does not believe that they have any basis in ethics and are largely pragmatic. For if a madman checked into a hotel in London with a shotgun and informed the police that he intended to shoot dead 60 people (1 in 100,000) or even one person (1 in 6 million), society would naturally expect him to be arrested and locked up, yet the release of radioactive materials from nuclear sites attracts no such penalties. Nor would any cost-benefit argument make an impact on social attitudes towards the hypothetical madman. He would not be permitted, for example, to shoot only people that he found mugging old ladies or robbing banks, since even robbers have rights.

The ICRP have clearly considered the problem they have with this argument and have tiptoed away from the idea of collective dose in favour of the concept of protection of (initially) a critical target group and more recently (2007) a target ‘representative individual’. This appears to avoid anyone multiplying the individual doses by the population receiving them and the

associated risk factor to obtain actual numbers of dead people. Of course, nothing has changed, these dead people still exist; it is just that it is no longer possible to use the ICRP data to accurately calculate their number. The radiation is still released and it enters the food chain and the air, so everyone gets some of it, however small, and receives a health detriment, always finite. ICRP no longer worries about these people as their dose is less than that of the nominal *representative person.*

The argument that exposures from nuclear sites are far below natural background and therefore are somehow acceptable is similarly disposed of on a rights basis. For if a branch were to fall off a tree and kill a person walking underneath, then this would be considered an Act of God. On the other hand, if someone picked up the selfsame branch and used it to hit someone else on the head and kill them this would be murder. The release of radioactive materials capable of causing harm or even death cannot be justified on the basis of comparisons with natural analogues.

More importantly, the Committee notes also that the epidemiological identification of anthropogenic radiation-induced cancer from a putative point source is dependent upon the statistical comparison of cancer rates across ***exposed*** and ***unexposed populations***. The Committee points out that the general increase in radiation exposure associated with environmental accumulation of anthropogenic radionuclides from nuclear sites has made such comparisons impossible, since there are ***no longer any uncontaminated controls.*** The Committee recommends the employment of methods which are based on assumptions about background radiation associated with only natural isotopes at levels which existed prior to the year 1900.

4.5 ICRP: Collective Dose, Controllable Dose and Justification

As noted above, the ICRP has effectively abandoned the earlier concept of Collective Dose in the low-dose region and has replaced it with a “controllable dose” process which considers the dose to a nominal Representative Person (ICRP2007). This representative individual has now to be considered within the Concept of Justification of the practice which is being considered. Every change in radiation exposure which occurs has to be justified according to the dose to the “representative person”. This person is an average member of what used to be termed the critical group, those who are to be exposed to radiation as a result of the practice being considered. Thus protection of the public would be considered adequate so long as the risk to the Representative Person could be justified in terms of good to that person or to Society. This might be welcomed as a rights-based change of emphasis were it not for the justification in terms of “Society”. For there are many situations (indeed all bar medical exposures) where the Good to Society takes precedence over the Good to the Individual. Thus the ICRP philosophical basis is still squarely in the Utilitarian camp. Furthermore, the concentration on one individual in the group of those

most critically exposed entirely neglects the many thousands, perhaps millions, who receive doses lower than this quantity yet suffer finite harm. Their harm is real and though their doses are lower the same risk factors apply and their numbers are much greater. The Committee believes that the ICRP's acceptance that there is no threshold dose for potentially lethal mutation logically and ethically demands some measure of collective harm and that, while it may be reasonable to employ the concept of Controllable Dose in the context of regulating workforce exposures, Collective Dose must be retained as a means of estimating detriment from radionuclides released into the environment by whatever route. Abandoning Collective Dose is clearly a political discourse manipulation and is not compatible with the Justification Principle. It cannot be reconciled with the ICRP's earlier position that … *far future … doses should be taken into account in the protection of both populations and individuals.* (ICRP 1990 Para. 101).

Further, the use of the "representative person" (usually ICRP Reference Man) in methodology involving controllable dose should be changed to "most at-risk person" in order to include considerations of variation in radiation sensitivity. For example, the foetus or child may have a lower dose than the 'most exposed person' who might be a high-tension linesman or farmer, but the foetus is much more sensitive to radiation and could suffer ill health at lower levels of exposure. Similar considerations apply to radiosensitive individuals.

4.6 Conclusion

In this short chapter the Committee has discussed the ethical basis of the contamination of the environment which results from an unavoidable by-product of civilian nuclear power, military nuclear weapons testing and the use of Uranium weapons. The consequent detriment to human health makes the ethical justification of these activities virtually impossible in all but the most extreme cases (medical interventions, research and technological uses of radiation). If the nuclear industry and the military are to continue within a sound ethical framework serious questions need to be addressed and those who will suffer its health consequences need to be informed and consulted to a far greater extent than they ever have been. This is a political matter since it is assumed in a democracy the electorate or their representatives have access to the best information. In the case of radiation risk, the electorate and their representatives have no access to accurate information on the effects of these processes and the contamination of their bodies or its consequence. Parliamentary democracy fails under these conditions.

In many instances it is the environmental destruction that appalls citizens, but that they nevertheless find difficult to reverse. This results from the universal intellectual domination of the ethic of capitalism, an economic system which, to paraphrase Wilde, knows the price of everything and the

value of nothing. As Midgley points out, rationality is no longer an adequate discourse for justifying human activity. Its limitations are made clear by the conclusion implicit in policy-making that while children will inevitably die from leukemia as a result of radioactive discharges, causality will be denied and in any case their numbers are ‘absolutely small’ and therefore not worthy of consideration. The moral bankruptcy of such a justification is intuitively apparent. If we broaden our conception of value beyond that which exists within the economic growth-driven world system it becomes clear that far from being too cheap to meter, civilian nuclear power is in fact too costly to permit.

The question of the systematic increases of medium and very long-lived radionuclides in the environment from military-associated activities (weapons tests, Uranium weapons) has never been justified and therefore could be taken to be beyond the framework of any ethical system, including utilitarianism. Owing to the cross-border and indiscriminate nature of the contamination it should be considered to be a universal crime against humanity of the type discussed at Nuremberg following World War II.

5
The Risk Assessment Black Box
The International Commission on Radiological Protection

5.1 *The Black Boxes of Science*

The Committee takes the view that the dissonance between model and observation in the area of radiation risk is now so great that it is necessary to begin without any assumptions regarding the predictions of accepted scientific models and to take a fresh view of the whole system. Having examined the scientific method, we move on to examine the origins of scientific belief.

Although scientists may believe that science moves forward through the formal philosophical frameworks outlined in Chapter 3, in reality it is less rational. In the last twenty years, sociologists have begun to direct their critical gaze at scientists and their real world. In the fields of sociology and social anthropology fundamental questions about objectivity led, after the Second World War, to the examination of the origins of belief and the application of reflexive methods. We cannot escape from our culture, claimed the philosophers and anthropologists. What we appear to find when we look at other societies and cultures is largely a reflection of our own subjective view. And this interpretation is so embedded in the way we ourselves think about or understand the world that what we find is only our own interpretation of what we would be doing or thinking if we were the person being studied. Thus what we find is essentially what we put there ourselves through our interpretative assumptions.

The early search for objectivity in the late 19th century followed questions raised by discoveries in the field of relativity. The questions raised led to the logical positivist view that science was the most objective description of the physical world if the formulations were mathematical. This was because it was believed that there were somehow ‘scientific facts’ wrested from Nature and elevated to the level of ‘physical laws’, like Newton’s Laws of Motion. However, recent and closer examination of scientists at work and study and of how their theories and discoveries eventually come to be accepted in their own and the wider community shows that science is not as objective as it has been portrayed. ‘Science studies’, as this sociology has come to be known, finds that science is not free from the bias and inaccuracy which permeates all other areas of knowledge, and for the same reasons. Scientists are human beings like non-scientists. And scientific facts are not the unassailable result of forcing Nature to reveal her Truths, but are assembled from the interplay of many different items, actors, machines and procedures, all of which may be faulty, biased, inaccurate or uncertain.

In reviewing the evidence available, the Committee found that Latour’s model of scientific development through the encapsulation of theory in ‘black boxes’ is very relevant to its enquiry. Latour (1987) finds that scientific truths

are not unassailable, nor final, nor always without components derived from muddier sources than Nature herself. His model suggests that what is accepted at any period of history is a scientific world-view that consists of a system of 'black boxes'. These are encapsulations of earlier theory that are used as discrete components in the understanding and interpretation of new discoveries. Most significantly, he finds that as time passes and more knowledge is included in these black boxes it becomes increasingly difficult for scientists to unravel the components of their structures or to attack the complex system of connections that maintain their status.

The science of radiation risk is exactly such a black box. It was constructed during an atmosphere of Cold War secrecy and control largely by physicists (supported by the military) at a time before the discovery of DNA and when many of the biological responses of living cells to radiation were unknown. The body largely responsible for the construction, development and present maintenance of the model defining the radiation-risk black box is the ICRP. The Committee believes that a brief review of the history, structure and composition of the ICRP is necessary in order to understand the nature and provenance of the models which presently underpin statutory radiation-risk models.

5.2 Historical provenance of the ICRP radiological models for external and internal exposure

The ICRP claims its origins in the International X-Ray and Radium Protection Committee of 1928. The truth is that the ICRP developed out of the need, in 1945, to establish a radiation-risk body to advise and reassure those who were concerned about the new radiation exposures which followed the development and testing of nuclear bombs in the US. The immediate forerunner of the ICRP was the US National Council on Radiation Protection (NCRP). In 1946 the US Government, having tested the bomb and used it on Japan, clearly recognised the sensitive nature of nuclear science. It outlawed the private ownership of nuclear materials and set up the Atomic Energy Commission (AEC) to administer the area. At the same time, the NCRP was formed by reviving the US Advisory Committee on X-Ray and Radium Protection. This was a period when nuclear bomb development, rather than medical X-rays, was the area where most exposures would occur. The medical profession had originally established the Committee to provide itself with advice on radiation protection. Now that there was a new source of risk involving the military, government, and private companies with research contracts it was clearly necessary to rapidly set up a body with sufficient credibility to claim to be the ultimate authority on radiation risk. Because recent discoveries had shown that ionising radiation caused genetic mutations in fruit flies (implying a similar risk to people), there was a pressing need to revise the existing limits for exposure to

X-rays and extend these to the new risks from external gamma rays which resulted from weapons-development research and nuclear-bomb-test exposures. There was also need to develop exposure limits to internal radiation from the host of novel radioisotopes which were being discovered, produced and handled by workers, and discharged into the environment. There is now ample evidence that the NCRP was under pressure from the AEC to fix exposure limits which would not cause blocks to research and development.

The NCRP had eight sub-committees looking at various aspects of nuclear risk, but the two most important ones were Committee One, on external radiation limits chaired by G. Failla, and Committee Two, on internal radiation risks chaired by Karl Z. Morgan, chief health physicist at Oak Ridge. Following what now appears as negotiation with the AEC over acceptable exposure limits, the NCRP had decided on its external exposure limits by 1947. These were 0.3 rem (3mSv) per week, a reduction of the existing standard of 0.7 rem (7mSv) per week. In passing, we note that this value is 8 times higher than that which is accepted today (e.g. in the Euratom Basic Safety Standards Directive) for workers and more than 160 times that which is accepted for members of the public.

Despite the agreement on this value reached by Failla's (external radiation) Committee One in 1947, it was not until 1953 that the full report from the NCRP was published. The reason for the delay was that Morgan's Committee Two was finding it very difficult to agree on values and methods which could be easily applied to determining the doses and risks from the many different radioisotopes which could become internal sources of irradiation to organs and cells within the body. Part of this difficulty had to do with lack of knowledge at the time of the concentrations and affinities of the radioisotopes for the various organs and their constituent cells. Part of the difficulty must also have been the problem of applying the averaging concepts implicit in the definition of dose (i.e. the units themselves) to the distribution of energy density in non-uniform structures. In the event, the NCRP became tired of waiting for a resolution of these problems and in 1951, its executive committee summarily ended Committee Two's deliberations and insisted that its report on internal emitters be prepared for publication, possibly on the basis that some guidance on risk was necessary. Nevertheless, the final report was not published until 1953.

This was the time when the radiation-risk black box was sealed up. Its internal workings had been constructed under pressure for the rapid development of some convenient methodology for defining exposure. Following the use of ionisation measuring devices like Geiger counters and gas-filled ionisation chambers it was, perhaps, natural that the new system quantified dose as energy per unit volume, although the first measurements were of ionisation, not energy (Roentgens). The energy units were the Rad and the Rem, now translated to the Gray and the Sievert, but it was clear even then that these units and the energy per unit volume approach are not applicable

unless the system being irradiated is truly uniform. The model cannot deal with small volumes and inhomogeneities of dose, and for this reason, is unsafe to apply to internal irradiation. This point will be elaborated elsewhere. But the problem today is that this is the black box for radiation risk which represents the model used by the ICRP. It developed out of the NCRP. The Chair of the NCRP, Lauriston Taylor, was instrumental in setting up an international version of the NCRP, perhaps to divert attention from the clear evidence that the NCRP was associated with the development of nuclear technology in the US and also perhaps to suggest that there was some independent international agreement over the risk factors for radiation. The new body was named the International Commission on Radiological Protection.

Taylor was a member of the ICRP Committee and the NCRP Chairman at the same time. The NCRP Committees One and Two were duplicated on the ICRP with the identical chairmen, Failla and Morgan. The interpenetration of personnel between these two bodies was a precedent to a similar movement of personnel between the risk agencies of the present day.

Unlike the ECRR, whose members include researchers and which carries out and commissions fundamental studies, ICRP has always been a desk organization. It has one permanent paid member, from the late 1980s until recently its Scientific Secretary Jack Valentin. It is a desktop organization and carries out no research. ICRP has stated that it relies for its information on the reviews of scientific papers provided by UNSCEAR. But UNSCEAR carries out no research either: UNSCEAR's reports choose to cite other research which its editors choose carefully. These editors are selective in their references as is the latest ICRP 2007 report.

These radiation risk organisations have personnel in common. For example, there are overlaps between ICRP and UNSCEAR, the USA BEIR VII and the International Atomic Energy Agency. In ECRR2003 it was reported that the then Chair of the ICRP, Roger Clarke, was also the Director of the UK National Radiological Protection Board (NRPB). Clarke was also a member of the 2007 ICRP task group responsible for determining the risk coefficients (and ignoring the data that showed they were wrong). The Chair of this task group was Dr Roger Cox, Chair of the NRPB (now the HPA) but who was Chairman of ICRP Committee 1 (2001-2005), is also presently Vice Chair of ICRP and also contributing author to the 2000 UNSCEAR report. Cox was on the CERRIE Committee which voted to exclude the evidence that ICRP's model was wrong from the majority final report and is also on the USA BEIR VII Committee for its 2005 report. When these groups refer to each other for independent support, they don't have to walk far. There is Dr Abel Gonzalez of the IAEA, who is also a full member of the ICRP Committee and is listed as drafting the ICRP 2007 report. Dr Lars Eric Holm of Sweden was the current Chair of ICRP until very recently and also was also Chair of the Swedish Radiological Protection organization SSI, was Chair of UNSCEAR in 2001 and is a delegate to UNSCEAR 2006. Holm has famously gone on record as stating

that the total death toll of Chernobyl is limited to 30 seriously irradiated clean-up workers, something that is also stated regularly in public and at conferences by Abel Gonzalez of the IAEA. The point here is that all the organizations that governments depend upon for a scientific consensus argument ultimately interconnect and rely on one risk model: that of the ICRP. The ICRP is not independent of the organizations that it depends upon for its evidence, and they are not independent of it. The system is an internally consistent and epicyclically-maintained fortress of bad science, bias and false conclusions. What of that other UN organization, which might be reasonable expected to have a concern about radiation exposures and health, the World Health Organisation? In 1959 WHO was constrained into an agreement with IAEA which left the IAEA in charge of all research into the health effects of radiation. This agreement is still in force, and covers not only WHO but also FAO. At the 2001 Kiev Conference on the health effects of the Chernobyl accident, the Chair of WHO, Prof H Nakajima stated in a public interview: '*in the research into the effects of radiation WHO is subservient to IAEA, health is subservient to the atom'*. The mandate of IAEA is the development of peaceful uses of the atom, though currently it is more of an international policeman aimed at limiting the spread of nuclear weapons beyond the USA and other current nuclear states. The lack of research into the health effects of the Chernobyl accident has been blamed on the involvement of the IAEA and the emasculation of the WHO (Fernex 2001). The relevant agreement states:

. . . it is recognized by the WHO that the IAEA has the primary responsibility for encouraging, assisting and co-ordinating research on, and development and practical application of atomic energy for peaceful uses throughout the world... Whenever either organization proposes to initiate a programme or activity on a subject in which the other organization has or may have a substantial interest, the first party shall consult the other with a view to adjusting the matter by mutual agreement. (Article 1, §§ 2-3, ResWHA 12 - 40, May, 28th, 1959).

This has not prevented the NRPB from telling the UK's regulator, the Environment Agency, that UNSCEAR and ICRP are 'constituted entirely separately', a statement which the Environment Agency accepted. Thus credibility for statements on risk is spuriously acquired by organisations citing other organisations, but it can be seen as a consequence of the fact that they all have their origins in the same development and the same model: the NCRP/ICRP post-war process. This black box has never been properly opened or examined. A full and readable history of the development of radiation-risk standards is to be found in Caufield (1989). Taylor himself has described these developments in some detail (Taylor, 1971) and in an interview on the development of radiation risk in the post-war period Karl Morgan, who left

both the NCRP and ICRP, said of these organisations and their satellites, *I feel like a father who is ashamed of his children* (Caufield 1989).

In this report the ECRR Committee is not primarily concerned to criticise the ICRP, but merely to place the development of the contemporary low-level radiation-risk model into a historical context. The Committee takes the view that this approach makes it easier to see how such a wide discrepancy between theory and observation came to exist.

5.3 Criticisms of the ICRP and its methodology made to the STOA unit of the European Parliament in February 1998.

There were four main areas of criticism made at this meeting. However, the proceedings were inadequately reported by the organisers (Assimakopoulos 1998). They are outlined in Table 5.1. The criticisms of the Hiroshima basis of risk modelling as discussed by Busby (1995, 1998, 2006) are shown in Table 5.2.

Table 5.1 Criticisms of the ICRP low-dose models made at the European Parliament meeting in February 1998.

Criticism	**Author/Presenter**
1. Hiroshima basis of risk model flawed because the study and control groups were not representative of a normal population.	Prof. Alice Stewart
2. ICRP basis of risk assessment is undemocratic and biased by the membership and historic provenance of the Committee	Dr Rosalie Bertell
3. Hiroshima and all other bases of risk model unable to inform on risk from internal exposure due to averaging and other errors implicit in the units of exposure.	Dr Chris Busby
4. Hiroshima base of risk model did not include contribution from internal exposure from fallout or residual contamination as controls were exposed to fallout	Several
5. Units of exposure themselves (Sieverts) contain inappropriate value judgments and are not physical units.	Dr David Sumner

Table 5.2 Failures of Hiroshima study to explain or predict consequences of exposure

Failure mechanism	**Notes**
Inappropriate controls	Both study group and controls exposed to internal irradiation from fallout
Extrapolation from high dose to low dose	Cells killed at high dose, mutated at low dose
Extrapolation from acute to chronic	Variation in cell sensitivity following earlier exposure
Extrapolation from external to internal	External gives homogenous doses (single tracks) whereas internal can give high doses (multiple or sequential tracks) to cells local to the source.
Assumption of linear no threshold	Patently not true, though the best fit was linear exponential and linear quadratic for leukemia.
Extrapolation from Japanese to world populations	Different susceptibility of different populations is well established
Extrapolation from war survivors	War survivors selected for resistance
Begun too late and missed early deaths	Total yield not accurate, lag incorrect
Excluded illness apart from cancer	Total health detriment ignored for later exposures
Genetic damage modelled on gross abnormality	Ignored cases in first 5 years , ignored sex ratio effects on birth rates

5.3 More recent arguments about the A-Bomb studies

Since 2003 there have been some further developments in the interpretation of the A-Bomb Life Span Studies which will be briefly reported here and which show that these studies themselves are questionable as a basis for developing radiation-risk models even for external exposures. The following issues are relevant:

1. The US-funded Atomic Bomb Casualty Commission ABCC selected its study groups and began comparing them some seven years after the exposures. It has been suggested that the total cancer and leukemia yield is therefore higher than that tabulated by ABCC due to cancers developing in the early period and missed by ABCC. This is now known to be true since reports have been discovered which publish the total numbers of cases in this period (Kusano 1953).
2. The gap between exposure and clinical expression of cancer and leukemia, the lag period, has been consistently given by current risk models as greater than 5 years. This has enabled governments and risk agencies to deny causality in many situations where leukemia and

lymphoma seemed to develop almost immediately after exposures (A-Bomb Test Veterans, Uranium-exposed veterans of the Gulf War and Balkans). The early Japanese reports show that leukemia cases began developing in the first year after the bombing (the first case 3 months after exposure) and also developed in those who were not present at the time of the bomb but entered later (Kusano 1953).

3. Non-cancer data released by the RERF (e.g. epilation, skin burns) have been recently analysed by Sawada to show that there were significant health effects on populations who were too far from the epicentre to have received doses from the prompt radiation capable of causing these conditions. Sawada's analysis, presented at the 2009 ECRR conference and also published shows the anomalously great effect of internal exposure to fallout (Sawada 2007 and ECRR 2009). A similar point was made in an analysis of this dataset by Stewart and Kneale in 1999.
4. The Indian geneticist, Padmanabhan, has shown that there are genetic effects on the children of the Japanese A-Bomb survivors if the correct controls are employed (CERRIE 2004b, Busby 2006). Gross malformations and observable genetic effects were reported in the Kusano 1953 and also in anecdotal reports discussed in Busby 2006. The ABCC geneticists Neel and Schull reported that there were no observable genetic effects of the A-Bomb, which they must have known was untrue.

6
Ionising Radiation: Units and Definitions in the ICRP System and Extension by ECRR

6.1 Admission by ICRP of inadequacies in the model

Before laying out the system of quantification of dose that is used in its model, the ICRP admits the possibility of the errors likely to be associated with its use to which the ECRR report also draws attention. The 1990 recommendations of the ICRP (ICRP, 1990) stated:

(17) Historically, the quantities used to measure the 'amount' of ionising radiation dose have been based on the gross number of ionising events in a defined situation or on the gross amount of energy deposited, usually in a defined mass of material. These approaches omit consideration of the discontinuous nature of the process of ionisation, but are justified empirically by the observation that the gross quantities (with adjustments for different types of radiation) correlate fairly well with the resulting biological effects.
(18) Future developments may well show that it would be better to use other quantities based on the statistical distribution of events in a small volume of material corresponding to the dimensions of biological entities such as the nucleus of the cell or its molecular DNA. Meanwhile, however, the Commission continues to recommend the use of macroscopic quantities

In passing, the Committee notes that the ICRP's justification in (17) is based on external irradiation experiments. But since 2009 Dr Valentin himself has stated in public that the ICRP model cannot be used to assess risk to populations following exposures since the uncertainties associated with internal exposures are too great (greater than two orders of magnitude). Essentially this statement by the editor of both the 1990 and 2007 ICRP reports dismisses the ICRP model totally and makes it valueless.

6.2 Introduction to the basic dosimetric system

Radiation causes damage in living tissue by ionisation of the atoms and molecules which make up the constituent cells. Fig. 6.1 schematically describes the interaction of the main three types of ionising radiation with matter.

The process of ionisation is one in which the bonds holding the constituent atoms of the molecules in tissue together are broken. These broken ionised fragments may reform but may also react with other molecules to form new reactive materials which may be harmful to the cell. If cellular damage does occur and is not adequately repaired it may prevent the cell from surviving or reproducing, or it may result in a viable but altered cell.

The energy required to break bonds in biologically important molecules varies with the bond but is between 6 to 10 electron volts for large biological molecules like DNA or RNA. Thus the energy available in a single

650keV decay of the isotope Cs-137 is, in principle, sufficient to cause the breakage of some 65,000 bonds in such molecules.

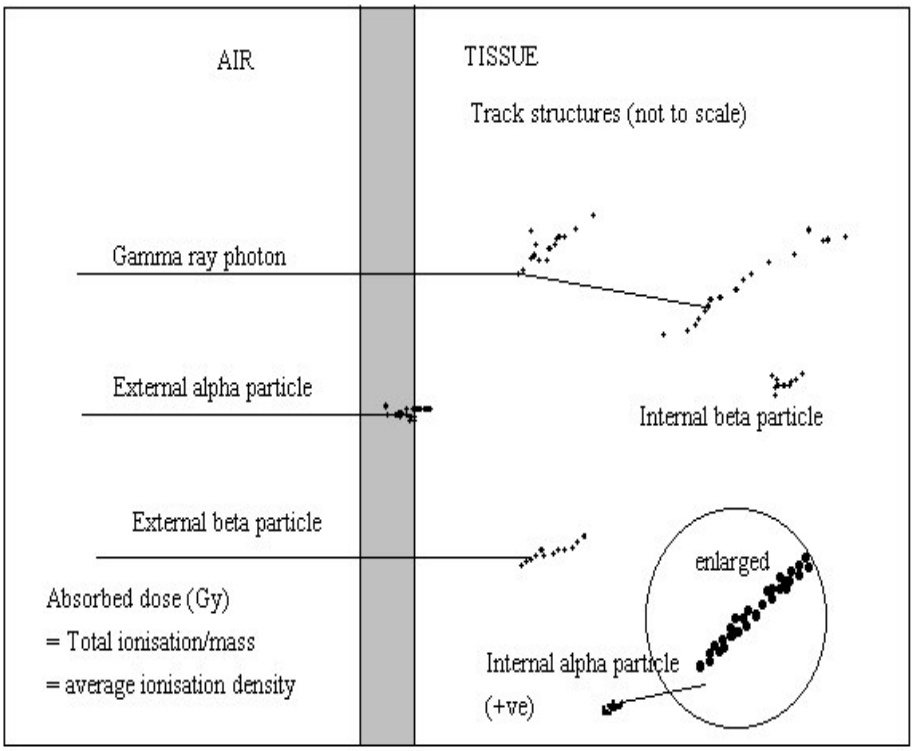

Fig 6.1. The interaction of ionising radiation with matter to produce ionised molecules.

If a large fraction of cells comprising an organ are killed then there will be an observable overall effect on the function of the organ and the health of the organism. The ICRP model distinguishes between such major 'non-stochastic' or deterministic damage and the damage which results from the probabilistic or stochastic development of effects consequent upon the acquisition of harmful but survivable alterations. In this report the Committee is not primarily concerned with the gross immediate consequences of high-dose acute irradiation but with stochastic effects following low-dose irradiation. The probability of cancer resulting from radiation may be expected to increase with increments in dose to each individual target cell up to a level where the cell cannot sustain the damage and dies.

For this reason it is important to emphasise that it is the dose to the individual cell which is the parameter of interest and indeed it is most likely, from Auger substitution experiments, to be the chromosomal DNA and associated replication apparatus (e.g. membranes) which are the target for critical damage by ionization effected chemical reactions. For internal or non-isotropically distributed radiation exposure the macroscopically estimated dose to the tissue is unlikely accurately to reflect the doses to individual cells of these organelles. In other words, averaging the energy transferred in a given mass of tissue may suggest a low-dose whilst in reality all the energy may be transferred into a very small part of the tissue. Some cells will then receive a very large dose whilst most will get none. Thus, depending on the severity of the dose, the boundary between deterministic and stochastic effects is dependent on the mass of the tissue into which the energy is absorbed

This has implications for, *inter alia*, the irradiation of the foetus by internal particulates. In the event that an irradiated cell is altered rather than killed, the outcome is very different. Despite the existence of cell repair mechanisms and, in the whole organism, further surveillance systems for the elimination of such cells, the clone of cells which carry the modification induced by the radiation will have a higher probability than the original cell of acquiring the set of genetic changes necessary to cause uncontrolled replication. This may result in the manifestation of a malignant condition, a cancer. It may also result in a detrimental effect on the efficiency of the organ or system which the cell is part, with resultant ill-health of the individual. The severity of the cancer is not affected by the dose. This kind of damage is called ‘stochastic’ meaning ‘random or resulting from chance factors’.

In the last fifteen years it has become increasingly clear from experimental results that direct damage to the chromosomal DNA and the production of a clone carrying a fixed mutation is not primarily the source of radiation-induced changes in exposed organisms. It turns out that radiation (and other types of mutagenic) damage to DNA and associated apparatus cause the induction of a signalling phenomenon termed genomic instability. This results in a signal which causes random genetic mutation to occur in the target cell and its descendants. The signal is also transferred in some way to other cells nearby, a so-called bystander effect. This important discovery, and its implications are discussed briefly in Chapter 9.

For the ICRP, however, the only late health effects that are expected to occur in populations following exposure to ionising radiation are increases in cancer incidence in those exposed and hereditary disease in their descendants.

It may be, however, that random damage to the genetic material of many cells in an organ results in a loss of efficiency of the organ. Such effects may reveal themselves clinically many years after the original exposure and may result from changes in the efficiency of descendants of the original exposed cells. For example, non-cancerous thyroid gland dysfunction may follow exposure to radioactive Iodine. Such an outcome is not easily classified

as deterministic or stochastic and is not addressed in the system of risk used by ICRP. Nor is the significant effect on heart function associated with radiation exposure. However, the Committee believes that such effects should be acknowledged and their risk quantified if possible, since they represent considerable suffering in exposed populations which is presently unacknowledged. Such general effects may be termed ‘non specific ageing’ but it should be noted that this concept is not to be identified with the idea of 'life shortening' utilised by a number of risk agencies to examine the moral implications of premature cancer death. If damage to the genes of a cell occurs in cells which function to transmit genetic information to later generations then these alterations may become expressed in the progeny of the exposed person. Such effects are termed ‘hereditary’.

Finally, it must be emphasised that genetic damage entering the human gene pool remains there until it is lost by the death of the carrier prior to reproduction. Thus heritable damage will always be expressed either in the exposed individual or a descendant until it is lost through death of the individual without issue.

6.3 The Committee’s approach to quantifying risk: weighting dose or weighting risk?

As the ICRP preamble concedes (see para. 6.1), the quantity of interest in radiation risk assessment is the ionisation energy density in the irradiated cell. ICRP approximates this as an average quantity, the absorbed dose (defined below). This absorbed energy density (dose) is weighted twice by ICRP to allow for variations in (1) biological effectiveness and (2) organ sensitivity. The final dose unit employed in radiation protection by the ICRP is a complex extension of the basic absorbed dose. The units, Sieverts, which are tabulated against particular types of exposure regime, are a mixture of physical units of average energy density and value judgements about likely health consequences based upon animal studies, epidemiology, the physical nature of the radiation types, organ sensitivity and so forth. Originally, the ICRP included the possibility of extending this system of weighting the fundamental physical quantity to considerations other than radiation quality and organ sensitivity. The ICRP noted in 1990:

In previous formulations, provision was made for possible weighting factors other than the radiation weighting and tissue weighting factors. The product of these other unspecified weighting factors was called N.

(ICRP 1990 para 30)

The Committee has learned that one of the main components of N was the affinity of an internal isotope for DNA; in this system, had it been implemented, Sr-90, Ba-140 and Uranium would have had their dose coefficients weighted to increase the effective dose equivalent because of the

locality on the DNA. The idea was quickly abandoned (Jensen 2009). In the event the ICRP chose to shift the variations in hazard associated with different exposure types and temporal fractionations away from the calculated doses and onto their published risk factors for fatal cancers—in other words the idea of modifying the units of dose was abandoned in favour of modifying the risk factors per unit dose. This suggested (misleadingly) that the units of equivalent dose had some fundamental or physical significance. In order to account for the qualitatively different exposures at the cell level associated with internal radionuclide point sources the ECRR was faced with the problem of whether to modify or wholly restructure the ICRP scheme. The Committee decided that, whilst it would be preferable to begin from first principles and develop a model in which the deposition of ionisation events at the cell level was accurately described, in the first instance it is necessary to have a simple system in which historic exposure calculations based on the ICRP model may be adjusted to provide more accurate information about health deficits.

The ECRR takes the view that the weighting factors used by the ICRP to allow for different biological effectiveness of radiations and those allowing for organ sensitivity are not qualitatively different from weighting factors to allow for different fractionations of radiation dose or for the differing abilities of various isotopes, particles or contamination types to cause mutation (and which it turns out ICRP considered in the 1970s). Consequently, the ECRR proposes to revive and employ the weighting factor N of the original ICRP model. This approach has the great advantage that although the new risks of low-level radiation doses from internal or exotic regimes of exposure may be slightly higher than supposed by the ICRP, there is no great need to alter the present legal frameworks in relation to maximum permissible doses. It is the doses themselves that will be calculated differently. The ECRR has therefore developed a range of hazard enhancement factors for various exposures which are incorporated into the **Hazard Enhancement Weighting factor N** which will be described further below.

6.4 Absorbed Dose and Equivalent Dose

The fundamental dosimetric quantity in the ICRP radiological model is the **Absorbed Dose,** D. This is the energy absorbed per unit mass, and its unit is now the Joule per kilogram, or Gray (Gy). The early unit which used to be used is the Rad. One hundred Rad equals one Gray.

$$D = \Delta E / \Delta M$$

where D is absorbed dose in Grays, M is mass of tissue into which the dose is absorbed in kilograms and E is energy in Joules. Because there exist in nature different types of ionising radiation, which have different abilities to ionise tissue, it was found necessary to allow for this by weighting the absorbed dose

by a factor which accounts for the different ionising power of the radiation. The ICRP use the term **Dose Equivalent** for their fundamental unit for radiological protection and define this as the 'absorbed dose averaged over a tissue or organ (rather than at a point) and weighted for the radiation quality that is of interest'. The weighting factor for this purpose is defined as the **radiation weighting factor** w_R and is selected for the type and energy of the radiation incident on the body or, in the case of internal sources, emitted by the source. The final weighted absorbed dose is termed the **Equivalent Dose** in a tissue or organ and the units are **Sieverts.** 1 Sievert is equivalent to 100 rem, the earlier unit.

The equivalent dose H in tissue T is given by the expression:

$$H_T = \Sigma_R W_R D_{T,R}$$

where $D_{T,R}$ is the absorbed dose averaged over the tissue or organ T, due to radiation R. The unit of equivalent dose is stated by ICRP to be the Joule per kilogram, but the weighting factor values are chosen by the ICRP Committee and so the equation is not one based in physics but contains human value judgements about the relative effectiveness of different radiations. For example, true average absorption of 1 Joule per kilogram may be weighted in such a way as to be tabulated as 20 Joules per kilogram in the case of alpha-particle exposures. This choice is made by a committee.

The values of the radiation weighting factors w_R were chosen by the ICRP to represent average values of the **relative biological effectiveness (RBE)** of one radiation type (α,β,γ) compared with another. The RBE was taken to be the inverse ratio of the absorbed doses producing the same degree of a defined biological end point. The values of w_R are roughly equivalent to the quantity **Linear Energy Transfer** (LET) a measure of the density of ionisation along the track of the ionising particle or electron track produced following absorption of a photon. ICRP chose to reference all radiation to the effects produced by X-rays and Gamma-rays of all energies to which they gave a weighting factor of unity (1.0).

When the radiation being considered is composed of more than one type then the absorbed dose must be subdivided in blocks, each with its own value of w_R, and summed to give the total equivalent dose. Radiation weighting factors chosen by ICRP are given in Table 6.1 below. In general, these weightings followed the effectiveness of producing cell death *in vitro* and it was assumed that the *in vivo* mutagenic effectiveness would follow a similar relationship.

It should be noted that these equations on which absorbed doses are calculated are employed by ICRP and were employed by ECRR2003 in a modified form as if the mass ΔM, were a mass of living tissue, essentially water. The nature of the absorbing material is not normally a consideration, but recent work would suggest that if there is contamination with elements of high

atomic number like Uranium, Gold or Platinum. The absorption of gamma and photon radiation below about 500keV photon energy is proportional to the fourth or fifth power of the atomic number of the atom absorbing the radiation. Therefore such elements whether as atoms, molecules or particles absorb very great amounts of energy from the incident photon field and emit the energy as photoelectrons which are indistinguishable from beta radiation. This is apart from any intrinsic radioactivity and is termed the Secondary Photoelectron Effect or SPE. The matter is important, mainly for Uranium (Z=92) and Iodine (Z=53) and will be discussed below and in Chapter 9.

Table 6.1 ICRP Input Radiation Weighting Factors.

Type of radiation	**Radiation weighting factor w_R**
X-rays and Gamma-rays, all energies	1
Electrons (beta particles)	1
Alpha particles	20
Neutrons and protons	Vary with energy from 5 to 20

The Committee has been made aware that suggestions from within ICRP in the 1980s to adopt weighting factors of 2 for Tritium and 5 for Auger emitters were not adopted owing to the implication that this would have for the nuclear industry. The ICRP in fact adopted unity weighting factors for these types of exposure.

There is added difficulty in assigning a radiation quality factor of unity to all X-rays and gamma rays. While medical X-rays are usually measured in Roentgens in air at skin entrance (partial body, site specific), gamma radiation is measured as bone marrow dose to the whole body. The bone-marrow dose from a medical X-ray may be significantly lower than the skin dose. For example, the skin dose for a medical chest X-ray may be 0.5 mSv, with 0.3 mSv soft tissue dose and 0.03 bone-marrow dose. This differential absorption of the ray corresponds to the sharpness of the image. A high energy gamma dose is usually taken as being the same for skin, soft-tissue and bone-marrow. It cannot be used to image internal organs. Therefore if one is using leukemia, for example, as the biological endpoint of concern, the high energy gamma ray dose of 0.5 mSv would have a higher risk than a 0.5 mSv medical chest X-ray dose (the latter is also a partial body dose).

6.5 ECRR's new system—allowance for biological response in the cell and other factors—the Biological Equivalent Dose.

Earlier it was noted that in the original ICRP formulation, provision was made for the extension of the weighting factor approach to any number of aspects of radiation-exposure regimes which might enhance or detract from the efficiency with which radiation caused cell death, mutation or ill health in the organism.

The ECRR proposes to use this approach to allow for a number of factors which have emerged through epidemiological and theoretical discoveries which have been made since the ICRP model was developed. The evidence for the existence of hazard enhancement associated with such exposures is outlined in Chapters 10 to 13. The ECRR thus defines the quantity **biological equivalent dose** as the product of the equivalent dose and the new **biological hazard weighting factor N** which may be fractional.

The biological equivalent dose B in tissue T resulting from the specified exposure E of quality R is given by the expression:

$$B_{T,E} = \Sigma_R N_E H_{T,R}$$

where $H_{T,R}$ is the absorbed dose averaged over the tissue or organ T, due to radiation R, and N_E is the hazard enhancement weighting factor for the specified exposure E.

N_E is made up of a number of hazard enhancement factors associated with different processes leading to genetic mutation and other relevant biological damage. For each type of exposure from each internal source S there will be assumed to be a weighting for the hazard associated with that exposure. This weighting is made up of biophysical and biochemical factors which are multiplicative since probabilistically they are deemed to be non-independent binomial factors which act on the same mechanism (DNA mutation). Thus:

$$N_E = \Sigma \ W_J W_K$$

in the case of J different biophysical aspects of the specified exposure and K different aspects of the internal exposure which the Committee believes carry enhanced risk of injury.

Components of the overall hazard enhancement weighting factor N are termed **biophysical hazard factors W_J** and **isotope biochemical hazard factors W_K** and these are given for some exposure types and isotopes in Tables 6.2 and 6.3. In the event that the exposure source S involves enhancement through more than one hazard aspect these are dealt with multiplicatively so long as the source and exposure (binomial probabilistic sequence) leading to mutation is the same. For example, Sr-90 binds to chromosomes but because it is also a second event decay atom, it carries an enhancement of 30 due to W_J and an enhancement of 10 due to W_K (DNA affinity), resulting in an overall enhancement of 300. For Sr-90, Table 6.3 also shows enhancement of hazard through interfacial adsorption. However, this is considered as a different exposure and is not included in the calculation for N_E but is added in at the stage of calculation of B. If Sr-90 hazard is through barrier transformation to Y-90 (e.g. Sr-90 enters a system as a divalent ion but transforms to a trivalent Y-90 and accumulates because of loss of reflexive transport) then only the

hazard factors appropriate to this exposure are used, for example, in establishing dose to brain tissue.

Table 6.2 Biophysical hazard factors W_J for exposures in the low-dose range.

Type of exposure	**Factor W_J**	**Notes**
1. External acute	1.0	
2. External protracted (see 3)	1.0	Dose rate sparing is not assumed
3. External: 2-hits in 24 hrs	10 to 50	Allows for repair interception
4. Internal atomic single decay	1.0	e.g. Potassium-40
5. Internal atomic 2nd Event	20 to 50	Depends on decay sequences and dose
6. Internal Auger or Coster-Kronig	1 to 100	Depends on location and energy
7. Internal insoluble particulate	20 to 1000	Depends on activity, particle size and dose*
8. Internal high atomic number Z^4 factor	2 to 2000	Multiply by external gamma dose rate factor (see Chapter 6 and 9)

**Tamplin and Cochran (1974) gave the enhancement of dose for Plutonium oxide hot particles as high as 115,000*

Table 6. 3 Specific internal isotopic biochemical enhancement factors W_K

Isotope or class	**Factor W_K**	**Mechanism of enhanced effect**
3-H; Tritium	10 to 30	Transmutation and local dose; Hydrogen bonding; enzyme amplification
Ionic equilibria cations e.g. K,Cs, Ba, Sr, Zn	2 to 10	Local concentration by interfacial ionic adsorption: depends on effect considered
DNA binding e.g. Sr, Ba, Pu, Ra, U	10 to 50	DNA primary, secondary and tertiary structure disruption. Local transmutation ionization
14-C	5 to 20	Transmutation and enzyme amplification
35-S, 132-Te	10	Transmutation and enzyme amplification; Hydrogen bonding
Enzyme and co-enzyme seekers e.g. Zn, Mn, Co, Fe	10	Enzyme amplification
Fat soluble noble gases e.g. Ar-41, Kr-85	2 to 10	Depends on effect considered
Barrier transmutation series e.g. Sr-90—Y-90	2 to 1000	Depends on effect considered

6.6 Allowance for organ sensitivity: Effective Dose

The critical target for ionising radiation is the individual cell. Deterministic and stochastic effects are expressed in the differentiated cells of organs, and the magnitude of both types of effect is dependent both on the identity of cell type and its location in the cell-cycle (a topic which will be addressed separately). It has been known since the beginning of the 20th century that rapidly replicating cell types (e.g. blood cells, epithelial cells of the gut) are more sensitive to ionising radiation than cells which rarely divide. Cells which are actively in division are also much more sensitive. In addition to this, cells of certain organs (e.g. the eye, the thyroid) are highly sensitive to exposure. The ICRP system allows only for variations in organ sensitivity and ignores variation in cell cycle sensitivity. It does the former by introducing an additional weighting factor, called the **Tissue Weighting Factor** W_T, which represents the relative contribution of the organ or tissue to the total detriment due to the effect being considered resulting from the uniform irradiation of the whole body. The weighting of the Equivalent Dose (or doubly weighting the absorbed dose) results in the **Effective Dose**, E. The unit is the Joule per kilogram, with the special name Sievert. However, as with the Equivalent Dose, the unit is not an objective one and depends on choices made by the ICRP Committee.

The effective dose is the sum of the weighted equivalent doses in all the tissues and organs of the body:

$$E_T = \Sigma_T W_T H_T$$

where H_T is the equivalent dose to tissue or organ T and W_T is the weighting factor for tissue T. The effective dose can also be expressed as the sum of the doubly weighted absorbed dose in all the tissues and organs of the body.

The ICRP system for Effective Dose has also been adopted by the present Committee with the replacement of the ICRP's equivalent dose with the new biological equivalent dose defined in 6. Thus

$$E_T = \Sigma_T W_T B_T$$

E_T is strictly the **biological effective dose** but the Committee believes that the term effective dose may be retained without confusion. Its incorporation into radiological safety and its units will therefore seamlessly follow prior usage.

6.7 Constructing the dose from the organ up or from the body down

It should be apparent that the overall total effective dose to a person built up by summing the individual effective doses to the different organs (in Sieverts, derived from double weighting) will not, in general, equal the effective dose calculated from the uniform equivalent dose derived from the external-

radiation-field over the whole body. In order to overcome this problem, the ICRP normalises the sum of the tissue-weighting factors to unity on the grounds that 'it is desirable that a uniform equivalent dose over the whole body should give an effective dose numerically equal to that uniform equivalent dose.' Thus:

$$\Sigma_T W_T = 1$$

The tissue weighting factors used by ICRP are given in Table 6.4. In general, the Committee favours the approach of estimating the doses to each organ or even organelle but includes the post-ICRP26 weighting factor system since much of the historic data is expressed in these terms.

Further, the weighting factors used by ICRP are based on an assumed ratio between radiogenic cancer in the tissue organ and radiogenic cancer in the whole body. This introduces major mathematical problems into such a system since a wide variation in the risk factors on an organ basis cannot be subsumed within the risk factor for overall cancer. In addition, some of the weighting factors used by ICRP in their partition modelling seem to have been chosen to diminish effects in organs which may carry high tissue loading of man-made radioactivity. In the ICRP66 lung model the tracheobronchial lymph nodes where radioactive material is stored, have been given a tissue weighting of 1/1000.

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Table 6. 4. ICRP tissue weighting factors

Tissue or organ	**Weighting factor W_T**
Gonads	0.2
Red bone marrow	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05

6.8 Dose rate, dose fractionation and protraction of exposure

The ICRP states that the consequences following an absorbed dose depend not only on the magnitude of the dose, the type and energy of the radiation (dealt

with by the radiation-weighting factor) and the distribution of the dose within the body (dealt with by the tissue-weighting factor), but also on the distribution of the dose with time, which they address as dose-rate and protraction of exposure. In earlier formulations, the ICRP solved this problem by including further weighting factors which they named N. The system was abandoned in favour of incorporating weighting factors into risk factors. The approach has been re-introduced by the present Committee (see section 6.5 above). The ICRP recognises dose-rate effects within the system of risk factors and weight these according to its beliefs using a term called the Dose and Dose-rate effectiveness factor DDREF. Thus, giving a dose over a long period of time is believed to have a lower effect (called 'sparing') compared with the acute delivery of the same dose. There is some dispute over the magnitude of such effects. No attempt is made by ICRP to examine the consequences of fractionation of doses in the time scale of the induced repair-replication period of cells.

The ECRR does not accept dose-rate sparing and has subsumed fractionation enhancement effects within the concept of biological and isotope weighting factors used to obtain biological equivalent dose. Factors for both are given in Table 6.2 and 6.3.

One particular regime of fractionation involves split doses over the period of the cell cycle: this process, involving a "Second Event' enhancement has been addressed elsewhere. The process is of importance in determining risk from sequentially decaying internal emitters like Sr-90/Y-90 but also occurs in medical imaging if more than one high-dose CAT scan is delivered within a period of 8 to 12 hours.

6.9 Time summed and collective dosimetric quantities

Following the intake to the body of a radioactive material there is a period during which the material gives rise to equivalent doses in the tissue of the body at varying rates. The total imparted equivalent dose resulting from this is affected by the rate of excretion of the material and also its physical decay characteristics (physical half-life). The time integral of the equivalent dose rate is called the **committed equivalent dose**, $H_T(\tau)$ where τ is the integration time following the intake. If τ is not specified it is taken to be 50 years for adults and from intake to 70 years for children. By extension, the **committed effective dose**, E_T is similarly defined.

In order to estimate the health detriment (defined by ICRP as cancer death and heritable damage) to large numbers of people who have been collectively exposed (e.g. residents exposed near Chernobyl) the ICRP has extended to such populations the averaging approach for cells which is implicit in the concept of absorbed dose. For such a population the average dose to an individual is multiplied by the number of individuals exposed. The relevant quantities are the **collective equivalent dose** S_T and the **collective effective**

dose S. If several groups are involved, the total collective quantity is the sum of the collective quantities of each group. The unit of these collective quantities is the **man-Sievert**, sometimes also called the **person-Sievert**.

The collective quantities can be seen as representing the total consequences of the exposure of a group. The ICRP provides the caveat that their use should be limited to situations in which the consequences are truly proportional to the dosimetric quantity and the number of people exposed and in which an appropriate risk factor is available. The collective effective dose resulting from the presence of radioactive materials in the environment may be accumulated over long periods, covering successive generations. The total collective effective dose to be expected from a given situation is the integral over all time of the collective effective dose rate resulting from (i.e. committed by) a single release. If the integral is not infinite then it is described as truncated at a definite time.

Following the increasing exposure of large (global) populations to relatively low doses from weapons fallout, reprocessing plant releases and accidents, it has become clear to ICRP that the development of these collective dose concepts has been a hostage to fortune. This is because the ICRP risk factors for exposure may be used with such large populations to calculate a finite number of cancer deaths, a situation which many people find unacceptable and which has political consequences both for the nuclear industry and the military development of nuclear weapons. The result has been a recent move by the ICRP to abandon the concept of collective dose in favour of a concentration on the individuals most exposed. Thus the ICRP would advise legislators that if the individuals most exposed in any model exposure were adequately protected at some acceptable level of risk, then all other exposed persons would be more protected and the overall cancer yield in the exposed population would be, by extension, also acceptable.

The ECRR takes the view that this is an immoral position to take and therefore an unacceptable approach, since it is the overall consequence of any proposed exposure to the whole exposed population which must be assessed. Any attempt to avoid recognising that a process will result in a finite number of deaths by focusing on low probability, high impact risks in individuals, is morally questionable. In addition, the Committee has pointed out that there is a significant difference between those who are 'most exposed' and those most 'at risk' e.g. women, children, the foetus, the radiosensitive.

The **dose commitment** ($H_{C,T}$ or E_C) is a calculational tool. It can be assessed for a critical group as well as for a large population. It is defined as the infinite time integral of the per capita dose rate (dH_T/dt or dE/dt) due to a specified event, such as a year of practice.

$$H_{c,T} = \int_0^\infty \dot{H}_T(t)dt$$

or

$$E_c = \int_0^{\infty} E(t)dt$$

In the case of an indefinite practice at a constant rate, the maximum annual per capita dose rate (dH/dt or dE/dt) in the future for the specified population will equal the dose commitment for one year of practice, irrespective of changes in the population size. If the practice is continued over time τ then the maximum future annual per capita dose will be equal to the corresponding truncated dose commitment defined as:

$$H_{c,T}(\tau) = \int_0^{\tau} \dot{H}_T(t)dt$$

or

$$E_c(\tau) = \int_0^{\tau} \dot{E}(t)dt$$

6.10 Other quantities used in radiological assessments

The activity A of a radionuclide (or radioisotope) or any radioactive material is the average number of spontaneous decays (or transformations taking place in one second. The units are reciprocal seconds (sec^{-1}) given the name Becquerel. It is possible to calculate the number of atoms of a pure radioisotope in any material by multiplying the activity by the half life in seconds using the factor 1.44. Thus:

$$N = 1.44 \times T_{1/2}$$

The quantity of the radioisotope in grams can then conveniently be found by dividing by Avogadro's number (6.02×10^{23}) multiplying by the relative atomic mass of the isotope.

Activity has also historically been expressed in terms of the isotope Radium-226 as 'Curies'. The conversion is 1nCi = 37 Bq (1Ci = 37GBq). Several other operational quantities are defined and used in radiological protection but will not be addressed in this publication.

6.11 Secondary Photoelectron Effect

The quantity employed in radiation protection, *absorbed dose*, is defined in section 6.4 as $D = \Delta E / \Delta M$. Hitherto, the mass into which the energy has been diluted is that of living tissue; ICRU provide tables of absorption coefficients for different living tissue, adipose, bone, muscle etc. which can be employed for calculations involving doses, but generally all these denominator quantities have the absorption characteristics of water, H_2O (ICRU35 1984). The absorption of electromagnetic (photon) radiation is due to a number of processes, the main three being pair-production, Compton scattering and photoelectron production. For elements of atomic number greater than about 30, and for photon energies of less than about 500keV, the photoelectric effect predominates. Even for the low atomic number elements that make up living systems, below 200keV there is fairly quantitative conversion of incident photon radiation (and induced photon radiation from second order and third order processes) into photoelectrons. These are fast electrons which are indistinguishable from beta radiation and have the energy of the incident photon less that of their binding energy (which is generally far less than the incident photon energy and can be ignored). The absorption of photon radiation by elements is proportional to the fourth or fifth power of the atomic number Z. Thus the predominant absorber in water is the Oxygen atom Z=8 and it is reasonable to give the effective atomic number of water as 7.5. Of course, there are elements in tissue with higher atomic numbers than this, but interestingly, apart from Iodine (Z=53) few elements with Z>26 (Iron, Fe).The incorporation of high Z elements into living systems would generally be harmful since it would increase the radiation dose, and therefore such developments have been lost though evolutionary selection. Iodine is an exception, but it should be noted that the main sites for radiation damage in terms of sensitivity are the main sites for Iodine concentration, the thyroid gland and the blood. It has been suggested that the metabolic and cell repair status controls exercised by the thyroid gland are the reason why Iodine has been incorporated into living systems and is employed as a kind of radiation-repair control mechanism (Busby and Schnug 2008).

A problem in radiation protection arises when high Z elements are incorporated into living tissue, since the enormously greater absorption of photon radiation by such material will result in enhanced doses to tissues adjacent to the high Z material. The problem was first addressed in 1947 in relation to X-rays of bone (Speirs 1949) and has been studied in the past in relation to prostheses. More recently, interest has shifted to the use of high Z material to enhance photon radiotherapies for tumour destruction where it has been shown to be effective. Gold nanoparticles have been successfully employed (and patented) for radiotherapy enhancement (Hainfeld *et al* 2004).

Despite this knowledge, the enhancement of photon radiation by high Z contaminants has not been addressed in radiation protection. The situation may have arisen out of the fact that prosthetic materials are not intrinsically radioactive and contamination from high Z elements like Lead (Z=82) are considered under the heading of chemical toxicity.

There are two circumstances where the Secondary Photoelectron Effect would have significant radiological implications. These are for elements that bind to DNA and for internal particulates. In the latter case, the effect will be much greater as the particle size is reduced, since for massive high Z contamination e.g. prostheses, most of the photoelectrons are lost inside the bulk material. The emergence of the photoelectrons into tissue is a function of the mean electron path in the material, and the absorbed dose in local tissue is a function of the electron range and thus its energy.

The radiological implications of the idea emerged in considering the anomalous health effects of Depleted Uranium weapons and were presented to the CERRIE Committee in 2003 and the UK Ministry of Defence in 2004 although nothing was done. More recently there have been attempts to quantify the effects for particles through Monte Carlo modelling (Pattison *et al* 2009) but these have not generally been very credible treatments, or able to cope with the small volumes of complex media involved and the answers have been far removed from the few experimental data published (Regulla *et al* 1998, Hainfeld *et al* 2004).

The particular concern is for the element Uranium, since this has been employed since 1991 as a weapon; the Depleted Uranium (DU) penetrators used from the 1991 Persian Gulf War onward produce a fallout comprising sub-micron Uranium Oxide particles which are environmentally mobile and respirable. The case of DU is considered in Chapter 12.

Uranium has another quality which makes it of interest in SPE; as the uranyl ion UO_2^{++} it has a very high affinity for DNA phosphate: some 10^{10} M^{-1}. (Nielson *et al* 1992). This affinity has been known since the 1960s when it was first employed as an electron microscope stain for imaging chromosomes (Huxley and Zubay 1961).

The SPE effect is therefore likely here to cause enhanced photoelectron ionization at the DNA due to enhanced absorption of natural background radiation (or medical X-rays). A similar process occurs with the Platinum chemotherapeutic agent cisplatin which binds to the DNA and acts as an antenna for background radiation and radiotherapy beams.

The development of weighting factors to incorporate this development into the ECRR system of radioprotection is straightforward. The effect is, of course, proportional to the concentration of material in tissue. In the case of the most important element for radiation protection Uranium-238 it is possible to employ the activity concentration Bq/kg. There is a slight difference from normal biophysical weighting since the effect is a multiplier of natural background radiation. Therefore the photon dose rate has to be incorporated

into the assessment of effect. This is done by assuming a natural background photon dose rate D_0 of 100nGy/h (0.876mGy/y) and multiplying the enhancement due to Z^4 by this. Thus the dose coefficient from U-238 (due to its intrinsic radioactivity) as given in the table is divided by 20 (for the alpha weighting) and the resulting number is multiplied by the ratio of Z^4 for Uranium and tissue. The resulting weighting factor is taken to be 1000 for a dose rate of 100nGy/h. It is proportionally increased for increased background photon exposures and for other photon exposures.

For Uranium particulates below 1micron diameter the factors in Table 6.2 apply. Dose conversion factors for Uranium-238 are given in the Annex Table A1.

For SPE phantom radioactivity in other elements of high atomic number the tissue doses are enhancements of the incident photon dose at the point of the atom or particle being considered. Due to the complex interactions these local doses must be determined by experiment and the Committee is currently engaged in preliminary experiments to establish some enhancement factors in tissue for high Z elements. These experiments are straightforward and involve X-irradiation of high Z element contaminated tissue at different doses. In principle, this development suggests that the internalization of any high Z particle which is biologically long-lived will cause continuous irradiation of local tissue cell populations, which would represent a carcinogenic hazard. This has implications for those employing prosthetic materials and also for the dispersion of high Z particles (Tungsten, Platinum, Bismuth, lead) in the environment. It also suggests that it may be of interest to examine tumours for the presence of high Z particles at their centre. Table 6.5 lists a number of potentially hazardous SPE elements.

Finally it should be pointed out that physical modelling through Monte Carlo codes is unlikely to establish useful data and certainly should not be employed as an attempt to dismiss the importance of the proposed mechanism. Nevertheless, a FLUKA Monte Carlo model of the absorption by nanoparticles of Gold and Uranium carried out by Elsaesser *et al* 2007 graphically confirmed the effect. The results for photoelectron track production following absorption of 100keV photons is shown in Fig 6.2 below.

Fig 6.2 Photoelectron tracks emerging from (left to right) 10nm particles of water (Z=7.5), Gold (Au; Z =79) and Uranium (U;Z=92) after irradiation with 100keV photons. Monte Carlo (FLUKA code) analysis. Track numbers are in proportion to a 4th power Z law (tracks are shown as projections on a flat plane). Note that the model uses 1000 incident photons for Au and U but 10,000 for water (Elsaessear *et al* 2007)

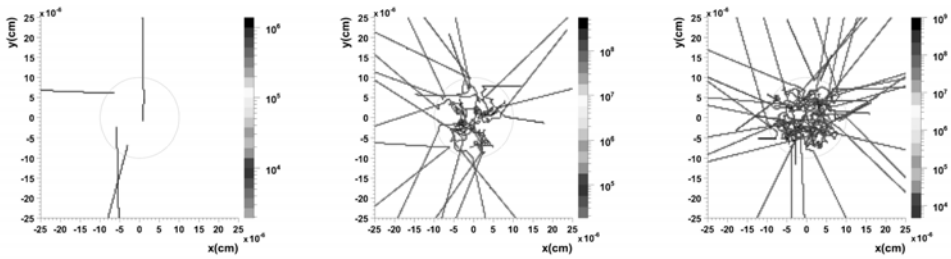

Table 6.5 Biologically significant environmental contaminants and materials exhibiting phantom radioactivity through the Secondary Photoelectron Enhancement (SPE) of natural background and medical X-rays

Material	**Z**	**Z^4/tissue**	**Source**	**Note**
U	92	22642	Weapons particles, nuclear fuel cycle, atomic and thermonuclear bomb tests	Binds to DNA; known to cause cancer in animals and genomic damage at very low concentrations
Th	90	20736	Incandescent mantles Contrast media	Highly insoluble
Bi	83	14999	General contaminant	Insoluble
Pb	82	14289	General contaminant	Toxic; SH binding
Hg	80	12945	General contaminant	Toxic; enzyme binding
Au	79	12310	Prostheses; colloid used for rheumatism	Friction particles may travel in body; inert and insoluble
Pt	78	11698	Vehicle catalysers, general contaminant	Inert and insoluble
W	74	9477	Weapons; general particle contaminant	Associated with child leukemia cluster Fallon Nevada; known to cause genomic damage and cancer in animals.
Ta	73	8975	Capacitors	
I	53	2493	Thyroid, blood plasma	Radiation sensitivity

7
Establishing the Health Effects at Low Dose
Part I: Risk

7.1 Sources of exposure in the low-dose region.

Populations are exposed to ionising radiation from both natural and anthropogenic sources and the estimation of health detriment is often made on the basis of comparing exposures from human practice with those from natural sources. Apart from the obvious points made in Chapter 4 about comparing "acts of God" with human activities, the Committee is anxious to establish the principle that each exposure should be assessed at the cellular or even the DNA level and that therefore comparisons across types of exposure are unsafe. In particular the comparisons in Table 7.1 below are major sources of error in the perception of risk.

Table 7.1 Unsafe comparisons used in radiological protection arguments.

Comparing	**With**	**Problem**
Natural	Novel	Unusual or *alien* radioisotopes for internal exposure
External	Internal	Exposure of cells is quantitatively different
Natural forms of isotopes	Technically enhanced natural isotopes	Different physico-chemical forms, concentration

Natural background radiation exposure arguments will be addressed elsewhere, but a brief review of the sources of radiation exposure will be given here. In general, the range of exposure from natural radiation is defined by the Committee as the low-dose region. This is the exposure dose range from 0 to about 5mSv, as defined by the ICRP system of measurement, though, of course, cell doses or tissue volume doses may be much higher.

7.2 Natural sources of radiation exposure

Sources of natural radiation can be divided into four categories:

- Cosmic radiation.
- External gamma radiation from natural elements in rocks and soil.
- Internal radiation from natural elements in the body.
- Radon and thoron gases from minerals in rocks and soil and their decay products.

The Committee distinguishes between these exposures and the enhanced exposure from the same sources due to human activity. In particular, there have been increases in exposure to Uranium and Thorium and their decay daughter products following:

- Burning of coal.
- Preparation and use of phosphate fertiliser.
- Commercial uses of natural radioactivity e.g. Thorium incandescent mantles, Uranium in ballast weights and shielding.
- Oil production pipe scale and process water (radium, Radon daughters)
- Natural Gas production (Radon, daughters).
- The nuclear fuel cycle (Uranium and daughters).
- The military use of Uranium including depleted Uranium (DU) weapons.
- Cosmic ray exposure from high altitude flights.

The ICRP has used its own methodology to quantify exposures to most of these sources. Examples are given below in Table 7.2.

The predominance of the dose from Radon and its decay products, it should be noted, is a consequence of the use of the weighting factor of 20 to multiply the estimated absorbed dose of 60μSv from this source. This matter is referred to since it shows the extent to which ICRP's value judgements and its choice of units of dose may inflate the appearance of hazard. The problem of Radon gas is reviewed briefly in section 7.3 below.

Table 7.2 Annual effective doses to the UK population from natural sources according to NRPB. These figures may be taken as a reasonable assessment of exposure to European populations using ICRP modelling.

Source	**Average (μSv)**	**Range (μSv)**
Cosmic ray secondaries	280	200-300
Cosmic ray neutrons	100	50-150
External terrestrial	480	100-1000
Internal Carbon-14	12	None
Internal Potassium-40	165	None
Internal Uranium and Thorium	120*	Variable
Radon and daughters	1105*	300-100,000*
Thoron and daughters	90*	50-500*
Total	2352*	1000-100000*

**These numbers include contribution from alpha decays which carry a weighting of 20. It is this weighting, a value judgment by ICRP, which has made Radon the main contribution to the total dose.*

The Committee is concerned that the definition of natural background radiation employed by the ICRP and other radiation protection agencies has not been sufficiently precise to prevent developers unscrupulously using the concept to subsume man-made radiation exposure from historic discharges within it. Thus the Committee defines the natural background level of exposure operationally

as that level which would exist in the area of interest before the advent of the nuclear age, which the Committee takes to be 1910. Any sources of exposure added to the local environment being considered since that date must be deemed to be anthropogenic and in addition to the basic level and their origin must be stated, irrespective of any question of liability.

7.3 Radon

The Committee feels that the situation with regard to assessing the effects of Radon gas should be clarified. It identifies another problem of the ICRP model in addition to that involving internal vs. external exposure: there is another large area of discussion involving whole body vs. partial body exposure. The latter category includes both Radon gas and medical X-rays. Both of these may have been misrepresented as greater hazards than nuclear pollution in exposure quantity arguments. Nevertheless, there are a number of open questions regarding the risk modelling of Radon exposures. For example the absorbed dose to bronchial epithelium is weighted by a factor of 0.2 by ICRP from 5.5mSv average (derived from the RBE of alphas and the ICRP66 model which dilutes the energy into surface cells) to an effective dose of around 1mSv. ICRP considers that contribution to other compartments of the body is negligible and it has been argued that due to this ICRP underestimates Radon dose to the bone marrow and other critical organs.

Estimates of Radon releases from natural soil vary widely from 0.2mBq/s per square metre, to 52 mBq/s per square metre. It is influenced by the condition of the soil, its porosity, moisture content and temperature. The emanation is reduced by snow and ice, heavy rainfall and increasing atmospheric pressure. There are also diurnal changes, with a maximum emanation towards the end of the night and a minimum (by half the rate) in the afternoon. Near Uranium mining the rate is several orders of magnitude greater due to the technologically enhanced releases (TENORM). Surface level crushed rock will release more Radon gas than radium buried in rock deep within the earth's crust. Much of the Radon gas problem of today has been generated by Uranium activities in support of nuclear weapons and nuclear power since 1950: this includes Radon released from Uranium wastes discharged to the sea (Hamilton 1989). To summarise, the Committee believes that the doses from Radon and its daughters may have been overstated and that this misrepresentation has had the effect of minimising the contributions to human exposure from artificial radionuclides. Nevertheless the health effects of Radon may include development of conditions not currently considered by the ICRP model which neglects the radiation exposures which lead to cancers other than lung cancer. A number of studies of miners and others exposed to Radon does support such an idea. Radon exposure and health will form the subject of a separate report.

7.4 Artificial sources of radiation

There are seven main categories of source of radiation from human activity:

- Fallout from nuclear explosions
- Discharges from accidents at nuclear plants
- Radioactive waste released both without authorisation and under licence from nuclear plants including resuspension, sea-to-land transfer and recycling of contaminated material.
- Artificial enhancement of natural radiation, e.g. fertiliser production, oil production, gas production, Uranium mining, military use of depleted Uranium, high altitude flights.
- Medical imaging and treatment
- Occupational exposure including research
- Electronic measuring devices, e.g. counters, smoke detectors, thickness gauges

UNSCEAR 2000 lists most of these sources and gives the approximate ICRP model doses from each source to the most affected group in the northern and southern hemisphere. Table 7.3 outlines the range of ICRP annual average dose from artificial sources to the UK population. There is a very wide range of doses and in general it is not possible to accurately calculate the exposure to local or distant groups. In this context the Committee is concerned that the assessment of risk from many of these sources has been based on partition modelling of the movement of radionuclides from the primary source to the exposed individual(s) followed by the application of the ICRP model outlined in Chapter 6. The resulting dose is a reductionist and complex combination of the errors implicit in both routines. However, the result is always a number which is compared both with average natural background doses and also with the results of studies of groups exposed to external radiation. This comparison is made in order to assess the risk to health of the exposed individual. Such risk to health is implicitly (and often explicitly) based on the idea that variations in levels of natural background radiation define a range of morbidity which sets a limit on the size of dose that can cause a measurable increase in some illness, usually cancer. However, such a comparison is not valid since the individual cell doses, dose rates or fractionations may be widely different. The biological dose-equivalent approach adopted by the Committee is intended to overcome this problem by making doses from all types of exposure strictly comparable.

Table 7.3. Sources of exposure to artificial radiation and ICRP calculated doses. Note that the Committee calculates these doses differently (Chapter 6).

Source	**Dose range (ICRP model)**	**Note**
Fallout from global weapons tests	Peaked in 1960s with cumulative dose of 1000 to 2000μSv. Now about 10μSv per annum.	Dose highest in high rainfall areas by factor of 3:1
Nuclear accidents affecting Europe	Windscale 1957 (10-4000μSv) and Chernobyl 1986 (10-1000μSv)	Highest doses from Chernobyl were in Bulgaria, Austria and Greece
Nuclear plant releases	Varies but not more than 5000μSv to critical groups per annum at peak discharges in mid 1970s. Average dose to public given as <10μSv per annum	'Critical group' are fish and shellfish eating; inhalation is a more important pathway but not adequately assessed by model
Uranium weapons nanoparticle fallout	Not assessed by ICRP model; assumed negligible <100μSv to exposed populations	Thousands of tons used in Iraq, Afghanistan, Balkans,
Enhanced natural radiation TENORM	Varies	Not adequately assessed
Medical imaging and treatment	Varies	Generally elective
Occupational including research	Effective dose limit 100mSv in 5 years (average 20mSv/y)	Internal exposure not distinguished

7.4 Estimating the exposure

Measuring the impact of nuclear activities begins with measuring the effluence from the industry into air and water and retained radioactive waste, the distribution of this debris in the biosphere over space and time; its uptake in the ecosystem and food web and its persistence in the biosphere; together with transfer factors in the environment; human uptake, physiological distribution in the body and biochemical properties; energy deposits; dose estimates to the public and workers; and the human and environmental health implications of this exposure. Some method for quantifying the impact on living systems is necessary to relate concentration levels to health effects. Historically, and for reasons of simplicity, this impact has been measured in terms of a quantity involving energy absorbed by unit mass, called absorbed dose. The general ICRP methodological framework is based on biochemical, physiological and health responses to absorbed dose and deciding how many such detriments are acceptable as a penalty for the benefits of the endeavour (see Chapter 4). The

question of the general utility of the physical quantity, 'absorbed dose' will be explored further below.

7.5 Estimating the risk to health

The health consequences of exposure to ionising radiation follow damage to somatic cells and germ cells and thus involve almost all illnesses. The ICRP discourse distinguishes between deterministic and stochastic effects but assumes that deterministic effects do not exist at low doses and that there are no stochastic effects except cancer and heritable effects.

Thus, in the stochastic range of effects, ICRP concentrates on cancer as a major outcome of exposure and has established probability factors or risk factors for cancer based mainly on epidemiological studies of high exposure groups. In the low and intermediate dose region the ICRP and other risk agencies have assumed a linear response between dose and cancer yield.

The Committee will not follow the ICRP in assuming that the only stochastic outcome of radiation exposure is cancer. It will address the general effects of radiation on non-cancer outcomes including adult heart disease, infant mortality and foetal death. A comparison of the ICRP's assumptions of effect following low-dose exposure and those of the Committee is given in Table 7.4

Table 7.4 The health effects of low-level radiation to be considered by ECRR compared with ICRP and other risk agencies.

Possible health effects	ICRP and risk agencies*	ECRR Committee
Fatal cancer	Yes	Yes
Non-fatal cancer	No	Yes
Benign neoplasm	No	Yes
Heritable damage	Yes	Yes
Infant mortality	No	Yes
Birthrate reduction	No	Yes
Low Birthweight	No	Yes
IQ retardation	Yes	Yes
Heart disease	No	Yes
General reduction in health and non-specific life shortening	No	Yes

**UNSCEAR, BEIR, NCRP, NRPB and EU member state agencies*

The outcome of radiation exposure in the exposed individual follows from the effects of somatic damage to cells. In the case of cancer as an outcome, there is seen both an immediate effect and a delayed effect. This pattern of risk with time is a consequence of the multi-stage aetiology of cancer (Busby 1995). Cancer is now believed to result from the accumulation of genetic damage in cells or their descendants. The particular pattern of incidence of cancer with age is most easily explained by assuming that a geometric increase in the numbers of a damaged cell clone ultimately results in a high enough probability that one of the cell descendants will acquire a second or subsequent necessary genetic mutation for cancer to develop in that cell (or group of cells). It follows that an exposure episode may either cause initial genetic damage in cells which have none or add to genetic damage which is already present. For those cells which have already acquired the initial set of genetic damage, the exposure may produce the final requirement for cancer. For undamaged cells the episode will supply the initial damage and start the process.

In addition, the exposure may also promote the cancer process in two ways. The first is by promotion, i.e. causing a general increase in cell replication rate (and therefore increasing the likelihood of mutation and also the development of the numbers of damaged cells). The second is by causing general immune system stress and thereby inhibiting the normal cancer surveillance mechanisms based in the immune system.

7.6 Detriment

In order to extend the linear system of dose modelling to risk assessment, the ICRP has introduced a number of weighting factors under the heading of ‘detriment’. Detriment is a measure of the total harm attributable to their exposure that would be experienced by an exposed group of people. In practice, this system of weighting factors is employed for a number of purposes. One is to assess the consequence of continued or cumulative exposures. Another is to compare the results of different distributions of equivalent dose within the body and to choose tissue weighting factors. The method, which is a pragmatic attempt to devise a single set of linear equations that translate every kind of exposure to every kind of radiation in every kind of population becomes extraordinarily complex and unwieldy. In addition, many errors and false assumptions are made invisible by the process which is used to give a final relationship between effective dose (which contains within it a multitude of value judgements) and cancer yield. Ultimately, the concept of detriment, though useful qualitatively, cannot be employed accurately in a rational way.

The Committee’s response to this issue is to establish a risk factor for loss of quality of life of 0.1% per mSv ECRR exposure pertaining to a general reduction in general health excluding cancer. For loss of quality of life an exposure to internal fission products conventionally assessed at 0.8mSv by ICRP calculations may translate approximately to 200mSv ECRR and convey a

20% loss of quality of life; this will involve 20% increased risk from all illnesses with a genetic or somatic genetic component in the lifetime of those exposed. In this 2010 report the Committee also includes a specific risk factor for heart disease of 0.05 per Sv. This is based on the increased risk of heart disease in exposed individuals from radiotherapists, from those exposed to weapons test fallout and those exposed after Chernobyl. The matter is further addressed in Chapter13.

Since loss of quality of life also subsumes deaths from causes other than cancer, a focus on radiation cancer epidemiology may give incorrect results due to confounding causes of death. You cannot die of cancer if you have already died of a heart attack.

7.7 ICRP models for the risk of cancer

For reasons which it does not elaborate, the ICRP assumes that there is always a latency period between exposure and clinical expression and assumes further that there is a linear relationship between the cancer yield and exposure. There are two models available for the expression of cancer following an exposure. The first assumes that the excess mortality has the same pattern in time as the natural mortality for the same cancer site. This is called the multiplicative risk projection model. If this pattern is continued throughout life, there will be a simple proportion between the natural cancer mortality and the excess due to the radiation exposure. An alternative, the additive risk projection model, postulates that the excess mortality is broadly independent of the natural mortality. The rate would rise following exposure and remain constant or fall. On the basis of epidemiological evidence, mainly from the Hiroshima study, the ICRP chose to imply a multiplicative risk projection model for all cancers except leukemia.

Following the assumptions made on linearity of effect and risk projection, the final estimates of the cancer yield per unit exposure is given by ICRP as the nominal probability coefficient, also called the risk factor. This value is a risk factor for representative populations with well-defined exposure patterns. It applies to low doses at all dose rates. In providing values for the nominal probability coefficient the ICRP makes allowance for the reduction of that probability resulting from competing causes of death. This is necessary following the adoption of a multiplicative model (see above).

In addition, owing to arguments relating to non-linearity of the measured dose-response curve for external irradiation the ICRP employs a coefficient called the Dose and Dose Rate Effectiveness Factor DDREF which is chosen to reduce the risk factor for low-level radiation exposure in the belief that at low doses the effects are less severe than at high doses. The ECRR will not employ the DDREF approach and has subsumed it within the concept of biological equivalent dose.

The risk factors expressed by ICRP are given as probabilities and may be translated in a number of ways, for example:

- The ICRP 2007 absolute risk value for cancer probability in the high dose and high dose rate region is 5.5x 10^{-2} per Sv (i.e. if this number is used to multiply by the dose and the number of people exposed to that dose, the result is the number of cancers).
- This may also be expressed as 550 fatal cancers per 10,000 people per Sievert (i.e. if 10,000 people each receive 1 Sievert, then there will be 550 cancers in this population as a result)
- Another way of expressing this risk is as a percentage; 5.5% per Sievert (i.e. if 100 persons received a dose of 1 Sievert each then 5.5 will develop cancer)

7.8 Stochastic effects in progeny: heritable damage

Apart from cancer, which is modelled as a result of somatic cell damage, the ICRP also recognises that damage to germ cells (mutation and chromosomal aberrations) may be communicated to offspring. This may manifest itself as hereditary disorders in the descendants of exposed individuals. The 1990 recommendations of the ICRP, which are presently those which underpin radiation risk models, state that radiation has not been identified as a cause of such effects in man, but experimental studies on plants and animals suggest that such effects will occur, and that such effects will range from the undetectably trivial, through gross malformations and loss of function, to premature death. Since this statement was made, applications of the minisatellite DNA testing procedure have shown unambiguous evidence of such mutation in the offspring of the Chernobyl ‘liquidators’. The matter is addressed in Chapter 13.

The nominal hereditary effect probability coefficient for severe hereditary effects (excluding multi-factorial effects) over all generations and related to gonadal dose distributed over the whole exposed population is now given as 0.2 x 10^{-2} Sv^{-1}; this is actually a reduction from the ICRP1990 value. About 80% of the effects are due to dominant and X-chromosome linked mutations.

ICRP also includes a weighting for years of life lost if the harm occurs: this is a factor which is part of the system of ‘detriment’ described in 7.5.

7.9 Effects of exposure in utero and other effects

The Oxford Survey data of Alice Stewart showed a 40% increase in cancer in children who received a 10mSv X-ray dose *in utero*. This data has now been accepted as defining an *in utero* risk for external photon radiation of 40 per Sv (Wakeford and Little 2003, CERRIE 2004, 2004a). For the internal exposures to Chernobyl fallout and employing a meta-analysis of 4 countries in Europe

and addressing infant leukemia this value is at least 160 times too low, though the effects in the children as they age is not included in this analysis and the dose response is biphasic. Different analyses by foetal dose in the several countries where data was analysed give error factors of 100 to 600-fold (Busby 2009) The Committee use a value of 50 Sv^{-1} for external X-rays of the fetus; the internal effects will be subsumed within the method of adjusting doses for internal isotopes and so the value above may be retained.

7.10 ICRP risk factors for whole body effects

The ICRP risk factors for the different consequences of exposure to radiation in the low-dose region are given in Table 7.5 These factors include all the various weightings involved in the concept of detriment but represent values which the ECRR will use as a basis for its system of risk assessment. A number of studies have suggested that these risk factors are in error by between 2- and 20-fold i.e. the cancer risks may slightly greater than suggested but the problem of distinguishing internal and external exposures has not been addressed in this context until now. The Committee's risk factors are given also in Table 7.5. The matter is discussed in Chapters 10 to 13.

Table 7.5 ICRP2007 and ECRR modified risk factors for whole population for whole body effects.

Outcome	**ICRP risk factor (per Sievert)**	**ECRR risk factor (per Sievert ECRR)**
Fatal cancer	Not specified	0.1
Non-fatal cancer	0.055	0.2
Benign neoplasm	Not considered	being assessed[b]
Heritable disease	0.02	0.04
Malformation after *in utero* exposure	100mSv threshold	No threshold
Heart disease	Not assumed	0.05
Cancer after *in utero* exposure	0.2[a]	50
IQ lowering after *in utero* exposure	30 IQ points; 100mSv threshold	30 IQ points; no threshold
Severe retardation after *in utero* exposure	0.4; 100mSv threshold	0.8; no threshold

Nominal probability coefficient expressed as Sv^{-1}
[a] *This is the ICRP 1990 value, ICRP2007 avoids giving a value but states that the risk is the same as for exposure in early childhood and neglects to give that value.*
[b] *For radiogenic benign intracranial tumours see Schmitz Feuerhake et al 2009*

Note: Values for workers, where applicable, are slightly less than these owing to the different age distribution of workers. Refer to the ICRP publications for details.

7.11 ICRP risk factors for individual organs and tissues.

The tissue weighting factors used by ICRP (described in 5.5) for defining the quantity 'effective dose' were chosen by ICRP to ensure that a weighted tissue equivalent dose would produce broadly the same detriment irrespective of the tissue or organ involved. The weightings applied include:

- The probability of fatal cancer attributable to the exposure.
- The weighted probability of non-fatal cancer.
- The weighted probability of severe hereditary defects.
- The relative length of life lost.

The model enables the ICRP to partition fatal risk according to tissue sensitivity and other factors in such a way that fatal cancer risk following irradiation of individual organs may be assessed. Factors chosen for this partitioning are given in Table 7.6.

ICRP also gives figures for aggregated detriment and separate sets of figures for workers, which allow for the different age breakdown for the latter. These are not reproduced in Table 7.6 as the present approach does not require their use.

Table 7.6 ICRP cancer incidence risk factors for individual tissues and organs for low-dose exposure

Tissue or organ	**[a]Risk factor**
Bladder	43
Bone marrow	42
Bone surface	7
Breast	112
Colon	65
Liver	30
Lung	114
Oesophagus	15
Ovary	11
Skin	1000
Stomach	79
Thyroid	33
heritable	20
Remainder	144
Total	1715

[a] *Nominal probability coefficients per Sv per 10,000 exposed*

7.12 Calculating the fatal cancer yield in an exposed population

Over the low-dose range to a few mSv ECRR assumes that a linear no threshold dose response is assumed as an approximation. Thus, to the same approximation, excess cancer incidence is proportional to radiation dose (the linear no threshold model LNT); then over this low-dose region the number of cancer cases that will occur in a population that is exposed to radiation is:

Cases = (number exposed x equivalent dose Sv) x Risk factor (per Sv)

If the collective dose is known (in person Sieverts) then the right hand side of the equation is simplified to:

Collective equivalent dose (PSv) x Risk factor (per Sv)

Because the ECRR has modified the calculation of equivalent dose by including weighting factors for the effectiveness of the radiation in causing mutation at the molecular level, the calculation is the same except that the biological equivalent dose is substituted. The ECRR calculation of excess cancer cases would thus take the form:

Cases = (number of people exposed) x (biological equivalent dose, Sv) x Risk factor (per Sv)

If the collective dose is known (in person Sieverts) then the right hand side of the equation is simplified to:

Collective biological equivalent dose (PSv) x Risk factor (per Sv)

In Chapter 14 the method is applied to global fallout and other exposures.
Excess cancer mortality may be calculated by applying the incidence-to-mortality ratio for the cancer site, population and period as tabulated by cancer registries for the area.

It will be clear that for the purposes of calculating the cancer yield above, the Committee have employed the linear no threshold LNT approach of the ICRP. The true dose-response relation is elsewhere argued to be complex and generally biphasic, i.e. after a certain dose it falls and then rises again. However, over the region and type of exposure being considered (fallout and nuclear power station doses, these are less than 1 mSv and it is assumed that the dose response is in the rising-from-zero part of the biphasic curve and can be assumed in that region to be approximately linear. This point is explicitly made in response to criticism from the French IRSN commentary published in 2005.

The evidence for the need for this approach is presented in the following chapters.

8
Establishing the Health Effects at Low Dose: Epidemiology

8.1 Evidence and inference: Bradford Hill's canons

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In Chapter 3 the scientific method was reviewed, and it was established that the method was essentially one of induction. If we wish to know the answer to the question: 'What effect does exposure to ionising radiation have on human beings?' then the most accurate answer will come from a study of a group of human beings who have been exposed in a laboratory to a known dose compared with an exactly similar group who were not exposed. This experiment cannot, of course, be performed. However, since the beginning of the last century, there have been a very large number of radiation exposures to different groups of people in different parts of the world and the outcome of many of these exposures has been studied by epidemiologists in order to construct an understanding of the health consequences of different doses and ultimately provide some evidence that would enable risk to be quantified.

Before moving on to review the evidence on which the risk factors of the ICRP and those of the ECRR are based, some account of the procedures and complications of epidemiology are presented.

Epidemiology is the study of the distribution and determinants of disease in human populations. A key aspect of epidemiology is that it is observational rather than experimental and therefore has to operate in an area where bias or confounding of the inferences drawn from the data may occur. In chemistry, a blue liquid may be mixed with a green liquid to give a red precipitate: this will always happen so long as the experiment is exactly repeated and the results can be used to draw inferences about the nature of the processes involved. But it is rare that an epidemiological study has the specificity of design and sufficient exclusion of uncontrolled variables between the study and control groups to enable unequivocal conclusions to be drawn. Therefore this is an area where studies may be electively biased or even directed to find either a result or no result. In addition, all studies may be subject to considerable criticism by groups who hold opposite views for reasons which may include culture, employment or political pressure. The Committee has found evidence of all three of these mechanisms of bias in published papers and review articles. In drawing inferences from all the epidemiological studies of radiation and health, the Committee has considered very carefully the provenance of the study and in particular the likely directional bias of the study's funding bodies and researchers.

All epidemiological studies compare a study group or groups, in this case those exposed to a known quantity of radiation, with a control group, who should be matched in every way except that they are not exposed. Before examining the real studies that attempt to translate this ideal study and quantify the risks we will first introduce some aspects of the analytical procedure. The

most valuable list of procedures which should be followed in order to draw safe inferences from evidence in epidemiological studies was devised by Sir Austin Bradford Hill in the 1950s and is termed Bradford Hill's Canons. They are sufficiently valuable in assessing the case of radiation and health to give a short account of them so that they may be applied to the radiation studies presented.

8.2 Bradford Hill's Canons

8.2.1 Statistical significance

A secure foundation for argument in any comparison of an exposed study group with an unexposed control group is that the difference in health deficit, cancer mortality for example, is statistically significant and could not have occurred by chance. Significance testing is an area of statistics and a number of basic tests may be applied to see if a result is statistically significant.

The word 'significant' is one that within the scientific community has a specific, technical meaning, but can also be interpreted generally by those without a scientific background. When a research finding is said to be 'significant' this means that it may be considered to be meaningful, in the sense of not being a chance finding. Since statistics is a methodology based on probability, it accepts a certain level of error as inevitable, meaning that some scientific findings that have passed the 'significance' test are still bound to be wrong.

The level of 'significance', which, of course, is directly related to the level of error, is chosen by the researcher, and should be set higher if the findings have more potentially dangerous implications. The level of significance generally adopted in scientific research is 5 per cent. This means that researchers are accepting a 5 per cent level of error, or that they will be wrong 1 in 20 times.

The procedure of testing whether results are 'significant' is known as 'hypothesis testing'. The scientist tests the 'null' hypothesis, which is the proposition that there is nothing unusual going on, or that the distribution of results found does not differ from what would be expected by chance.

Statistics defines two types of error that can be made when undertaking research. The first, known as a Type I error, is the one of most concern to scientists. It involves making a claim to have a research finding when in fact the results were generated by chance. An example might be a medical trial which indicated that a certain drug was effective in slowing the progress of AIDS; follow-up research might fail to find a similar result, suggesting that the original findings fell into the 5 per cent error area. For professional and credibility reasons, this is the kind of error most feared by a researcher: the error of claiming a significant result when in fact the finding resulted from chance.

But there is another type of error which is equally important, particularly in terms of potentially harmful consequences of radiation exposure.

This is the Type II error, defined as the failure to find a significant result when the hypothesis is in fact correct. It represents the risk of carrying out a study and, for reasons which may relate to technical issues such as the size of the sample, failing to find a statistically significant result. It may not necessarily mean that the hypothesis is wrong, only that significance was not found this time. However, it may allow conclusions to be drawn, either to justify use of a technology or because of extreme caution, that processes are not causing any ill effects when in fact they are.

Radiation risk studies in the low-level radiation range very often involve small numbers of people in the exposed study group, those living near a point source such as a nuclear power station for example. Studies with large populations may have small numbers of cancer cases due to very low natural rates from the disease in question: an example is childhood leukemia. In each of these types of situation, statistical methods have been developed to deal with the mathematical problem, yet finally there may not be sufficient evidence in each study to draw an inference from measured excess risk from the radiation exposure because chance could not be ruled out i.e. the result was not significant at the 5% level. This is usually a consequence of the numbers involved. When a material difference is apparent between two groups, but, with the numbers involved, is insufficient to pass the significance test Bradford Hill argues that it is better to take ‘statistically not significant’ as the ‘non-proven’ of Scottish law rather than the ‘not guilty’ of English law. It is nevertheless true that policy decisions in the area of radiation and health have fallen into the trap of assuming that 'there is no evidence that low-level radiation exposure is hazardous' means 'low-level radiation exposure is not hazardous.'

In giving weight to such evidence, the Committee made two decisions. The first was to take a precautionary approach and avoid making a Type II error in such an area of low-probability high-impact risk, for if the evidence showing excess risk from the exposure were in fact a chance finding, the mistaken inclusion of it as evidence of radiation-induced effects would not harm the human race. If on the other hand, the Committee were to take the opposite view and exclude it as evidence when it was, in fact, a true measure of a real effect but merely formally non-significant, then much harm would follow its dismissal. Consequently the second decision was to use a Bayesian approach to the refinement of belief in the area of risk assessment and allow each non-significant observation (including unpublished results) to weight and modify the overall probability of belief in the area of radiation risk according to their degree of significance. Thus the discovery of a child leukemia cluster in the 1980s near the nuclear reprocessing plant at Sellafield in Cumbria, UK has been criticised on the basis that the statistical significance of the result for the ward ($p = .002$) enabled no inference to be drawn since there are more wards in the UK than the 500 wards needed for such a result to be a chance occurrence. However, since this discovery, child leukemia excesses have been discovered near two other reprocessing plants and a number of nuclear installations in

Europe. The most recent example, a very large study of nuclear plants in Germany, found that living within 5 km of the plant doubled the risk of childhood leukemia in those aged 0-4 (Spix *et al* 2008, Kaatsch *et al*, 2008)). The Bayesian modification of the probability of the causal relation by each new example gives the Committee a firm basis of belief in the association and enables robust conclusions to be drawn about the levels of risk from exposure under these circumstances.

8.2.2 Strength of association

There should be evidence of a strong association between the risk factor and the disease: in other words, it is necessary to consider the relative incidence of the condition under study in the populations contrasted.

8.2.3 Consistency

The association should have been repeatedly observed by different persons in different places, different circumstances and times. With much research work in progress many environmental associations may be thrown up. On the customary tests of statistical significance, some of them will appear unlikely to be due to chance. Nevertheless, whether chance is an explanation or whether a true hazard has been revealed may sometimes only be answered by a repetition of the circumstances and the observations. Broadly the same answer should be given by studies using a wide variety of techniques and in different situations.

8.2.4 Specificity and reversibility

The association should be specific. The disease association should be limited, ideally, to exposure to the putative cause and those exposed should not suffer an excess risk from other kinds of illness or modes of dying. In the area of radiation risk, where the plausible biological model involves genetic and somatic damage, disease specificity may be hard to define. One condition which has become considered as a specific consequence of radiation exposure is leukemia, particularly in children. However, the specificity should be defined accurately in terms both of cause and effect. In the case of low-level radiation exposure, the lack of distinction between external and internal exposure has led to conclusions being drawn which are incorrect. Associated with *specificity* is *reversibility*. Thus removal of the cause should ideally reduce the incidence of the disease, although this is a consideration which is difficult to apply in the case of cancer, where genetic damage is not removed by removing the cause of the damage.

8.2.5 Relationship in time.

There should clearly be evidence that the risk factor preceded the onset of the disease.

8.2.6 Biological gradient

There should be evidence of a dose-response effect. This is usually taken to mean that as the dose increase, the illness rate should also increase in some proportion. However, some thought will reveal that this may not be true for certain end-points. Take, for example, birth malformations due to an exposure. Increasing the stress from zero will cause increasing damage to embryos which may eventually present as increasing risk of malformation. At some point, the weight of damage will prove too great and the embryos will die: at this dose, there will be no further congenital malformation, merely a reduction in the birth rate. It has been shown that women exposed to internal radioactive isotopes suffer an increase in miscarriages (Fucic *et al* 2007). Since there are many possible reasons for reduction in the birth rate, including social ones, the fact that exposure to a large dose of some putative mutagen has not caused any increase in birth defects ought not be taken as evidence of no effect unless lower doses are also considered and the dose-response relation adequately considered. This exact misunderstanding appears to have led to the belief that exposure to radiation from Chernobyl caused no harmful effect on birth defect, stillbirth and infant mortality rates in European populations. A number of papers asserted this on the basis of the data without drawing attention to the sharp fall in the birth rate that occurred some nine to twelve months after the exposure. A similar type of error also applies to ecological studies where some groups of individuals may have greater susceptibility to radiation. The existence of a dual sensitivity to radiation as a consequence of normal cell division also results in a dose-response relation which is biphasic, i.e. has two areas where increased effect follows increased dose, with an intervening area where increased dose results in reduced effect. The existence of inducible cell-damage repair results in a similar biphasic relationship between cause and effect.

8.2.7 Biological plausibility: mechanism

Bradford Hill stated: ‘It will be helpful if the causation we suspect is biologically plausible, though this is a feature we cannot demand. What is biologically plausible depends upon the biological knowledge of the day. It was lack of biological knowledge in the 19th century that led a prize essayist writing on the value and fallacy of statistics to conclude that among other 'absurd associations . . . it could be no more ridiculous for the stranger who passed the night in the steerage of an emigrant ship to ascribe the typhus, which he there contracted, to the vermin with which the bodies of the sick might be

infected'. For this reason the Committee is anxious to avoid dismissing evidence of health detriment following low-level radiation exposure on the grounds of lack of a plausible biological mechanism. In particular, the ICRP's assumptions about cell dose at low-level exposures provide a good example of how mechanistic arguments have been used to argue for a linear relation between dose and response, a thesis which is only valid for external random irradiation of large tissue volumes and which, in any case, is being overtaken by recent research on genomic instability and bystander effects which will be reviewed below.

8.2.8 Alternative explanation

There should be no convincing alternative explanation or confounding for the observed association.

8.3 Application to radiation epidemiology

The purpose of this chapter has been to outline the generally accepted method of assessing causation following questions about environmental causes of disease. In the following chapters, these methods will be used implicitly or explicitly to analyse the evidence that low level radiation exposure has harmful impacts on human health and to approach quantitative assessments of these impacts. The position of ICRP is that there is no impact whatever: that doses below 5mSv, as defined by their system, can have no measurable consequences. Indeed, their risk factors predict that for a dose of 1mSv (as defined by ICRP) this 'maximum permissable legal dose' gives a fatal cancer risk of 5×10^{-5}. This is one excess cancer death in the 70 year lifespan of 20,000 persons exposed. For those who have increased rates of cancer, and who live near nuclear installations, and who are exposed to radioactive pollution at low-dose levels *as calculated by the ICRP*, causality is clearly going to be rejected. But apart from the obvious and major criticism that the risk factors are culled from studies of external acute irradiation, strangely there has been no effort on the part of ICRP to apply Bradford Hill's principles of causation to their problem. The Committee has attempted such an analysis with results which are presented in Table 8.1 below.

8.4 Animal Studies

The Committee has reviewed the studies which examine the effects of low-level exposures on various animals. They note that the majority of these studies examine the effects of large external acute doses of various types of ionising radiation and accept that these may provide useful information. They also note that a number of studies have examined the health consequences of internal exposure from a number of radioisotopes. With regard to late effects following exposure the Committee has three main reservations about the extrapolation of

such results to human beings. First, with studies of short-lived animals, the time period available for the development of cancer following initial genetic damage is very restricted and is probably considerably longer than the lifespan of the individual. Second, the need to obtain observable effects results in the doses used in the study (which for cost reasons must use a limited number of animals) being very high and the controls or low-dose groups very often show anomalously high levels of cancer owing to the assumptions of linear (or continuously increasing) dose response. Finally, the use of animals may not be justified due to inter-species differences in cell repair or cancer surveillance mechanisms.

The Committee notes with interest that a wide range of animal studies of internal irradiation have revealed profound developmental and offspring mortality effects which have not been addressed by ICRP or other risk agencies.

Table 8.2 Errors in published epidemiological studies of radiation risk.

Error	**Note**
Wrong doses	Studies invariably use measured or modelled external dose as the cause covariate and subsume internal dose within it. If the latter is more hazardous no safe conclusions can be drawn from the results.
Wrong controls	1. If the controls were also contaminated the relative risk (deaths in study group/deaths in controls) will be low, possibly non-significant. This mistake has been made consistently, e.g. Hiroshima LSS, Marshall Islands, Chernobyl fallout. 2. In ecological studies of populations near nuclear sites study group and controls are generally defined by radii of circles drawn around the source. This approach makes no allowance for real movements of radioactive material via wind, water and ground topology. Controls may thus be more exposed or equally exposed. The method has been consistently used in the UK to deny risk. 3. Use of the general population as control group may be inappropriate if the study (exposed) group is not representative, e.g. healthy worker effect (nuclear workers), war survivor effect (Hiroshima LSS cohort).

Wrong sample	1. If the sample shows an effect, it may be diluted with those who were less exposed to reduce the statistical significance of the result. This is 'boundary loosening' e.g. the NRPB study of UK atomic test veterans. 2. Many different groups with different genetic susceptibility to radiation and different doses may be aggregated together and studied through time over a period of radiation exposure episodes. Lack of any step change is used to argue that there is no effect. e.g. the Nordic leukemia study; ECLIS study of leukemia after Chernobyl in Europe.
Wrong assumptions	1. The assumption of a linear no-threshold dose response has resulted in many clear observations of effect being discounted because high-dose groups may have lower cancer rates than intermediate-dose groups. e.g. nuclear workers, Chernobyl effects in Europe 2. Inducible radiation resistance has been demonstrated in animal studies yet no allowance has been made for this when comparing populations in Natural Background studies. 3. Cancer as the main outcome of exposure is modelled as a consequence of a single event. The genetic theory of cancer causation used as a model omits analysis of later effects on progression through e.g. immune system stress.
Wrong methodology	Statistical regression methods using multiple covariates are suspect because they are easy to design in a way that loses significant effects.
Wrong methodology	Ecological studies which 'lose 'significant data following Bayesian smoothing may falsely conclude that there is no effect.
Wrong end point	The ICRP has focused heavily on cancer as the end point. Many other diseases and conditions have been excluded, including infant and perinatal mortality.
Wrong conclusions	It is common for there to be a study whose conclusion or abstract claims to show no effect but which, on close examination of the results in tables and text shows clear evidence of one
Wrong data	The data itself is often suspect. Following Chernobyl, the 'liquidators' appeared to have lower rates of leukemia than the general population, but reports emerged that Soviet doctors were forbidden to record the disease (see text). In Wales, cancer cases have been removed from the database with the result that Sellafield effects on coastal population have been diminished or erased. After the Windscale fire, the direction of the fallout plume was changed and meteorological records were tampered with to minimise the effects on Ireland and the Isle of Man. In Germany, infant mortality records were altered to 'lose' Chernobyl effects.

8.5 Ideal epidemiological studies

The Committee believes that epidemiological studies and animal studies should ideally compare a specific end point and accurate data relating to the irradiated group with similar data from the same source for an exactly matched control group who were not irradiated. The irradiation pathway and exposure type must be specified well and not mixed. Outside the laboratory there are few situations where this type of study can be made, but the Committee observes that very often where such studies are possible, they are not undertaken or else the data are kept confidential. The Committee strongly recommends that morbidity and mortality data for populations of small areas be made freely available for independent research so that studies may be made which more closely approximate to the ideal. The Committee believes, moreover, that time series data on a well-defined population exposed to ionising radiation are likely to provide the best opportunity for examining its effects since the study group may be compared with itself.

8.6 Unequivocal evidence

The Committee draws attention to the unequivocal evidence of low-level radiation exposure effects demonstrated by the increase in infant leukemia in six countries following *in utero* exposure to radioactive material dispersed following the Chernobyl accident. These results show with no ambiguity that the ICRP model for low-level radiation exposure is unsound. Epidemiologically, the observation cannot be faulted since the control group in each country was the same population, unirradiated, and the lag between exposure and effect was so short that no other confounding cause existed which might explain the leukemia increase. The observation is reviewed in Chapter 10.

9
Establishing the Health Effects at Low Dose: Mechanisms and Models.

9.1 The need to consider mechanism

'Have the releases from nuclear site X caused the increase in cancer in people living nearby?' This question, and variations of it, must be addressed within the framework of Sir Austin Bradford Hill's epidemiological canon outlined in Chapter 8 and is also one of the principles of scientific method given in Chapter 3. One of the requirements for causality is that there should be a biologically plausible explanation. However, Bradford Hill himself did not insist on a mechanism, arguing that the mechanisms for an effect may not yet be understood. The Committee has considered this area carefully and concludes that the decisions made by risk agencies like ICRP to dismiss causality in such cases have been made on the basis of flawed mechanistic reasoning and lack of knowledge. The ICRP's arguments based on mechanism have led to its belief that low-level internal irradiation is harmless. Perhaps because of this, there has been inadequate research in the area with the result that there is a poor state of knowledge about low-dose effects, particularly from internal irradiation.

The Committee will review the evidence available and also outline number of mechanisms which predict and explain health detriment associated with certain types of internal irradiation.

9.2 Biological damage following exposure to ionising radiation

The damage produced by ionising radiation exposure is a consequence of four kinds of effect: They are:

- Direct ionisation of critical molecules like DNA with consequent rearrangement and destruction or alteration
- Indirect destruction or alteration of critical molecules (like DNA) through free-radical and mobile-solvent-derived ion formation
- Enhanced absorption of natural background (or medical) photon radiation by contaminants of high atomic number leading to enhanced ionization though photoelectron production
- Direct destruction or alteration of critical molecules through transmutation of a bonded or Hydrogen-bonded radioisotope.
- Indirect alteration of the cellular genome through epigenetic mechanisms which result in changes in cellular signalling processes, e.g. genomic instability, the bystander effect, induced repair efficiency.

Critical molecules are those associated with the viability and integrity of the cell, the most important being chromosomal DNA. In addition to direct attacks on constituent DNA bases by free radicals, direct hits and transmutation effects, there are also likely to be secondary causes of damage to DNA

replication associated with damage to cell membranes, repair/replication enzymes or cell communication systems. All these systems include substances which have very high molecular weight and consist of very large numbers of atoms whose position and identity are critical to their effective functioning through primary, secondary and tertiary (morphological) structure.

Following the damage to the components of a cell which are necessary for its genetic integrity and viability, the cell may repair the damage, misrepair the damage or die. Evidence has emerged recently that the cell may also exhibit the phenomenon of genomic instability, where the progeny of an irradiated cell may unexpectedly become highly susceptible to general mutation. This may also occur in the progeny of cells close to the cell which is traversed by the radiation track but which themselves are not directly hit (Mothershill and Seymour 2001).

9.3 Relationship between absorbed dose and cell dose

Both direct and indirect ionisation is a consequence of the absorption of energy from the incident ionising radiation beam or track. This causes chemical bond breakages with the formation of free radicals and reactive species. Because the main constituent of cells is water, the main free radicals and other ‘hot’ species are those which follow the rupture of the OH bond in water. For every 100 electron volts of energy absorbed, about four water molecules are split into OH' and H' free radicals. The ' stands for a free electron and these species are therefore very reactive. They may either react with each other to generate more water or react with other molecules like DNA to cause alteration or destruction of their chemical identity and biological activity (BEIRV, 1990).

The breakage of a chemical bond may be accompanied by release of excess energy in the form of electrons which can cause further bond breakages and so forth until all the energy is dissipated. Therefore, radiation manifests its effects in tissue through the formation of structured tracks of charged particles which cause, in turn, the formation of clusters of highly energetic free radicals and other electrically charged and reactive chemical species along the track. From the point of view of biological damage the effects are likely to be proportional to the concentration of such species and this in turn depends on the number of tracks in unit tissue volume in unit time and also the density of ionisation within the track. At high free-radical concentrations, however, the proportionality is affected by the increasing number of reverse reactions taking place.

The track density depends on both the exposure magnitude and type of radiation. For example, large highly charged alpha particles are relatively slow-moving compared with electrons, and the polarisation effects occurring as they move through tissue result in high ionisation density. It is largely for this reason that the ICRP has given them a relative biological effectiveness (RBE) weighting of 20 compared with beta particle irradiation and the secondary electrons generated following gamma ray absorption. The quality of radiation

that represents its ability to cause ionisation along a track is called Linear Energy Transfer (LET). Low LET radiation includes gamma rays, X-rays and beta particles. High LET includes alpha particles, which are slow and highly ionising. However, this is an approximation since the ionisation density of electrons is not uniform and increases at the end of their track as they slow down.

In the low-dose region, track density is considered to be sparse. The average track density per unit time for any absorption of radiation energy can easily be calculated. Table 9.1 gives the results of calculations in which energy is averaged over tissue to show the number of cell nucleus track traversals per year at different doses for low and high radiation energy externally incident on a human body.

9.4 Phantom radioactivity: the Secondary Photoelectron Effect, SPE.

In the above account, it is assumed that absorption and production of electron tracks is constant across tissues, in other words, it is the variation in the quantity and quality of radiation introduced to the organism as a result of some radioactivity added to the environment, either internally or externally that is the sole determinant of ionization density. This is not the case: the density of ionization tracks is also a function of the molecular and atomic components of the absorbing tissue. This is an important issue which has been entirely overlooked for radiation protection purposes, although it has received some attention from medical radiologists. The matter was outlined in Section 6.5 but will also be briefly discussed here as it is a relevant mechanism for radiation damage. Since it is universally agreed that it is the ionization density at any point which is the critical quantity in radiation protection, and since it is also argued (and shown by countless experiments) that it is the chromosomal DNA that is a critical target for radiation induced harm, the ability of the DNA to absorb photon radiation is clearly an important variable. Ionisation on or very near the chromosomal or other critical DNA is unarguably more likely to cause more damage than ionization in the bulk liquid in the cell or in interstitial material. Auger substitution experiments have demonstrated this quite clearly (Baverstock and Charlton 1988, CERRIE 2004a). It has been pointed out that the absorption of photon radiation, e.g. from natural background radiation is proportional to the fourth power of the atomic number Z of the element. This energy is re-emitted as photoelectrons. Internal rearrangements in the atom may result also in the re-emission of short-range Auger electron showers. Therefore, high Z elements which are bound to DNA, like Uranium, present a particular hazard since they act as an antenna for background radiation, re-emitting photoelectrons and Auger electrons into the DNA continuously.

The effect also is of significance for micron or nanoparticles of the element; a situation which exists in the case of Uranium particles from weapons usage and possibly Platinum particles from catalysers on vehicles, or

Gold particles abraded from prostheses. The effect is not small, and it is entirely independent of the intrinsic radioactivity of the absorbing and photoelectron re-emitting atom. There is also no doubt of its reality: radiologists have accepted it for more than 50 years and most recently are employing it to enhance radiotherapy for cancer. This mechanism is interesting in the Bradford Hill sense since massive epidemiological evidence of harm from Uranium exposures has been discounted by governments and the military and even the UK Royal Society on the basis that there is no mechanism to account for such effects at the low doses involved. The evidence for Uranium effects on health will be reviewed in Chapter 12.

9.5 The consequences of cell damage following radiation exposure

All life has been exposed to ionising radiation from natural sources over evolutionary time scales. The damage caused by radiation has two main outcomes. First, it results in finite lifespans for all living creatures by contribution to the thermal error (Boltzmann) erosion of the genetic material over the lifetime of the individual and the effects of free radicals formed through cellular oxidative metabolism. It has been well known since the 1960s that in mammals the species lifespan is directly proportional to the radiation resistance (Sacher 1955, Busby 1995,). Second, it adds to the probability of genetic mutation of species. Both of these result in health detriment, since the former is the cause of non-specific ageing and the latter is believed to be a major element in the causation of cancer and other diseases and conditions of genetic origin.

The addition of further and novel sources of radiation exposure as a consequence of human activity results in increases in exposure but also, in the case of internal isotopes, exposures which are qualitatively different. From considerations of radiation action, it is clear that cells in tissue do not suffer incremental damage as dose increases. A cell is either hit or not hit and even for low LET radiation, traversal of the cell nucleus by the primary electron track results in about 70 ionisations and a dose of 1mSv. The consequence of this hit depends upon the critical nature of the part of the cell which is affected by the ionisation, and how sensitive the cell is in that part of its lifespan that is intercepted by the radiation event.

The variation of cell sensitivity over its lifespan is not considered by the ICRP model, although a very wide variation has been known for 40 years. Variations in the normal replication rates of cells from one tissue to another cause the varying radiosensitivity of the tissues. The overall outcome of the radiation hits will also depend on DNA repair and replication systems and factors affecting their efficiency, a matter which is outlined below. Thus the result of a hit for the cell may range from 'no measurable effect' through 'accurate repair of damage' through 'fixed mutation' to 'cell death', and these are given in Table 9.2.

Table 9.1 Typical annual doses and average number of tracks to uncontaminated human tissue based on cell diameter of 8 microns, and ignoring multiple decays from certain internal isotopes.

Condition	**Radiation LET**	**Absorbed dose mGy**	**Dose equivalent mSv**	**Average number of tracks per cell nucleus per year**
Average public whole body:	Low	~0.9	1	1
Lung:	High (alpha)	0.4	20	0.001
Bone marrow	High (alpha)	0.005	0.1	0.00001
Worker whole body:	Low	<50	<50	<50
Worker whole body:	Medium (neutron)	<5	<50	<0.5
Worker whole body:	High (alpha)	<2.5	<50	0.007

Following increasing radiation exposure the effects in individuals, who are made up of many cells, will range from no measurable outcome through mutation effects to loss of viability and ultimately they may die. The same range of effects may occur in their progeny. Following the discoveries which have been made in the area of genomic instability, it now seems that about one third of all hits result in cell damage to the cell. In addition, nearby cells seem to be affected by some kind of local signalling process which causes genomic instability in them also. This is known as the 'bystander effect'. These two effects seem to be very important in the mechanistic understanding of cancer since they are associated with a general multiplication of genetic damage and this is detectable as chromosome aberration frequency increase.

As a result of examining the variation of cancer rates with age, cancer is now believed to be the result of up to six separate genetic changes. These include acquisition of specific oncogenes and loss of tumour suppressor genes. Since the normal rate of genetic mutation on replication is about 10^{-5} per gene, it has been difficult to explain how the acquisition of enough mutations to cause cancer can occur in the lifetime of an individual. Advances in technology have recently enabled computer control of microbeam radiation sources so that single cells may be hit, and new chromosome staining techniques have enabled their descendants to be identified and checked for damage. This has shown important effects. Genomic instability engendered by radiation at very low doses (i.e. up to 10mSv) results in an increased general level of genetic

mutation in the progeny of a cell which is hit. In addition, general levels of mutation increase, through bystander signalling in the progeny of a significant proportion of cells in the vicinity of the cell which is hit. These effects increase the general rate of mutation in cell volume elements to a level where enough mutations are produced to explain the development of cancer (Little 2002, Hall 2002, CERRIE 2004, CERRIE 2004b, Mothershill, 2009 ECRR 2009). Table 9.2 lists the range of outcomes to the individual following increasing dose to the individual cell.

Table 9.2 The effects of increasing doses on cells and individuals

Increasing dose range group	**Effect on cell**	**Effect on individual**
1	No measurable effect	No measurable effect.
2	Induction of genomic instability/ invisible damage: cell descendants prone to mutation	Unknown but likely to be finite and include most health conditions. Effect increases vertically from 2 to 3 hits, then saturates rapidly.
3	DNA damage with accurate repair: cell replicates accurately	No measurable effect.
4	DNA damage with irrelevant mutation: cell replicates with fixed mutation	No measurable effect.
5	DNA damage with critical mutation: cell replicates with fixed mutation	Cancer or leukemia. Genetic malformation or genetic disease if in germ cells.
6	DNA damage with lethal mutation: cell dies on replication	From no measurable effect through loss of viability of organ or individual to death of individual depending on number and type of affected cells.
7	Localised DNA damage to many cells in a community	Field cancerisation though failure of cell communication inhibitions

9.6 The dose- response relationship

The relationship between radiation dose and response has been studied extensively. The ICRP risk model assumes that in the low-dose range, the relationship is a linear one with no threshold for onset of effect, known as LNT. This means first, that there is no safe dose and even the lowest dose has a finite probability of causing health detriment. Second, doubling the dose causes a doubling of the effect. There are basically two reasons for this assumption.

The first is that it follows from the considerations of what is known about radiation action outlined in Section 9.2 above. Clearly if the health deficit is related to cell DNA damage, which in turn is a consequence of 'hits', and if these hits act independently owing to their distance apart in time and space, the effect must be linearly proportional to dose. Because a cell is either hit or not hit there is no condition lower than a single hit. There is no safe dose.

The second reason for believing in linear dose response is that data from experimental cell cultures and animals, and people exposed to external radiation, has been taken to show effects which are linearly proportional to dose. However, this has been disputed by those who argue that there is a smaller (or even beneficial) effect at low dose and others who argue that data show a higher effect at low dose. In the case of external irradiation studies, the small populations studied result in wide confidence intervals and a number of different curves can be drawn through the data.

The Committee has studied this area very carefully, since assumptions about dose-response are critical to the understanding of epidemiological studies of radiation exposure. The Committee concludes that there is sufficient evidence to believe that the dose response relationship is unlikely to be linear in the low-dose region, except as an approximation for external radiation, and has rejected the LNT approximation in favour of relationships which show much higher effects at low dose. The reasons for this are reviewed below.

9.6.1 ICRP linear and linear quadratic response: two-hit kinetics

In experimental results from external exposure studies of cell cultures, animals and human populations (primarily Hiroshima) over the full range of effect from medium dose to high dose (but before the death of the individual when the relationship fails altogether), it has been observed in many systems (e.g. leukemia induction in the LSS group) that the response is best described by a linear quadratic relationship. This is written:

$$\text{Effect} = \alpha(\text{dose}) + \beta(\text{dose})^2$$

The shape of this curve is shown in Fig 9.1. There are sound theoretical reasons for interpreting this as due to independent track action in the linear range with a much increased effect when the dose is so great that two tracks impinge on a cell at the same time. These two tracks (or correlated tracks) are thought by

most to have a high probability of inducing a fixed mutation because they can cause damage to both DNA strands in such a way that there is a 'double strand break', an event which is difficult for the cell to repair. This may not be the true reason for the increased mutation efficiency but the observation that two hits have a very much larger chance of causing mutation is now well accepted. Recent work with alpha particles and cell cultures has confirmed this empirically.

For a dose of 1mSv of 600eV external irradiation, the probability of producing two hits within the period of 10 hours which is associated with cell repair and replication has been calculated to be between $1x10^{-4}$ per year assuming a mathematical model for close packing of 8μm diameter cells and 5 x 10^{-6} if an experimentally determined packing fraction is used (Busby 2000, Cox and Edwards 2000). In other words, the two-hit process is very rare at normal background levels i.e. in the low-dose range. However, the same is not true for a number of situations involving internal irradiation. There are basically three types of internal exposure which may lead to a high probability of two hits in the low-dose range. They are:

- Immobilised sequential emitters like Strontium-90/Yttrium-90, tellurium-132/Iodine-132
- Immobilised, insoluble 'hot particles' or aggregates of e.g. Plutonium or Uranium Oxides; also high Z sources for SPE effects bound to DNA
- The very low energy beta emitter, Tritium which has a high number of tracks per unit dose

Clearly, if the dose-squared region of the accepted risk model, as defined above, is due to correlated double hits, then it follows that for these exposures response should be proportional to the square of the dose, and internal exposures like this cannot be subsumed within the external risk model without a weighting for this effect. Indeed, it may be that the true dose-response is a polynomial, in which case, triple-correlated events carry a cubed hazard weighting and so on. ICRP2007 have discounted the 2nd Event effect on no research basis whatever. But there is another reason why these types of exposure should be considered separately and this is considered below.

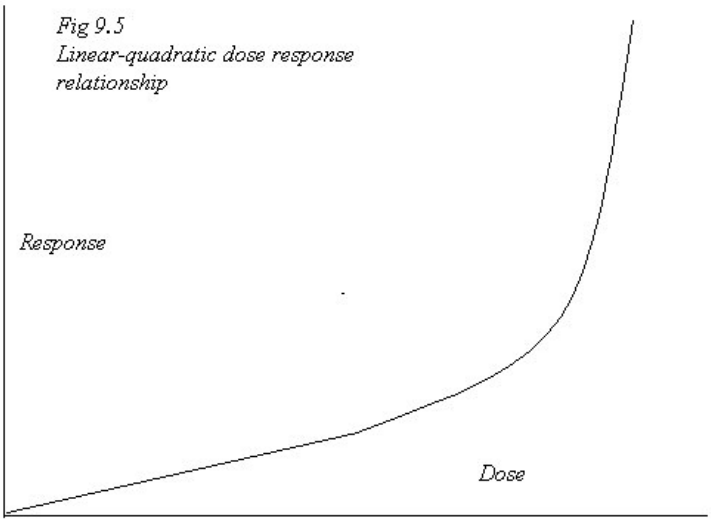

Fig 9.1 Linear-quadratic dose response relation

9.6.2 Petkau response

A number of independent researchers have drawn attention to the empirical work of Petkau, who irradiated lipid membranes in water with external X-rays and also with beta radiation from solvated radioactive sodium ions (Na-23). Petkau was interested in the effects of ionising radiation on cell membranes, which he and others came to consider to be critical targets for radiation action. Petkau showed that the membranes were extremely sensitive to radiation from ions in solution and collapsed at doses in the low-dose range. Using enzymes, particularly the anti-oxidation stress enzyme *superoxide dismutase*, he identified the hydrated peroxide species formed by radiolytic cleavage of water molecules as the cause of the lipid membrane destruction. He also demonstrated that the dose response curve for these systems is what is now called supra-linear. This is a response which rises sharply from zero dose but flattens out at higher dose. The curve is shown in Fig. 9.2.

The explanation of the curve is straightforward in kinetic theory and follows from the recombination of radical species at high concentration. Integration of the rate equation for such a system leads to a dose response of the form:

$$(\text{Response})^2 = \text{Dose}$$

It is possible, however, that Petkau was seeing partly or wholly a Langmuir type isotherm for the adsorption of radioactive sodium ions on the lipid

membrane. Nevertheless, Gofman has re-analysed the Hiroshima LSS data to show that it conforms to the Petkau type of supralinear curve, and many others have used it to argue against the extrapolation of the Hiroshima data from high dose to the low-dose region.

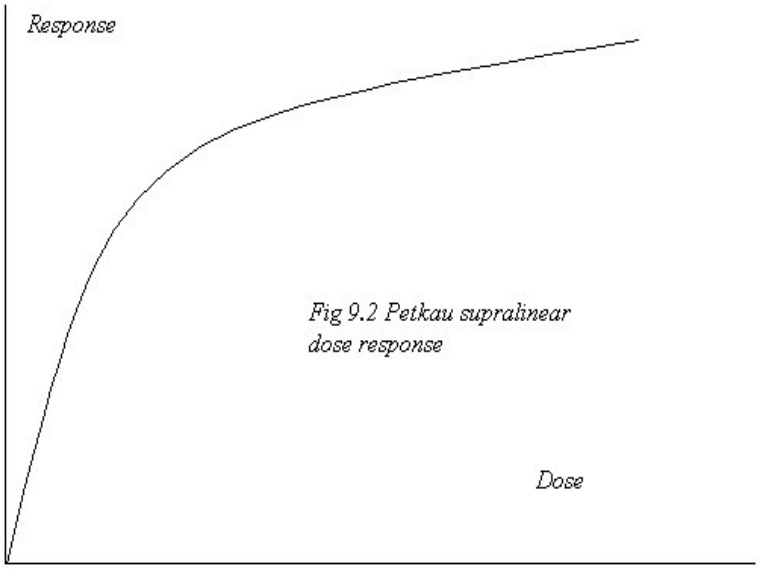

Fig 9.2 Petkau supralinear dose response curve (genomic instability bystander yields of damaged chromosomes appear to follow this type of response)

The dose response obtained empirically from microbeam irradiation of cells *in vitro* shows such relation when the damage is plotted against the number of tracks traversing a single cell, with saturation of genomic instability effects at 3 tracks. Whether this is a dose effect or track sequence effect is not known.

9.6.3 Burlakova response: inducible repair and/or sensitive elements

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Burlakova has shown in many studies that a number of different cell culture test systems respond to external low level radiation exposure with a biphasic response (Burlakova *et al* 2000). The effect increases from the zero dose point to a maximum, but then falls back to a minimum as dose is increased. Further increase in dose past this point causes a second rise in effect. In order to explain this curious result, Burlakova has suggested that the curve is a consequence of two separate processes. The first she assumed to be the Petkau or some other supralinear response to increasing radiation dose. The second is

that increasing dose increases repair through a system of inducible repair efficiency. Such systems have indeed been shown to exist in animals, but they usually take some time to develop. The biphasic dose response is thus a consequence of the opposing operation of these two effects. It is shown in Fig 9.3. Burlakova has also been able to show in a meta-analysis of studies of leukemia and radiation that these studies taken together conform to this biphasic pattern. More recently, she has suggested that the effect may be due to the superposition of response functions from several different classes of system whose response to radiation damage indirectly affects the end point being considered. Thus, increased effects at very low dose below 1mSv may reflect damage to the cell membrane insofar as it is able to support accurate replication of DNA: at higher dose this mechanism is swamped by a different mechanism, perhaps direct DNA damage or damage to some other organelle.

9.6.4 Variation in cell population sensitivity

An alternative explanation of the biphasic dose response has been suggested by Busby, but is also implicit in an idea which Elkind advanced to explain certain results of experiments showing that split doses of X-rays produced a greater effect than the same dose given acutely (see Busby 1995 and CERRIE 2004b for a discussion and references).

It has been known from almost the beginning of the radiation age that rapidly replicating cells are more sensitive to radiation damage (Bergonie and Tribondeau, 1906). Indeed, this is the basis of radiotherapy for cancer where it is the rapidly proliferating cancer cells that are preferentially destroyed. Most cells in a living organism are in a non-replication mode, sometimes labelled G0. However, it is clear that, as a consequence for a need to replenish cells that have died or are senescent, there will always be a proportion of cells actively engaged in replication, or mitosis. This involves a complex sequence involving DNA repair and replication, and during these phases, it is well established that cells are more easily killed. In some cell-culture studies, there is a 600-fold difference in the sensitivity of cells to killing by radiation during this repair-replication period, which lasts about ten hours. Experiments with the Auger emitter, Iodine-125 bound to uridine, one of the DNA bases, have shown that cells engaged in repair-replication are also much more susceptible to mutation, and that the target for the effect is either the DNA or some structure that comes into close proximity to it during this replication phase.

It follows that, if there is a sub-group of any specified cell type which has high sensitivity to mutation and killing, the dose-response will be biphasic. These sensitive cells will be mutated at low dose, increasing the magnitude of the end point effect, and as the dose in increased more, they will die, thus decreasing it. At still greater doses, the less sensitive cells will be mutated and the magnitude of the end-point effect will again rise. The result is shown in Fig 9.3.

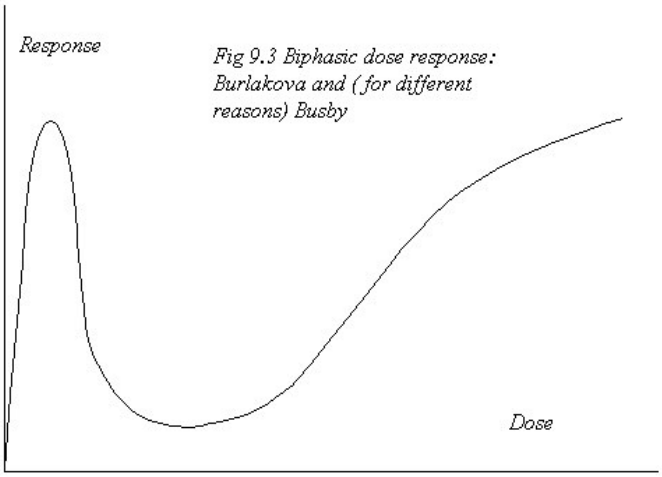

Fig 9.3 Biphasic dose response curve.

Elkind originally made the suggestion in the mid 1990s that there must be a sub-group of sensitive cells in all tissues, but this idea does not seem to have been followed up. This is remarkable, since the idea that cell killing can occur at high dose has been used to explain dose-response relationships at high dose, particularly for alpha particle effects and 'hot particle' effects. In the latter, it is argued that the high doses in the region of hot particles (highlighted by those who argue that such doses are omitted from consideration following the averaging process implicit in the concept of absorbed dose), are less likely to result in cancer due to cell killing.

The results of animal studies on beagle dogs and mice appear to show these biphasic effects in the low-dose region (Busby, 1995) as do the results of recent mortality studies of radiation workers in the UK.

It has been remarked that for the purposes of radiation-protection models, ECRR2003 employs a linear no-threshold model despite espousing the biphasic dose response (IRSN 2005). This is because in the region being addressed by the ECRR applications the dose response is in the rising part of the curve, from zero-dose and this can be approximated as a linear relationship.

9.6.5 Intra-population and individual sensitivity

There are radiosensitivity group differences both between population subgroups and between separate populations and also individual variabilities. There are data about radiosensitivity for:

- races
- populations (in the strict sense)
- sex
- age
- physiology

All three main racial groups (caucasian, negroid, mongolian) differ in radiosensitivity (see Doll and Peto 1981 for a discussion of cancer incidence rates by race). Animal and human studies have identified genetic sub-groups with enhanced sensitivity to radiation, e.g. in the Japanese LSS study and in women developing early breast cancer. There are data that show that different strains of laboratory mice differ by an order of magnitude to radiogenic liver cancer (Ito, 1999 cited by Yablokov 2002).

In Table 9.3 are listed some examples of the sexual variability of radiosensitivity in humans and Table 9.4 lists variation in some animals.

There are many studies about age-dependent differences in radiosensitivity in human beings, in vertebrates (fish, amphibians, birds, mammals) and invertebrates (Majeikite 1978 cited by Yablokov 2002). Beginning at fertilization each stage of individual development is different in its radiosensitivity. The radiosensitivity of children, adolescents, adults, elders and the very old are different. Even adults become more radiosensitive after 45 years.

The foetus is particularly sensitive as has been shown by the work for Stewart *et al* 1958 which has been translated into a risk factor for radiogenic cancer of 50 per Sievert (compared with 0.05 per Sievert for the adult (Wakeford and Little 2003). For effects in children (and possibly also adults, who are thus selected for survival) increasing dose will eventually result in decreasing end point in the viable individual due to spontaneous abortions (miscarriages). Fucic *et al* (2008) showed that there was a 4-fold excess of miscarriages in women exposed to internal radioactivity in the workplace compared with those exposed to external X-rays; external radiation is also associated with an approximate doubling in miscarriage rate (Steele and Wilkins 1996, Lindholm and Taskinen 2000) therefore the internal exposures have an approximately 8-fold effect on miscarriages.

Table 9.3 Examples of different sexual radiosensitivity (references *op.cit.*Yablokov 2002)

Character	**Differences**	**Reference**
Embryo and foetus radiosensitive	Male	Sherb *et al.* 2001)
Total cancer mortality	Higher for female in Chernobyl contaminated territories	Antipkin, 2001
Leukemia mortality	Twice higher for female	Wing *et al.* 1991
All cancer morbidity	More in girl +5 years (compared with same age boys) on Chernobyl contaminated territories	Suslin, 2001
	More in boys 0- 4 years old (compare with same age girls) on Chernobyl contaminated territories	
Bone and cartilage cancers	More in girls 0 – 4 years old (compared with same age dose) on Chernobyl contaminated territories	Suslin 2001
	Sixfold more in boys than in girls (average for globe)	
Lympho-reticulosarcomas	Female 7, male - 21 per 100 000 on Chernobyl contaminated territories	Health consequences …, 1995
Monocytic leucosis	Female 1.77± 0.42, male 3.47± 0.74 per 100 000 on Chernobyl contaminated territories	
Skin cancer	Female 16.7 (1.1 – 29.0), male 21.6 (3.2 – 36.0) per 100 000 , 19 USSR provinces	Suslin 2001
Cs-90 half-time of remote from body	Average for female 80 days , for male – 110 days	Mel'nov 2001
Newborn sex ratio	More newborn girls after intensive X irradiation; in second generation	Golovachev 1983

Table 9.4 Differencies in radiosensitivity between female and male in some mammalian species (references *op.cit*. Yablokov 2002)

Species	**Differences**	**Reference**
Rat, *Rattus norvegicus*	Level of Cs-137 incorporation for female three times higher that for male	Bandashevsky 2001
Mice. *Mus musculus*	Radio induced hepatic cancer after Californium-252 irradiation was ten times higher in female	Ito 1999
Vole, *Microtus oeconomus*	Sencitivity of bone marrow and epithelial cells higher in males	Zainullin 1998
Capra, *Meriones* and some other species	Differ between male and female	Majeikite 1978
Vole, *Clethrionomys rutilus*	Female during breeding season on radioactively contaminated territory incorporated twice more Cs-137	Il'enko and Krapivko 1989
Vole, *Microtus agrestis*	More Sr-90 incorporated in bones of females	
Hare, *Lepus europaeus*	More I-131 in bones of females	

The level of incorporation of radionuclides in some organs is different for adults and children (Bandashevsky and Nesterenko 2001). The scale of age-dependent variations of radiosensitivity (several times) is usually higher than sex-dependent ones. There are known time-dependent variations of radiosensitivity (in day, month, seasons) in insects (i.e. *Laspereysia pomonella*), rodents, dogs and other mammals (see reviews Majeikite 1978, Il'enko and Krapivko 1989)

Within any mammal (including *Homo*) population's subgroup there are real individual variations in radiosensitivity. In the extreme cases of those carrying the ATM gene for *ataxia telangiectasia*, there is extreme radiosensitivity and tendency to leukemia, lymphoma and some solid tumours. The gene which is defective is associated with a DNA damage sensor protein. Although the condition is rare and the gene recessive, there is evidence which suggests that there is increased risk of cancer from radiation in the larger sub group which is heterozygous with respect to the ATM gene, about 6% of the population.

The existence of radiosensitive group variations is in fact seen in radiotherapy patients. From the foregoing it suggests that ethical considerations demand setting permitted radiation exposures at levels where people who are radiosensitive are protected rather than basing the limits on some standard man concept. This is another area where the releases of radionuclides to the

environment, where there are indiscriminate exposures, demands an ethical reassessment.

9.6.6 Hormesis response

A number of animal and *in vitro* studies have been cited as evidence that small doses of radiation have a protective effect named 'hormesis'(from the Greek *hormein*, 'to excite'). In this dose response, the curve dips as the radiation dose is first increased. The lowest dose controls thus exhibit a greater health deficit than those who are given slightly greater though still low doses, although as the dose is increased the curve rises again and the effect increases. The curve is shown in Fig. 9.4

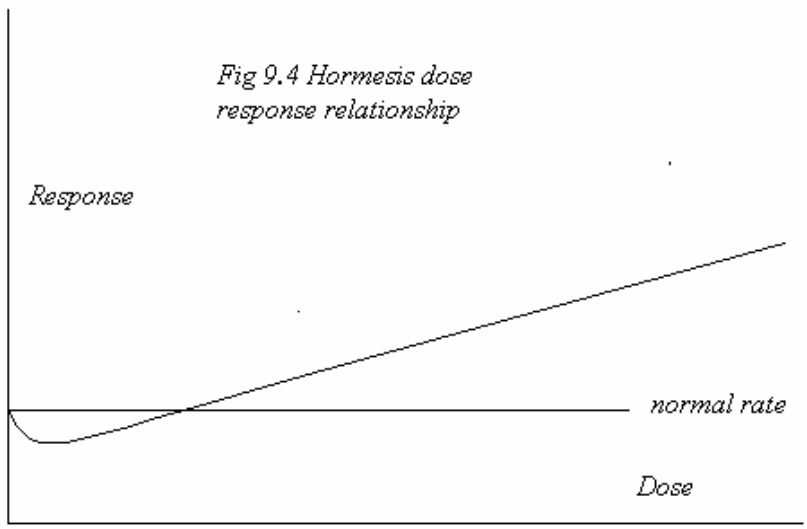

Fig 9.4 Hormesis dose response curve

The explanation given for this effect is that at the lowest doses, increased efficiency of cell repair is induced by the radiation exposure. Thus, as dose increases, the radiation first has a protective effect, with a reduction of cancer yield. The Committee has considered hormesis and its supporting evidence carefully and concludes that such a process is possible. Effects appear to occur at intermediate dose ranges (i.e. above 20mSv) and may have a number of explanations:

1. Sensitive sub-groups of cells are being killed rather than mutated.
2. Immune system surveillance is being potentiated in the short term (with possible long term detriment).

3. In the case of high background effects, foetal and infant death of sensitive individuals results in selection for radiation resistance. This is a population version of the cellular effect in (1) above.

It may be that inducible repair efficiency exists, comparable with other inducible systems, like Haemoglobin-Oxygen dissociation at altitude or suntanning in tropical climates. This may be one explanation (among others) for the lack of variation in cancer rates between regions of different natural background. However, the existence of radiation-inducible repair means that the repair systems themselves may be open to attack, also by radiation (see below). In addition, the existence of such a process may have other complications. The question needs to be asked why, if repair replication were inducible in this way, would any species not automatically evolve to the highest state of repair efficiency and remain permanently in that state? The answer may be that if cells were induced into a state of high sensitivity for repair replication, then the cell line would undergo a greater rate of replication throughout the period of stress, and since it is now well established that non-specific ageing is a function of the total number of cell replications, the consequence of the short-term advantage conferred by hormesis is probably a long-term loss of viability due to accumulated DNA damage caused by high numbers of replication-copying processes.

It may be, however, that some of the hormesis evidence results from an artifact. If the dose response in the low range follows a biphasic curve, all that is needed to show an apparent hormetic effect is to leave out the zero-dose/zero-effect point. It may be that because deductive conclusions from high-dose experiments could not be squared with the possibility of such variation in this low-dose region, either the points were interpreted as scatter or they were forced into a hormesis dip by leaving out the lowest dose responses as outliers.

The Committee provisionally concludes that hormesis may exist, but if it does exist its long-term effects are likely to be harmful for the reasons detailed above. The Committee recommends that no consideration should be made of hormesis in respect of radiation protection.

9.6.7 The Committee's conclusions on dose-response relationships

The Committee has agreed that the ICRP's linear no threshold assumption is invalid except as an approximation which may hold over a small range, and indeed the Committee employ such a relation in the low-dose region as a matter of pragmatism. There is not sufficient evidence to show that there is a universal dose-response relation for all types of exposure and all end points, and to assume such a function is an example of a fatal reductionism. However, there are good reasons for assuming that effects in the low-dose range from zero dose to about 10mSv (ICRP) are likely to follow some kind of supralinear or fractional exponent function. Since there is good theoretical and empirical evidence for the existence of biphasic dose-response relationships, the

Committee strongly recommends that no epidemiological finding should be dismissed on the basis that it does not conform to a continuously increasing dose-response relation of any form.

9.7 Factors affecting the biological efficiency of radiation action

The damage caused by radiation exposure has been shown to be a function of ionisation energy density. However, the cell is not a passive target in this process, nor is the organism. Since the discovery in the 1960s that cells repaired radiation damage the emphasis of research has been to examine how, and what factors augment or inhibit such repair. For the overall scheme of radiation damage, outlined in Table 9.1, there is a set of damage-inhibition systems based on cellular and systemic responses. Thus, for stochastic end points like cancer, there are a number of processes involved, shown in Table 9.3. Discussion of all the factors listed in Table 9.3 is beyond the scope of this work. The factors are listed in order to show that the emphasis on initial radiation-damage processes implicit in the ICRP system is only valid for high doses delivered externally. In the low-dose range, other factors are of major importance in deciding the outcome of any exposure. The response of the cell to low-level non-lethal radiation exposure is a critical system in this progression. The discovery of the system of cellular responses to sub-lethal exposure has an important consequence which was pointed out by Busby in 1995. If cells in the repair-replication cycle are significantly more susceptible to radiation exposure than cells which are not replicating then this phase of the cell lifespan therefore represents a window of opportunity for mutation. If circumstances could be arranged to irradiate in this window, a hazard enhancement would occur as we shall discuss below.

9.8 The Second Event Theory

It was pointed out that most cells in a living organism are in a non-replication mode, sometimes labelled G0. These cells are contributing to the organism as part of the normal living process and do not need to replicate unless there is some signal requiring it, perhaps because of tissue growth, damage or senescence. Throughout the growth and lifespan of individual organisms there is a constant need for cellular replication, and therefore there is always some small proportion of cells which will be replicating: the magnitude will naturally depend upon the type of cell. When cells receive the signal to move out of stasis or G0, they undertake a fixed sequence of DNA repair and replication, labelled G0-G1-S-G2-M, with various identifiable check points through the sequence which ends in replication M or Mitosis. The period of the repair replication sequence is about 10 to 15 hours and the sensitivity of replicating cells to damage including fixed mutation is extremely high at some points during this sequence. In the case of Chinese Hamster Ovary cells, for external

low LET radiation there is a 600-fold variation in the sensitivity for cell killing over the whole cycle though the sensitivity for mutation has not been studied.

If there is large variation in sensitivity for mutation over the cell lifespan, what follows? Although naturally dividing cells may accidentally receive a radiation 'hit', this process can be modelled by averaging over large masses of tissue, even if the dose response curve is not linear. However, unplanned cell division, preceded by DNA repair can be forced by a sub-lethal damaging radiation track: this is one of the signals which push the cell out of G0 into the repair-replication sequence. It follows that two hits separated by about ten hours can firstly generate a high sensitivity cell and then hit this same cell a second time in its sensitive phase. This idea, the 'Second Event Theory', is described, and supporting evidence for it advanced in Busby 1995 and its mathematical description has been approached slightly differently in Busby 2000. It has been the subject of some dispute by the UK's NRPB and has been discounted by ICRP 2007 on the basis of some questionable reasoning presented to the CERRIE Committee by Roger Cox, ICRP's Deputy Chair.

Developments in micro techniques have allowed the emergence some evidence that supports the two hit idea. Miller *et al*, (1999) in a consideration of Radon exposure risks, have been able to show that the measured oncogenicity from exactly one alpha particle hit per cell is significantly lower than for a Poisson-distributed mean of one alpha particle hit per cell. The authors argue that this implies that cells traversed by two alpha particles or more contribute most of the risk of mutation, i.e. single hits are not the cause of cancer. However, as yet, the differences in effect between two hits delivered within the space of a few minutes and within the cell cycle repair period of about 12 hours have not been compared.

There are three types of internal exposure which would be expected to result in an enhancement of risk from any Second Event source. The first is due to sequentially decaying radioisotopes like Strontium-90. Following an initial decay from a Sr-90 atom bound to a chromosome, the second decay from the daughter, Yttrium-90, whose half-life is 64hrs can hit the same cell within the time period of the induced replication sequence with a probability that is simple to calculate. The same dose from external radiation has a vanishingly small chance of effecting the same process since, in this case, the target DNA is within a few tens of nanometres of the source. The second type of Second Event exposure is from micron or sub-micron sized 'hot particles'. If lodged in tissue, these will decay again and again increasing the probability of multiple hits to the same cell inside the ten hour repair-replication period. Finally, high Z elements bound to DNA will catalytically increase the ionization rate at the DNA through the secondary photoelectron effect.

The Committee is aware of the speculative nature of these proposed mechanisms, but in view of their plausibility assumes that this kind of effect cannot be ruled out, and recommends further research in this area.

Table 9.3 Factors affecting the progression of radiation damage to cancer

Contributions to final cancer	**Factors**
Increasing density of ionization	1. Radiation quality; α,β,γ 2. Auger emitters, weak decays e.g. Tritium 3. Electromagnetic field interactions.
Increasing track density in space	1. Increasing dose 2. Internal exposure from point source 3. Internal exposure from hot particle 4. Internal exposure from immobilised sequential decay 5. Concentration of ionic radionuclides at interfacial layers by adsorption 6. Concentration of radionuclides in organelles by biochemical affinity
Increasing track density in time	1. Internal exposure from point source 2. Internal exposure from hot particle 3. Internal exposure from immobilised sequential decay 4. Concentration of ionic radionuclides at interfacial layers by adsorption 5. Concentration of radionuclides in organelles by biochemical affinity
Increased replication rate of cell	1. Cell type 2. Prior exposure/prior damage 3. Electromagnetic field 4. Growth rate of individual (e.g. children) 5. Concentration of replication promoters including radiation
Position in cell cycle	1. Prior exposure/prior damage 2. Electromagnetic field
Decreased repair efficiency	1. Genetic identity 2. Prior exposure/prior damage 3. Antioxidant status/repair enzyme status 4. Concentration of repair system poisons
Decreased immune surveillance	Various, including prior exposure
Decreased replication inhibitory field	1. High local doses 2. Hot particles

9.9 Other factors affecting cancer expression

9.9.1 Immune surveillance

Although it is now generally accepted that cancer has its origin in a single mutation event, there are a number of factors involved in the progression from this event to clinical expression. The most obvious of these is the system of immune surveillance, which prevents tumours from progressing. The suppression of immune response by organ transplant drugs or cytostatic drugs is associated with enhanced risk of cancer. Irradiation of the organism is a well-documented cause of immune-system suppression, not only from high-energy ionising radiation but also ultraviolet radiation. This aspect of radiation exposure has not been addressed by ICRP but is believed by Sternglass and others to provide a mechanism for low-level radiation effects. Thus low-efficiency immune-system response would augment the probability of developing cancer following an exposure, suggesting a mechanism which would cause enhancement of hazard if an individual who had been exposed were also to be chronically exposed over the period following the initial exposure.

9.9.2 Cell proliferation fields

Recent theories of cancer expression (Sonnenschein and Soto, 1999) address the finding that transplanted cancer cells do not grow in non-cancerous tissue whilst normal cells transplanted into cancerous tissue become cancerous. These researchers propose the existence of a cell-communication-field effect which requires a certain threshold number of genetically damaged cells to occur before cancer can develop. The arguments are based on the theory that the default state for cells in metazoa is, like metaphyta, proliferation: it follows that there has to be a permanent inhibitory signal. Sonnenschein and Soto assume that this involves various components of cell-cell communication collectively termed a ‘field’. If this is found to be generally so then the effects of high local doses, as occur in the region near hot particles, may be particularly effective in causing cancer, since the damaged cells are all close to one another. That such fields exist has been shown recently by the discovery of the ‘bystander effect’ in which genomic instability is found to occur in cells which are close to the cell which is traversed by a radiation track but which do not themselves receive any direct track traversals. Furthermore, the phenomenon of ‘field cancerization’ whereby certain cancers seem to start from independent loci in the same site (e.g. larynx cancer) supports the idea that cell communication within a community of cells is a critical component of cancer expression (Boudewijn *et al* 2003).

9.10 Biochemical and biophysical effects

The concentration of certain radioisotopes in organs through biochemical affinity is incorporated into the ICRP scheme only through the organ weightings. Thus it is accepted that Iodine concentrates in the thyroid and that this represents a hazard in terms of thyroid cancer and other thyroid conditions. However, arguments from chemical considerations should also be applied to all isotopes, and extended to concentration effects at the molecular level, as well as the organ level. For example, Strontium has a particular affinity for the DNA phosphate backbone: indeed, Strontium Phosphate co-precipitation is a method of choice in genetic research for removing DNA from solution. Thus exposure to the isotopes Strontium-90 and Strontium-89 should result in decays within the DNA itself. This effect should extend to isotopes of Barium also, which are also common environmental contaminants from nuclear processes.

There is also the 'Trojan Horse' exposure to a sequentially decaying isotope whereby the isotope enters a system with one chemical identity and on decay changes to a different chemical species which is also radioactive. An example here is the series Sr-90/Y-90. The radioactive decay product of the dipositive ion Sr-90 decay is a tripositive Y-90 ion. The Committee is concerned that such a sequence might result in accumulation of Y-90 in parts of the organism (e.g. the brain) where there are biological filters based on ionic strength or valency and that this might result in enhanced local doses.

A similar enhancement of local dose would occur as a result of adsorption of radioactive ions (e.g. Cs-137) at an interface. The positive ions involved in nervous system signalling collect at synaptic junctions and the similar concentration of radioactive species with the same chemical group affinity would increase the local dose.

9.11 Transmutation

One mechanism which is entirely absent from ICRP's deliberations results from the effect of the radioactive decay process changing one atom into another. There are three common radioisotopic pollutants where this effect is likely to have serious consequences: Carbon-14, Tritium and Sulphur-35. All three are major components of enzyme systems and critical to the processes which are fundamental to living systems. The macromolecules which are the operators of living systems—proteins, enzymes, DNA and RNA—depend upon their tertiary structure, or shape, for their activity and biological integrity. Alteration of this shape results in inactivity of the macromolecule. This inactivation could in principle be effected by the sudden transmutation or alteration of one atom in the macromolecule. Since the molecular weight of these macromolecules is usually greater than 100,000 it is clear that incorporation of one atom (of e.g. C-14 which decays to Nitrogen) may result in an enhancement of effect of many thousand-fold. The isotope Tritium is a form of Hydrogen and the biochemical processes in living systems depend on the weak bonds called

Hydrogen Bonds which bridge and support all enzyme systems and hold together the DNA helix. The sudden decay of such a Tritium atom to Helium (which is inert and does not support chemical bonds) may have a catastrophic effect on the activity and normal processing of such macromolecules. Hydrogen bonded in these systems is easily exchangeable and will exchange under equilibrium conditions with Tritium Oxide, or tritiated water, the normal form of this isotope in the environment. There is also some evidence that Tritium may be preferentially taken up in some systems. This needs to be confirmed by further research. Sulphur is also an important component in macromolecular proteins, forming disulphide bridges which support tertiary structures.

The Committee feels that this area has received insufficient attention and that more research is required to establish the risks to biological systems from transmutation effects. Although this sentiment was implicit in the review of internal radioactivity effects by Gracheva and Korolev published in 1980 nothing has followed.

9.12 Increase of dose due to particle in placenta and genomic signal transfer to foetus.

The size of particle which may be transferred across the placenta has not been determined. Recent unpublished research suggests that particles as large as 100nm (0.1μ) pass across the placenta into the foetus. For early developing foetuses, the local doses from particles of Plutonium Oxide or other actinide alpha emitters will be massively high and may result in a range of effects from foetal death and early miscarriage to effects in childhood. This is a case where the biological end-point may result from a very low-probability, high risk event. Plutonium particles are common contaminants in the atmosphere near the Irish sea and other areas close to nuclear plants.

Even if the particle may not transfer across the placenta, it is known that there are genomic instability signal molecules which are quite small in protein terms and are one origin of the bystander effect whereby cells remote from those receiving the radiation damage are caused to increase their rate of mutation. Thus genomic and bystander effects are in principle mechanistically likely to transfer from the placenta to the foetus, and indeed the transgenerational genomic instability effects have now been seen in Chernobyl-affected populations and in laboratory animals (ECRR2009).

10
Risk of Cancer following Exposure Part 1: Early Evidence

10.1 Basis of the ECRR risk model

Following the publication of ECRR2003, the French IRSN (and others) criticized the model for having failed to explain its basis in scientific evidence. The evidence which the Committee used as a basis for its new model of risk arises first from human epidemiology, next from a number of human, animal and cell studies and finally on knowledge of the physico-chemical and biological nature of the interactions between radiation and molecules at the cell level. It is not primarily physics-based (and this is the essential difference from ICRP) but instead begins inductively with the epidemiological evidence and then explains these in terms of the interactions between radiation and tissue at the molecular level as dilute solution biophenomena.

Primarily, the model is based on empirical data on internal exposures to fission-product radionuclides and Uranium in the fallout from atmospheric weapons tests. These tests, which peaked in 1959-63 were the first experiment on the effects of human exposure to internal radionuclides, since the radioactivity was globally dispersed. It was an experiment with an outcome that no-one examined. There was never any systematic study at the time, possibly because the World Health Organisation was rapidly constrained from any such effort in 1959, when the early health effects began to appear. WHO, as we previously noted, were made to sign an agreement with the International Atomic Energy Agency, which left research to IAEA, an agreement which is still in place. But behind the scenes, away from the public, it was clear to authorities that the fallout was beginning to kill people. Early suggestions that this might be so were quickly denied by reference to the Japanese A-Bomb studies; there were cover-ups at the highest level (Medical Research Council 1957). Nevertheless a Test Ban was signed in 1963.

Although weapons fallout was globally dispersed, it was not uniformly dispersed, and was greater in the northern hemisphere than the southern. It was also greater in regions of high rainfall than in regions of low rainfall, often by significant amounts. The overall doses, as assessed using the ICRP model, were calculated however, and tabulated by UNSCEAR. The contamination was measured in a number of countries and results were published. One country where excellent contamination data was available from the 1950s continuously to the present day was the United Kingdom. This included data on contamination by individual radionuclides in England and separately in Wales. These are also two countries in the UK which have functional cancer registries which collected incidence data from 1974, although separate mortality registers are available for causes of death back to the 1930s in the UK. Wales is on the Atlantic coast of the UK and the fallout in Wales and therefore (because of higher rainfall) the measured contamination, and thus doses (dominated by the

isotope Strontium-90) were twice to three times greater than those from the fallout in England.

It was a straightforward matter to compare the trends in the age standardized incidence of cancer in Wales and England, countries with similar genetic populations and similar lifestyles, to see what effect the higher level of exposure from weapons fallout produced. The effect was startling (Busby 1994, 1995, 2006). The age standardized cancer incidence trend in the two countries was similar. The trend was parallel and flat from 1974-1979 when the Wales rates began to climb relative to the England rates. By 1984, the rates in England began to rise, but by then the Wales rates had increase by about 30%. The peculiar shape of the trend in Wales exactly followed the earlier fallout exposures, even to the discontinuity which occurred in 1959 from the partial test ban treaty which resulted in a sharp fall in radioactive precipitation. The temporal correlation of the cancer trend on the earlier trend in fallout exposure, modeled as Sr-90, was highly statistically significant. Comparison of the doses and the predictions of cancer excess at those doses by the ICRP model gave an error of 300-fold in the application of the ICRP predictions (Busby 1994, 1995, 2002, 2006). This level of error (2 to 3 orders of magnitude) was to appear again and again in studies of internal fission product exposures. Recent powerful confirmation comes from the study by Tondel *et al* (2004) of cancer in northern Sweden after the Chernobyl accident where it can be shown that the error involved is upwards of 600-fold. This will be discussed in the following Chapter. Support also comes from the increased cancer rates in Belarus reported by Okeanov in 2004.

It is one thing to establish that there is an error in the application of the ICRP model to internal exposures; it is quite another to partition the error among the various isotopes that make up the fallout. Are they all equally mishandled mechanistically? Are some more dangerous than others? The doses from fallout as tabulated arise from the ICRP model in which Sr-90 confers the greatest dose, because of its relatively long half life (29 years) and its ability to store in bone as a Calcium substitute. It thus provides ‘energy per unit mass’ for longer. But on the basis of the Committee’s approach, Uranium might also represent a significant hazard. Yet Uranium levels were not measured nor were the doses from Uranium assessed. At this stage, it is necessary to bring in evidence on individual radionuclide risks from other sources, and this has been the Committee’s approach. If the main fallout exposures were to Cs-137, Sr-90 and Uranium, then it seems plausible that Cs-137 doses may be discounted for two reasons: that the element has a short biological half life and that, from its chemical nature, it is uniformly dispersed in the body. It can thus be modeled as an external radiation hazard for reasons given in Chapter 6. In animal experiments Cs-137 has far lower effect than Sr-90 on genetic damage (Luning and Frolen 1963). The same considerations apply to Carbon-14 (uniformly dispersed), Tritium (short biological half life) and many short half life radionuclides.

In the following two chapters, which review the main evidence, the Committee briefly presents the studies and results which inform the position that it has adopted. This chapter deals with the situation up to and including the effects of global weapons fallout in the period which ended with the Atmospheric Test Ban Treaty in 1963. Chapter 11 begins with evidence from the nuclear plant leukemia and cancer clusters to the present day. For reasons of space, these chapters are not a comprehensive review of all the evidence.

10.2 Specificity

The Committee has decided to address the risks of internal and external irradiation separately, for reasons which have been discussed. However, it is clear that the evidence upon which the risk factors depend are from real-world situations in which it is rare that exposure is entirely external or entirely internal but is usually a mixture of both. If internal irradiation carries a significantly higher risk than external, then it is easy to see that the external irradiation risk factors, deduced from a study of populations who had received a large external dose compared with internal dose to the same populations, would show higher yield than those obtained from the same dose delivered purely externally and that this discrepancy would increase as the overall proportion changed to include greater internal dose. For the Hiroshima LSS study, for example, at the lowest doses, such an effect would show itself as a supra-linear dose response or some other form of high response at low dose, although other factors will contribute to the empirical result. It is of interest that the US-directed studies of the Hiroshima survivors consistently denied that there was any internal component to the exposures received by the study group because the bombs were exploded in the air. However, measurements made since then have showed presence of Plutonium and Caesium in soil near Hiroshima and recently fallout isotopes from the Hiroshima bomb have been identified in ice cores from the Arctic. These findings may explain the puzzling increase in leukemia in the control group relative to all Japan recorded in the first studies. Evidence has now emerged that the ABCC failed to include early leukemia's in the bombed towns in the LSS and failed also to report the existence of fallout in the towns and also rates of general illnesses which would have falsified their ultimate conclusions: this is particularly true of non-cancer illnesses and heritable damage (Kusano 1953, Sawada 2007). It should be recalled that non-cancer illness affects cancer statistics and radiation cancer epidemiology since an individual that dies of any disease but cancer below the age of about 50 is not available to die of cancer above that age when cancer rates begin to increase exponentially.

The Committee has nevertheless decided to treat studies of mainly external irradiation, where the external dose, conventionally modelled, is more than 100 times the internal dose (as the ICRP expresses these) as external risk studies and to accept that some of the discrepancies and anomalies revealed

may have their basis in internal exposures. This approach has the advantage that it results in values for external irradiation risk factors, which may be used for radiation protection purposes in those pure external irradiation scenarios where advice is necessary.

10.3 Base studies of radiation risk.

The studies given in Table 10.1 are the main ones which underpin the risk factors used in the models of the ICRP and which define the present radiation protection regime. It is clear that these are almost exclusively studies of external irradiation risk, and with the exception of the Hiroshima study, are all comparisons of purely externally irradiated subjects with controls who were not irradiated. The risk factors for cancer which were obtained from these studies are largely in agreement one with the other, and the Committee believes therefore that for external acute irradiation and cancer as an end point, these risk factors are not likely to be wildly inaccurate.

The most recent data on late cancer effects in the Hiroshima LSS show that the yield of cancer continues to exceed that predicted by previous risk factors. The independent analysis by Gofman of the LSS data, Stewart's findings relating to the homogeneity of the LSS study populations and the work of Padmanabhan on the choice of control group suggest that the risk factors for cancer given by the LSS study may be in error by as much as 20-fold. The Committee is aware, however, that the LSS is based on an anomalous population exposed to both external and internal radiation and is not an ideal basis for obtaining pure external risk factors. The lifetime absolute risk of fatal cancer of 0.2 per Sievert, chosen by the Committee, represents a decision based on a review of all external irradiation studies.

Table 10.1 Summary of studies used to determine risk factors from ionising radiation by ICRP and others but used by ECRR to determine external exposure risk factors for cancer and leukemia.

Study	Persons	Doses (Gy)	Regime	Controls	Comments
1. Hiroshima Lifespan study (LSS)	91,000	0-5 high	Single acute	In city 'unexposed'	Abnormal population; bias in controls; late effects still developing
2. UK Ankylosing spondylitis	14,000	3-4 high	Acute	Average population	X-rays
3. Cervical cancer patients	150,000	high	Chronic	Average population	Radium capsule
4. Canadian fluoroscopy	31,700	0.5-1.2	Several acute	Unwell control	Unwell group, X-rays
5. Post partum mastitis	601	0.6-1.4	Several acute	Untreated mastitis	Small study, X-rays
6. Massachusetts fluoroscopy	1,700	high	Several acute	Average population	Highly fractionated, X-rays, small study

10.4 Natural Background Radiation

The Committee has examined the evidence regarding human health indicators including cancer and congenital illness with variation in natural background radiation exposure. The main studies which contribute to the understanding of the health consequences of living in high background radiation areas are given in Table 10.2

Table 10.2 Variation in cancer and other effects in areas of high Natural Background radiation

Area of study	Number studied	Exposure	Cancer increase?	Chromosome defects?
1. Austria	122	1-4mGy (γ).01-16mGy (α)	predicted	Yes
2. Finland	27	Radon in water	not investigated	Yes
3. Iowa	111 towns	Ra-226 4pCi/l ;controlled	+24% bone cancer.	Yes
4. Brazil	12,000	Monazite: 6.4mSv/yr.	No	Yes
5. Kerala, India	70,000	4 mGy/yr.	Disputed	Yes
6. China Yanjiang	70,000	3-4mSv/yr.	Apparently not	Yes
7. Brittany	16000	γ-background	+43% (+132% stomach cancer)	not examined
8. Iowa	28 towns	Ra-226	+68% more lung cancer	Yes
9. Japan	All areas	γ background	+stomach and liver cancer	Not investigated
10. Scotland	All areas	γ background +0.15mGy	+ 60% higher leukemia	Not measured

For a number of reasons, it is uncertain how the results of these studies can inform discussion about risk from radiation exposure. First, for many of these studies, the populations suffer stresses associated with living in the Third World where cancer is not a major cause of death owing to earlier competing causes and the generally shorter lifespan. In addition, natural selection for radiation resistance over a long period may be expected to confound any attempt to find a suitable control group: thus the repair efficiency for cancer-inducing lesions in genes might be expected to be higher in the exposed populations than the controls. In addition, the considerable amount of evidence which shows that different populations have different genetic susceptibility to cancer of different sites makes it impossible to draw any universally applicable conclusions from background radiation studies. There are also confounding geographical factors relating to the levels of man-made radioactive contaminants in high background regions. In Table 10.3 are listed possible confounding components of health indicators in areas of high natural radiation.

Table 10.3 Difficulties with interpretation of natural background studies

Problems with comparisons of health indicators across areas with high and low background
1. Competing causes of death in disadvantaged populations
2. Difficulty in establishing rates due to lack of health data
3. Difficulty in finding genetically comparable controls
4. Development of induced responses in study group in their lifetime
5. Natural selection for radiation resistance in populations over generations
6. Variation in fallout contamination due to rainfall effects
7. Lack of epidemiological strength for the range of external dose

Despite these difficulties, it is clear from all the studies that chromosome aberrations and breakages are present in populations exposed to high natural background radiation. This is often associated with other flags for genetic damage, like Down's syndrome frequency. Since cancer is a consequence of genetic damage, evidence of increases in chromosome damage would suggest that the cause of such damage would also be a cause of cancer increases if the group lifespan was greater. Increased cancer risk does not seem to be a general observation, although a number of studies have demonstrated increases in cancer rates for some cancers in high background areas. However, it may be that populations who had developed in conditions where such damage had occurred might enjoy increased evolutionary resistance to cancer as a consequence of the death before birth of sensitive individuals or even increases in resistance to cancer at the metabolic level bought at some expense to the overall lifespan.

There is also the problem of epidemiological strength over the ranges of dose found within the studies themselves. If this range is between 1 and 5 mGy delivered annually from natural radiation (mainly external gamma) then according to the ICRP risk model for fatal cancer (which ECRR largely accepts for external irradiation) the radiation component of the cancers after a 50 year accumulated dose would increase from 0.6% to 3%, which would be difficult to show.

The Committee concludes that evidence from this area of research is not useful for radiation protection purposes. In particular, arguments which are based on comparisons of cancer incidence across areas of high background and extrapolated to populations living in low background areas are inadmissible as evidence of low risk from low level exposure to fission-products or TENORM.

10.5 Cancer and Global Weapons Fallout

Overall, the dominating source for radioactive contamination from human activity is the global fallout of debris from the atmospheric nuclear bomb tests conducted in different parts of the world between 1945 and 1980. In total, 520

nuclear explosions were carried out, with periods of the most intensive testing in the years 1952-4, 1957-8 and 1961-2. 78 percent of the activity released by these tests has been spread over the earth, contributing the major component of the exposure to fission products and transuranics suffered by living creatures. These substances are now universal environmental contaminants and also universal in the cells of living systems, yet very little research has been aimed at investigating their possible health effects. Many of the isotopes are periodic-table-group mimics of elements which are utilised by living systems; they therefore become incorporated into cells and organs.

The period of major atmospheric weapons testing and fallout exposure which ended in 1963 with the Kennedy-Kruschev test ban was the first occasion that the health effects of such internal exposure could be assessed. However, very little research was undertaken and very few studies were published either drawing attention to or discounting the existence of any consequences. Suggestions by Sternglass and others that the fallout had caused increases in infant mortality were ridiculed and attacked. This climate of denial was probably due to the secrecy and control associated with Cold-War politics and, as has been pointed out above, this was institutionalised in 1959 in an agreement between the World Health Organisation (WHO) and the International Atomic Energy Agency (IAEA) which had the effect of giving the IAEA a power of veto over WHO research into radiation effects.

Thus, although during the weapons test period there was enormous activity in the fields both of cancer research and radiobiology, there is only a small number of reports and studies which throw useful light on the consequences of exposure to weapons fallout. Those that do exist are summarised in Table 10.4.

According to UNSCEAR, using ICRP models, the cumulative northern hemisphere internal fallout dose over the period 1955-65 varied between about 0.5 mSv to doses of between 1 and 3mSv in parts of Europe where high levels of rainfall caused increased deposition. The trend in dose showed a sharp increase between 1958 and 1963 due to increasing testing of megaton thermonuclear bombs. For internal isotopes the cumulative trend showed the same sharp increase and reached a plateau in 1965, after which the trend fell slowly (through biological loss and physical decay) by about 20% to the value in 1999. The internal dose was dominated by two isotopes: Caesium-137 with a half-life of 30 years and Strontium-90 with a half-life of 28 years, although other more active isotopes gave high doses at the time. Contamination by Uranium does not seem to have been reported. Details of the isotopes and doses calculated on the IRCP basis are summarised in UNSCEAR 1993 and UNSCEAR 2000 and the main components of exposure are given in Table 10.5

Table 10.4 Fallout cancer studies considered by the ECRR

Study Group	Exposure doses	Finding	Notes
1. Marshall Islanders	External + Internal: 1-10Gy	Thyroid cancer, leukemia, still birth, miscarriage.	Only 200 persons Controls also contaminated
2. U.S. Utah test contamination	External - Internal 1Gy	+ Thyroid + Leukemia	Dose unknown / Arizona controls
3. Utah test: Mormons (C.Johnson)	As above	Leukemia (4x) Thyroid (7x), breast (1.7x) bone (11x) etc.	Dose unknown
4. U.S. Leukemia *vs.* global fallout. (V.E.Archer)	Internal <NBR	Leukemia correlation with Strontium-90 levels in US	Highlights error in ICRP risk factors
5. Scandinavia: Leukemia vs. global fallout. (Darby et.al)	Internal < NBR	Found little correlation with childhood leukemia in Scandinavia .	Unconvincing analysis; questionable protocol
6. UK leukemia and rainfall. (Bentham 1995)	Internal <NBR	Found a significant correlation with childhood leukemia and rainfall in UK.	Disagrees with Study No 5
7. US fallout cohorts (RPHP: Gould and Sternglass 1995 -)	Internal <NBR Strontium-90 cited	Various cancer excess risks in fallout exposed birth cohort in USA	Present cancer epidemic predicated on the fallout
8. US NAS cancer study	Iodine from Nevada tests	+thyroid	
9. UK and Wales female breast cancer (Busby 1995, 1997)	Strontium-90 1mSv cumulative dose	Cohort effect in breast cancer	Breast cancer epidemic predicted and explained
10. UK and Wales all cancer incidence 1974-90 (Busby 1995-2002, 2006)	Internal Strontium-90 1mSv cumulative dose	Significant correlation in time lag study; all malignancy	Regression analysis gives error in risk factor of 300-fold

Table 10.5 UNSCEAR 1993 calculations of fallout average committed effective doses in person Sv to world populations. Doses were calculated using ICRP models and would be much larger using the ECRR model where internal doses carry various weightings

Period	External	Ingestion	Inhalation	Total
1945 – infinity	2,160,000	27,200,000	440,000	29,800,000

. [*UNSCEAR 1993 Table 11*]

The Committee interprets the evidence from the studies it has considered to suggest that the exposure to global weapons fallout has had a significant impact on human health. This impact has been both immediate, causing infant mortality at the time (a subject which is reviewed in the next chapter), and protracted, resulting in increases in cancer, leukemia and other diseases of genetic origin (including coronary heart disease) with a delay between exposure and the clinical expression of disease. In reaching this conclusion, the Committee has been impressed by the lack of evidence as to the origin of the global cancer epidemic which began in the period 1975-85. Cancer is now widely seen, in the medical community, as a genetic disease expressed at the cellular level, and both early and recent research have supported the idea that the origin of the disease is essentially environmental exposure to a mutagen. If cancer rates began to increase sharply in the period 1975-1985, and since research has shown that the disease is known to lag the exposure by 15-20 years, clearly, the origin of the epidemic must be the introduction of some cancer-producing mutagen quite suddenly into the environment in the period 1955 to 1965. The identification of this mutagen with radionuclide pollution from weapons fallout is persuasive. In addition, the variation in cancer incidence rates across regions of high and low rainfall and deposition points to radiation as the main cause of the cancer epidemic.

Only two groups appear to have studied this possibility: the Radiation and Public Health Project (RPHP) of Gould, Mangano and Sternglass in the US and the Green Audit group of Busby *et al*. in the UK. The latter has used cancer incidence in England and Wales to examine variation across similar populations with cumulative exposures to the isotope Strontium-90 of between 0.2 and 1 mSv and has been able to show that variations in the fallout exposure are highly correlated with later cancer incidence (R = 0.96). Green Audit researchers have shown that this demonstrates a 300-fold error in the ICRP risk model. Both groups are engaged in examining geophysical factors like estuaries and river valleys, where fallout becomes concentrated, and have shown that these areas consistently show excess risk for cancer and leukemia. The RPHP researchers have provided evidence that breast cancer is caused by Strontium-90 in fallout and downwind of nuclear sites, and are presently examining cancer rates in relation to Strontium-90 in measurements they have

made on deciduous teeth. Preliminary unpublished results of this study show a significant correlation between levels of Sr-90 in the teeth and cancer incidence later in life (Mangano 2009).

In addition to the increases in all cancers which have occurred since the peaks in fallout, there have also been some specific cancer sites which have shown notable increases. Significant and unexplained increases have occurred in female breast cancer and male prostate cancer. Both these diseases are caused by radiation. The Committee has noted the evidence which links breast cancer to Strontium-90 published by Sternglass *et al.* and the cohort studies of breast cancer mortality reported by Busby 1997, both of which provide persuasive evidence about the origin of recent increases in the disease. Prostate cancer has also been shown to have displayed its highest incidence in Wales following the fallout trend by about 15 years. The excess prostate cancer risk found by Roman *et al.* in nuclear workers who were monitored for internal contamination suggests an error of up to 1000-fold in the risk model used by the ICRP (Atkinson *et al*. 1994).

Table 11 of the 1993 report to the United Nations (given above as Table 10.5) shows that the committed effective dose to world populations as a consequence of the weapons testing is just under 30,000,000 person Sieverts. From this dose the ICRP 2007 cancer risk factor of 0.05 per Sievert predicts a total yield of 1,500,000 cancers in the world population. UNSCEAR 2000 gives similar calculations for the committed effective doses from weapons fallout but the results differ significantly (are smaller) than those given in the 1993 volume.

Table 10.6 (from UNSCEAR 1993) shows committed effective doses to northern temperate latitudes (40-50 deg. N) from each of the main isotopes involved. For comparison the table also shows the total doses calculated using the proposed model of ECRR, which recognises excess risk from internal emitters. Use of the ECRR adjustment for internal risk using the ratios of external to internal isotopes given in Table 10.6 would increase the cancer yield from the 1990 ICRP value given above to more than 60,000,000 persons. The greater part of this yield would be in the 50 years following the exposure, and these cancer increases predicted are, of course, only too visible. This calculation is revisited in Chapter 13.

Table 10.6 The major isotopes contributing to human exposure from different routes after weapons fallout together with average committed effective doses to the population of northern temperate latitudes (40-50 deg.) from each isotope calculated by UNSCEAR using ICRP models. * denotes an isotope and route that ECRR consider hazardous and would weight. The final two rows compare doses based on ICRP and ECRR models (see 6.9 above). Uranium is not included in this calculation as exposures are not known

External	**Dose (μSv)**	**Ingestion**	**Dose (μSv)**	**Inhalation**	**Dose (μSv)**
Cs-137	510	Cs-137	280	*Pu, Am	81 (24300)
Sb-125	47	*C-14	2600 (26000)	*Sr-90	15 (4500)
Ru,Rh-106	70	*H-3	48 (1440)	*Ru-106	110 (5500)
Mn-54	93	*Sr-90	170 (51000)	*Ce-144	86 (4300)
Zr,Nb-95	207	I-131	79		
Ru-103	20				
Ba,La-140	25				
Ce-144	23				
Total ICRP	995		3177		292
Total ECRR	995		78440		38600

(based on UNSCEAR 1993 Table 9)

10.6 Childhood cancer, leukemia and global weapons fallout

One of the most alarming developments in the period following the use and testing of nuclear weapons was the sharp increase in leukemia and brain tumours in children, which together make up the main types of childhood cancer. The early increases in childhood cancer in the 1950s were so remarkable that governments began to ask if they were caused by fallout, and attention focused on the isotope Strontium-90, which was becoming a significant contaminant of milk. In the UK, the Medical Research Council was asked to study the hypothesis and, advised by Sir Richard Doll, reported that the Hiroshima findings ruled it out on the basis that the doses were too low. Despite this, uncertainty fuelled by the contemporary discoveries of Alice Stewart that low-dose obstetric X-rays caused increases in leukemia in the children resulted in the banning of atmospheric tests in 1963.

A 1993 study by Darby, Doll *et al.* of childhood leukemia and fallout in Nordic countries has often been cited as support for the contention that low-dose internal radiation is safe. This study spliced together (in a time series) cancer registry data on childhood leukemia from Denmark, Norway, Sweden, Finland and Iceland—countries with very different sized populations and different exposures to fallout. The trend in leukemia rates in the 0-4 year olds over the period of the study, 1948-88 apparently showed a modest increase from 6 to 6.5 per 100,000 between the periods 1948-58 and 1965-85, which bracket the peak testing period of 1958-63 when the dose to children was about

0.5mSv, conventionally modelled. However, close examination of the study revealed that the early period is represented by data from the Danish cancer registry alone. After 1958 all registry data was pooled from the five countries. Thus the study was flawed (CERRIE2004b, Busby 2006). Close examination of the pooled data from 1958 suggests the increase in leukemia in the 0-4 year olds is from about 5 per 100,000 to 6.5 per 100,000, an increase of about 30%. This is in fair agreement with a study of childhood leukemia mortality in England and Wales published by Bentham.

The leukemia incidence increase of 30% in the children exposed over the 5-year period followed a cumulative dose of between the 0.15mSv bone marrow dose received *in utero* and the 0.8mSv received between ages 0 and 4. This suggests an error in the ICRP risk factor of between 3 and 15-fold if no further excess leukemia occurred in this cohort and an error of between 40 and 200-fold if this excess risk continued throughout their lives. In the US, Archer examined leukemia increases following fallout from Sr-90 and showed a fairly consistent increase of about 11% across all age groups following his estimated dose of 1.3mSv to adults and 4mSv to children. If these doses are accurate, then this suggests a higher rate at lower doses in the European studies. As with Bentham and Haynes, Archer was able to demonstrate a clear variation in leukemia related to high, medium and low rainfall areas.

The Committee notes that the childhood leukemia rate in the UK has risen steadily following the development of routine X-ray examinations, the extensive use of radium in the dials of wristwatches in the period 1930-40 and the first releases of fission isotopes to the world environment, with a sharp rise in 1945. Childhood leukemia mortality trends in the period 1916-1950 in England and Wales correlate with data for world radium and therefore Uranium production. The doses from radium dial sources have never been established. Attempts by the Committee to examine another possible source of leukemia increases by obtaining data on the mobile X-ray systems which were universally used in the period 1950-1960 to screen for tuberculosis have so far been proved fruitless.

10.7 Echoes of the fallout effects in the following generation

The Nordic leukemia trends published by Darby *et al.* show a rise in rates across the period of maximum weapons fallout 1958-63. However, they also show a marked increase in the rates from 6.5 to 7.5 per 100,000 beginning in 1983. This step-like increase began prior to the Chernobyl accident and is remarkable. It may be seen clearly in most datasets and shows itself in data from Wales and also Scotland as two close peaks centred on the two years 1984 and 1988. It is possible that these are trans-generational echoes of the genetic damage caused to parents born in or around the years 1959 and 1963, some 25 years earlier.

The Committee has investigated this hypothesis further by examining a small dataset obtained from a leukemia charity. This records the year of birth of the parents of children in England diagnosed with leukemia. Analysis shows that the highest risks are in those children whose parents were born around 1960, suggesting that their exposure to weapons test fallout may be a significant factor. The UK government's medical statistics department has refused to release additional data on the birth years of parents whose children were born after 1981. A study examining childhood leukemia by cohort year of birth of parent was commissioned as part of the CERRIE process but it was cancelled when the Minister who set up CERRIE was sacked.

Also supportive of this hypothesis is some evidence from animal experiments. In 1963, Luning and Frolen showed that the offspring of male mice exposed to Strontium-90 suffered significant genetic damage which showed itself as foetal death due to development defects. The genetic damage was passed on to the next generation, two generations away from the exposure. A similar effect on leukemia was found by Setsuda *et al.* in 1962 after administering Sr-90 to albino rats and examining leukemia in the offspring. Such an effect may be expected in human disease also and is further discussed in Chapter 13.

10.8 Other fallout studies: the overall effect

Studies which have been used to assess the risk of exposure to weapons fallout in global populations and in downwinders are listed in Table 10.4. These suffer from various problems which are noted in the table, but mainly from the same problem experienced by the Hiroshima study—the difficulty of finding unexposed controls. This is significant if the dose-response relationship is not linear, since low exposure controls may show a higher yield of cancer than higher exposure groups, where cells (or the foetus) may be killed rather than mutated. Nevertheless, the overall picture which emerges from the consideration of all these studies is not a reassuring one in view of the quantities of material released in the fallout. Even on the basis of UNSCEAR/ICRP calculated doses and risk factors the predicted cancer yield is between 1.6 and 3 million extra cancers world-wide—hardly a trivial figure. The ECRR model predicts between 60 and 130 million extra cancers or an approximate 20-30% increase in cancer incidence rates in those populations exposed over the period 1958-63 in Europe. This rise is apparent in the data. ECRR also predicts a cohort effect increase in cancer in those born between 1958 and 1966 and is concerned about evidence (e.g. the leukemia data considered above) which suggests an increase in risk in their children also.

11
Risk of cancer following exposure Part II: recent evidence

11.1 Nuclear sites and their proximity

In 1983, a TV company discovered the first of the nuclear site childhood cancer and leukemia clusters at Seascale near the nuclear fuel reprocessing plant Sellafield (earlier 'Windscale') in West Cumbria. Following the confirmation of this by epidemiologists and after a government enquiry, the UK government set up two new Committees to (a) develop epidemiological surveillance methods for small areas and (b) investigate the origin of the leukemia excesses near nuclear sites. In the 15 years following the Sellafield leukemia cluster, similar clusters were established near the other two reprocessing plants in Europe, Dounreay in Scotland and La Hague in northern France. In addition, childhood leukemia clusters were reported for other nuclear sites which released radioisotopes to the environment, Aldermaston, Burghfield, Harwell and Hinkley Point and Chepstow in the UK, Kruemmel in Germany and Barsebeck in Sweden. Recently, a study of childhood cancer and leukemia by distance from all the nuclear sites in Germany from 1984 to 2004 unequivocally demonstrated the effect; in children aged 0-4 the risk was more than doubled. The authors of the study argue that the ICRP risk model has to be in error by at least 1000-fold to explain this finding (Kaatsch *et al* 2007, Spix *et al* 2008). The sites which have been studied are given in Table 11.1.

The Committee has examined the considerable weight of evidence relating to the existence of childhood cancer clusters near nuclear sites, including evidence from aggregations of nuclear sites in the UK and Germany and has concluded that it is exposure to internal radiation from discharges from the sites which is the cause of the illness. The arguments against this position are well summarised in reports from the UK National Radiological Protection Board, the various reports of COMARE, and the three French government Nord-Cotentin missions. The response to the KiKK study has been more muted, nevertheless, despite this clear example of a finding which should force a re-appraisal of the risk model, nothing has been done.

For Sellafield (Seascale) these arguments were rehearsed in a court case in 1993 in which the Judge found, on the scientific evidence presented, that the leukemia cases could not have been caused by the radiation. However, the court in this instance was presented with the hypothesis that the cases were caused by the father's pre-conception irradiation, and little independent evidence to support this hypothesis was presented, owing to the unfortunate unexpected death of the chief witness, Prof Martin Gardner. No examination was made by the court of the alternative hypothesis, which was that the risk factor calculations presented were based on external acute irradiation and were therefore unsafe.

This is a general concern of the Committee. All the analyses of causality in the case of nuclear site clusters rely exclusively on the ICRP risk

model to show that the calculated doses to the children or their parents were insufficient to have been the cause of the disease since the linear ICRP model did not predict the leukaemias or cancers. The approximate discrepancy between the doses and the observed cases of leukemia in the various studies is given in Table 11.1.

Table 11.1 Studies establishing excess leukemia and cancer risk in children living near nuclear sites.

Nuclear Site	Year	Defined ICRP risk multiplier	Notes
[a]Sellafield/ Windscale, UK	1983	100-300	Well studied by COMARE: high level of discharge to atmosphere and sea
[a]Dounreay, UK	1986	100-1000	Well studied by COMARE: particle discharges to atmosphere and sea.
[a]La Hague, France	1993	100-1000	Particle discharges to atmosphere and sea: ecological and case control studies
[c]Aldermaston/ Burghfield, UK	1987	200-1000	Well studied by COMARE: particle discharges to atmosphere and rivers
[b]Hinkley Point, UK	1988	200-1000	Discharges to offshore mud bank
[d]Harwell	1997	200-1000	Discharges to atmosphere and river
[b]Kruemmel, Germany	1997	200-1000	Discharges to atmosphere and river
[d]Julich, Germany	1996	200-1000	Discharges to atmosphere and river
[b]Barsebaeck, Sweden	1998	200-1000	Discharges to atmosphere and sea
[b]Chepstow, UK	2001	200-1000	Discharges to offshore mud banks
Germany all; KiKK	2007	1000	Various types aggregated

[a]Reprocessing plants discharging to sea; [b]Nuclear power station discharging to sea or river; [c]Atomic weapon and nuclear material fabrication plants; [d] Atomic research with discharges to local rivers

The scientific basis for this approach has already been discussed in Chapter 3. The Committee concludes that these nuclear site cancer clusters together provide evidence for errors in the ICRP risk model resulting from the use of external irradiation studies to inform internal radiation risk. The explanation for the high level of risk associated with the discharges is that the exposures causing the leukemia and cancer are to novel radioisotopes like Strontium-90 and also to inhaled sub-micron diameter particles, including Uranium. These are translocated from the lung to the lymphatic system and thence in principle to any part of the body where they cause high doses to local tissue. The geophysical processes involved are well described and in the case of Plutonium and Sellafield, measurements have been made which show the presence of Plutonium and other radioactive particles in marine intertidal sediment, in the air near the coast, in sheep faeces, children's teeth and autopsy specimens taken from parts of the UK. The concentration of Plutonium with distance from the sea follows a trend with a sharp increase in levels within 1km of the sea falling rapidly and flattening out to a finite but reducing level up to 300km or more from the sea. The evidence is reviewed in the discussion of cancer near the Irish Sea below. However, the ICRP model used by COMARE and NRPB in their analyses of the Sellafield leukemia cluster averages the inhaled Plutonium doses over a very large mass of tissue and consequently the reports entirely fail to make the case that these exposures are a likely cause of the observed illness.

All of the other nuclear site clusters studied involve exposures either to the novel man-made isotopes which carry hazard weightings under the Committee's model or to airborne particle exposures. All the nuclear sites in Table 11.1 have in common that they contaminate local sea coasts or rivers which flood and are thus near areas where there are significant deposits of radioactive particles in intertidal, estuarial or river bank sediments. Pooled studies of leukemia and cancer near nuclear sites have shown that apart from some specific nuclear sites (those discussed), the existence of leukemia or cancer clusters is not a significant feature. These studies of aggregates of nuclear sites have various faults. The Committee believes that epidemiological studies of nuclear sites must establish which populations are most likely to be at risk on the basis of measurements of the dispersion of radioactive material in the environment near the source. Studies are usually made in which populations within a certain radius of the plant are compared with populations living at a greater radial distance, without consideration of the flow of radioactive material from the site, via rivers, sea-to-land transfer, land slopes and prevailing weather and wind directions. Good examples are afforded by recent studies of small area populations near two nuclear sites in the UK. In the vicinity of Bradwell in Essex, the UK Small Area Health Statistics Unit, SAHSU (one of the two Committees referred to in para. 1 of this chapter) drew radii of of 4, 10 and 17 km on the grounds that proximity to the plant was considered to be a proxy for radiation exposure. In a similar study of populations near the Nycomed Amersham plant in Cardiff SAHSU chose rings

of 2.5 and 7.5 km. Ward level studies by Green Audit established that the specific choice of radii enabled biased conclusions to be drawn (Busby 2006).

The nuclear sites listed in Table 11.1 have common factors; first, that they discharge novel radioactive materials in a way that results in their ingestion and inhalation, and second, that local cancer and leukemia clusters have been identified. This may be used to invite the application of the Bradford Hill canons for environmental causation. A Bayesian calculation to refine the assessment of the statistical probability of childhood cancer occurring near nuclear sites has never been applied to all the plants together although the NRPB and SAHSU epidemiologists in the UK have played down each cluster alone on the basis of the individual p-values.

In most of the cases in Table 11.1 the doses are not known but may be assumed to be small, on the basis of knowledge of the quantities released. However, for the most studied of these cases, Sellafield, the discrepancy between the modelled dose and the predicted number of leukemia cases based on the ICRP risk factors defines a discrepancy between the two of 300-fold and it is this value, and its similarity to the discrepancies found in other studies of internal radiation, that the Committee has used to develop the hazard adjusting coefficients employed in their model.

The confirmation of cancer and leukemia clusters in children living near nuclear sites has put considerable pressure on the scientific model of the ICRP. The confirmed observations of excess childhood cancer near the sites demonstrate a dissonance that cannot be accommodated within the scientific basis of ICRP paradigm. The only serious attempt to address this has been the work of Kinlen *et al.*, whose suggestion is based on studies of population mixing. Their idea is that the nuclear site leukemia clusters are caused by a rare response to a viral infection which is more likely in situations where there are new people mixing with rural groups whose immunity to the infection is poor. The Committee has carefully considered this theory and feels that it is unable to explain the Sellafield cluster, which has persisted long after any population mixing occurred, is more closely associated with the commencement of nuclear operations on the site than with its construction, and which involves a significant excess risk of cancer as well as leukemia. In addition, the magnitude of the effect found by Kinlen *et al.* for locations other than Sellafield is comparatively modest and could easily be explained by a number of less exotic mechanisms than the one they propose. In any case, there is no aetiological basis for it since no virus associated with childhood leukemia has ever been discovered; it is more likely that the modest increases in leukemia have a more prosaic explanation and that the effect of population mixing can be relegated to a second order phenomenon. Thus the Committee agrees that the existence of nuclear site leukemia and cancer clusters represents a response to exposure to the radioactive substances discharged and therefore is a ‘Popperian Falsification’ of the ICRP models.

11.2 Recent research on the Irish Sea and other contaminated coastal sites

The Committee has had access to unpublished results of a three-year study of cancer and radiation on the shores of the Irish Sea. Busby *et al.* examined cancer incidence from 1974-90 in Wales and 1994-96 in Ireland. They used small area data which were adjusted for socioeconomic disadvantagement, sex and age to look at the effect of living near the sea and made several discoveries.

For Wales they found:

- Risk of developing most cancers increases sharply near the coast.
- The increase is greatest in the 800 metre strip nearest to the sea.
- The increase is greatest near areas of low tidal energy where highest levels of radioactive material from Sellafield have been measured.
- The effect increased over the period and followed the peak releases from Sellafield in the mid 1970s by about five years.

By the end of the period, risks of childhood brain tumours or leukemia in some towns in north Wales near radioactive offshore mud banks were more than 5 times the national average.

For Ireland, using data only for all cancers, they found:

- The effect existed on the east coast but not on the south or west coast
- The effect existed in women but was weak or non-existent for men
- There was a strong cohort effect in both men and women born around the time of the Windscale reactor fire in 1957.

In addition, the group examined closely a part of Ireland, Carlingford, on the east coast. Using data from a local GP they were able to identify leukemia and brain tumour excesses in the period 1960-1986. They also conducted a questionnaire study in the area which revealed that the sea coast effect existed as close as 100 metres from the sea. People living within 100 metres of the sea had almost four times the probability of developing cancer than those living more than 1000 metres away.

The researchers believe the cause of the effect to be sea to land transfer of radioactive material trapped in intertidal sediment. This process was discovered in the mid 1980s and is well described. The trend of Plutonium with distance from the sea is similar to the trend in sodium chloride penetration, and shows a sharply rising concentration in air in the first kilometre. In the UK, Plutonium has been measured in sheep droppings across the whole country and the concentration in grassland, measured in the 1980s, shows a significant trend with distance from Sellafield. Plutonium has been measured also in children's teeth with the same trend, and has been found in autopsy specimens from all over the UK. Levels are highest in the tracheobronchial lymph nodes (TBN) which drain the lungs. Particles of about 1 micron diameter entering the lungs are transposed to the lymph nodes and lymphatic system where they can, in principle, reach any part of the body. Very recent work shows that in rare cases, particles of about 0.1 micron diameter can pass into the placenta and

possibly into the foetus. Such alpha emitting particles cause very high doses to local cells in the 40micron range of their disintegration tracks. In addition, cells will be hit again and again since the particle will continue to emit radiation. Thus the Second Event process considered in Chapter 8 is possible and represents a low probability/ high risk consideration. Beta emitting hot particles can irradiate the foetus from within the placenta. This is an area where there is insufficient evidence, and where more research is required.

Following the Irish Sea work, Busby *et al*. examined other nuclear sites which discharge to the sea, using cancer mortality data from 1995-1999. They discovered the same sea-coast effect on cancer near the Hinkley Point nuclear power station in Somerset and near the east coast nuclear power station at Bradwell in Essex which discharges to a muddy estuary. There was a high rate of cancer in those living near the sediment compared with those living inland. In the case of Bradwell, there was a good control town based on a similar estuary which had no nuclear power station, and which showed no increase in cancer above the national average.

The findings of these studies, which were supported by other recent work by the Radiation and Public Health Project in the US, may be seen as confirmation of the high degree of risk associated with internal exposure to micron sized radioactive particles.

The Committee is aware that these researches are based on ecological epidemiology and may suffer from all the problems of confounding associated with such studies but in view of the relevance of the results to human health are concerned to encourage further research in this area as a matter of urgency.

11.3 Nuclear accidents

The nuclear accidents which have contributed to significant releases to the global environment are listed in Table 11.2.

Table 11.2 Major nuclear accidents and their overall releases.

Accident	**Total (PBq)**	**Particles**	**Notes**
Kyshtym, USSR, 1957	74	High	High particulate yield from Ce-144: no adequate follow-up of health effects published
Windscale UK, 1957	0.83	Moderate	Attempts to cover up the direction of fallout;
3-Mile Island, US, 1979	566	No	Almost completely gaseous: no adequate follow-up
Chernobyl USSR, 1986	2088	High	Cover-up of early data. High and anomalous thyroid cancer admitted. Other effects disputed and an area of considerable argument (see text).

The Committee is concerned that the health consequences of the three nuclear accidents which occurred prior to the Chernobyl explosion in 1986 have not been studied by epidemiology. Evidence has been obtained that the Windscale accident may have caused increases in Down's Syndrome births in eastern Ireland, and there is recent evidence from the Irish Sea studies that there is a significant cancer cohort effect in those who were born around 1957. In addition the Isle of Man, a small island in the Irish Sea some 70km to the west of Windscale, has provided some evidence for a sharp increase in mortality from all causes beginning shortly after the accident. This is seen in data supplied by the government of the Isle of Man. The Committee has also seen evidence that the official meteorological wind direction records from this event have been tampered with, with the apparent motive of concealing the likely location of any effects.

The most recent nuclear accident, the Chernobyl explosion in 1986, was the largest accidental release of radioactive material to the environment and caused contamination in most countries in the northern hemisphere. A number of studies of health in the affected countries have been published or have been presented at conferences. The overall picture which emerged in the West by the time of the ECRR2003 report was one of confusing and mutually exclusive reports of increases in cancer, leukemia and genetic illnesses on the one hand, and of denial of any adverse health effect associated with the exposures on the other.

It now is clear that this was due to falsification or concealment of the basic data by the Soviet authorities. A number of official regulatory orders have been discovered and are reproduced in Yablokov *et al* 2009. For example, an order from the USSRs First Deputy Minister of Public Health, O.Shchepin on May 21st 1986 wrote:

. . . for specified persons hospitalized after exposure to ionizing radiation and having no signs or symptoms of acute radiation sickness at the time of release, the diagnosis shall be vegetovascular dystonia.

Another example is given in an explanatory note of the Central Military Medical Commission of the USSR Ministry of Defence:

(1) for remote consequences caused by ionizing radiation and a cause-and-effect-relationship, it is necessary to consider: leukemia or leucosis 5-10 years after radiation in doses exceeding 50 rad. . . . the presence of acute somatic illness and activation of chronic disease in [liquidators who] *do not have acute radiation sickness, the effect of ionizing radiation should not be included as a causal relationship.*

Assessment of the true health effects of the accident has been dominated by heavy handed cover-ups and suppression of data by UNSCEAR and IAEA. The WHO have been excluded from any real involvement in the issue, as the WHO President H Nakajima stated on camera at the conference in Kiev in 2001. At this conference, the UNSCEAR representative, Dr N Gentner wrote the conclusions of the conference himself, producing a statement which

denied the existence of any measurable effects of the radiation, and although the conference refused this version and voted strongly for an highly amended statement calling for research, the final statement was adjusted back to the original one (Busby 2006).

The Committee believes that a significant proportion of conclusions regarding increases in radiogenic illness are misguidedly based on a pre-supposed linear response between dose and effect. Such an assumption is invalid because of the confusion between external and internal doses and also because of the considerations relating to cell doses and cell sensitivities discussed Chapter 8. In addition, epidemiological studies have been influenced by or countered with the predictions of the ICRP risk models for populations exposed to the discharges. These predict very modest effects which would generally be difficult to establish against the large background cancer rates experienced by the study populations and therefore, when increases in cancer are seen in such populations, they are ignored or at least not ascribed to exposures from Chernobyl. The main reports examined by the Committee are listed in Table 11.3. With regard to cancer, the first evidence of late effects may be divided into evidence for increases in thyroid cancer, leukemia and solid tumours.

Since the 2003 report the situation with regard to Chernobyl health effects has markedly changed. This is largely because the Russian language peer reviewed reports of ill health and of animal and genetic studies which were ignored by UNSCEAR and ICRP have now been translated and reviewed (Yablokov and Busby 2006, Yablokov *et al.* 2009). Profs Yablokov and Burlakova attended the CERRIE conference in Oxford in 2003 and told the CERRIE secretariat that the evidence from Russian language publications was quite different from that being reported by IAEA and UNSCEAR. Many of these papers were then translated and were abstracted into English by Yablokov and Busby. However, the CERRIE report omitted them and any reference to them: they were included in the CERRIE Minority report (CERRIEa, CERRIEb). ECRR set up a sub-Committee on Chernobyl in 2003, chaired by Prof Yablokov: this resulted in the publication of *ECRR2006-Chernobyl 20 Years on* which can be downloaded free from the euradcom website. This monograph has contributions by several eminent scientists from the Russian Federation, Ukraine and Belarus and the reader is referred to the book for details. Briefly, it reported serious health effects of the accident, including genetic and genomic effects in humans and in animal and plant populations. Thus the total exclusion of Chernobyl epidemiology and Chernobyl effects from UNSCEAR 2000, UNSCEAR 2006 and ICRP 2007 is astonishing. In UNSCEAR 2006, a volume apparently devoted to radiation epidemiology, there is little mention in to Chernobyl and not one of the papers in any of the above are cited or discussed.

The evidence of the situation in these countries and in Europe is more recently reviewed by Yablokov *et al.* 2009, a publication of the prestigious *New York Academy of Sciences*.

Chernobyl effects were also a major topic of discussion and the subject of several papers given at the 2009 ECRR conference in Lesvos Greece; the proceedings of this conference are in preparation. The concluding statement from the conference calls for governments and researchers to see the Chernobyl accident as an opportunity to properly assess the health effects of radiation exposure. This statement, the Lesvos Declaration is in Appendix C.
ECRR is also currently assembling a report by Bandashevsky on his findings in the contaminated territories on Belarus.

In summary, the effects of the Chernobyl accident have been downplayed or dismissed by the ICRP, UNSCEAR and IAEA. It is quite clear that this was necessary in order to continue to support the ICRP model; the Chernobyl effects reported in the Russian language papers falsify that model. There are two cancer studies which have been published using data from western sources: the infant leukemia analyses and the analysis of cancer incidence in northern Sweden. Both falsify the ICRP model and the error in the risk model is approximately the same in both cases and the same as that predicted by ECRR on the basis of the earlier considerations. These will be discussed below.

Table 11.3 Chernobyl studies and reviews used as basis for the Committee's examination of the effects of the accident.

Reports/ assessments	**Note**
IAEA, 1994	Official Atomic Energy Agency Conference in Vienna characterised by reports showing either massive health deficit or little significant effect apart from thyroid cancer. Arguments from the floor. Proceedings still not published.
IPPNW, 1994	Independent conference held in Vienna at the time of the IAEA conference where scientists reported significant adverse health effects.
Savchenko 1995	UNESCO book by Belarus academician Savchenko reports thyroid cancer solid tumours, leukemia and congenital disease increases.
Burlakova, 1996	Edited by Russian academician Burlakova, reports various cancer, leukemia and ill health related biochemical and immune system indicator changes and also novel dose response to radiation.
Nesterenko, 1998	Book published by BELRAD organisation in Minsk reporting increases in thyroid cancer, leukemia and solid tumours; in children from Belarus.
UNSCEAR, 2000	Draws together selection of published studies with a commentary suggesting the only significant increase in ill health from radiation is due to thyroid cancer. Clumsy attempt to show results follow ICRP predictions even for thyroid cancer.
WHO, 2001	Conference in Kiev characterised by reports showing either massive health deficit or little significant effect apart from thyroid cancer. Conference resolution to ask for a re-assessment of risk models.
Kyoto, 1998	International collaborative work reports including accounts of dissonance between the 'official reports of radiation effects' and the real results in the affected territories.
Bandashevsky, 2000	Book showing increase in cardiac pathologies associated with measured internal contamination in children from Belarus.
Poland, Bulgaria, various	Various reports from Poland and Bulgaria show sharp increases in cancer and ill health effects in infants and anomalous birth outcomes immediately following Chernobyl.

Busby, 2001	Report to Belarus Embassy with review of data and predictions of new risk model from cancer yield in Belarus
Infant leukaemias	Infant leukemia reported in six countries in cohort exposed *in utero* defines error in ICRP risk factor of 100-fold or more (see text).
Minisatellite mutations	Various papers report increase in minisatellite mutation rate in children from high exposure region and in offspring of liquidators: implied error of up to 2000-fold in ICRP model.
IARC, various	'Official' examination of leukemia increases in Europe using pooled database suggests no increase that is ascribable to Chernobyl: flawed approach.
Belarus and Ukraine reports in Russian	Many reports from Belarus, Ukraine and Russian Federation contain evidence of increases in leukemia, solid tumours, thyroid cancers, congenital malformations and general massive health deficit following and ascribable to the exposure. Reports not translated or included in official reviews.
CERRIE 2004b	Contains section with abstracts of 40 major peer reviewed papers in Russian n the health effects of the Chernobyl accident
Okeanov *et al* 2004	Report in Geneva of levels of cancer increase registered by the Belarus Cancer Registries (see text Chapter 14).
Tondel *et al* 2004	Study of cancer in northern Sweden associated with Chernobyl fallout (see text Chapter 14).
ECRR2006 ECRR2009	*Chernobyl 20 Years On* Eds. AV Yablokov, CC Busby. Compilation and review of peer reviewed studies on the health effects of the Chernobyl accident published in Russian; 2nd edition 2009
Bandashevsky 2008	Update of Yuri Bandashevsky's work published in Lithuania.
ECRR 2009	3rd International Conference of the ECRR Lesvos Greece May 5/7th 2009
Yablokov *et al* 2009	*Chernobyl. Consequences of the Catastrophe for People and the Environment*

11.4 Dispersion of Chernobyl fallout and doses

Owing to prevailing weather conditions, the dispersion of the material from the Chernobyl explosion and subsequent fire was very variable, but was well characterized due to measurements which were made in many countries of the world. It was global, with detection of radionuclides as far away as the USA, south-east Asia and Japan. It is therefore predicted by the ECRR risk model that health effects will be detectable in many countries, but very little seems to have been done to look for such evidence, possibly because the doses were very low and at such low doses the ICRP model predicts no measurable effects. But when researchers did look, they found results. The increase in infant leukemia in Europe in the *in utero* cohort has been mentioned; this increase was also reported for the USA by Mangano (Mangano 1997). Cancer increases in Sweden were reported by Tondel *et al.* 2004. There was a sharp increase in cancer incidence in the UK registries for Wales and Scotland (Busby 2006).The average first year committed effective (ICRP) doses reported by Savchenko 1995 by country for these various contaminations ranged from about 2mSv in Belarus through 1mSv in Ukraine, between 0.5 and 0.7mSv in Bulgaria, Austria, Greece, Romania, Finland and the Russian Federation, to 0.08mSv in the UK and China. Of course, the ECRR weightings would increase these doses significantly but without an analytical breakdown of the isotopes involved only an approximation can be made. The radionuclides involved in the dispersion were very high levels of initial Tellurium-132/Iodine 132, a gaseous Second Event pair, Caesium-137, Plutonium 239, Strontium-90 and Uranium fuel particles containing various fission-product beta-emitters. The amount of uranium in the fallout was not measured, although these particles contaminated the air and crops (Hohenemser *et al*, 1986). The ECRR weightings for such exposures are very high, some 3 orders of magnitude. On the basis of the increases in cancer in the intervening period, the doses calculated by application of the ECRR weightings to the mainly Cs-137 gamma doses which represent the basis of the Savchenko estimates seem to predict the increases fairly well.

11.5 Reported effects of the exposures

On the basis of the ECRR effective doses which are certainly greater than 600mSv in Belarus it would be expected that there would be clear health effects in that country and indeed such are reported in the Belarussian literature: cancer, birth defects, a sick population, loss of lifespan (Yablokov *et al* 2009, Okeanov 2004, Bandashevsky 2000, 2000a -2000c, Bandashevskaya 2003, Busby and Yablokov 2006, Busby and Yablokov 2009, Yablokov *et al* 2009). What is most apparent in the data is the enormous range of health effects and conditions which correlate with exposure to radionuclides from the contamination. This wide range makes it difficult to assess health outcomes in

terms of a simple indicator of cancer rates. As pointed out above, increased death rates in children and young adults from any disease or condition will lower the incidence rates from cancer since this is a disease of old age. In this publication, the Committee has no space to review the full spectrum of disease which has followed the Chernobyl fallout exposures and refers those who wish to follow up this issue to its own publication Busby and Yablokov 2006 (2nd Edition 2009) and to Yablokov *et al* 2009. Effects of the Chernobyl accident exposures also appeared in many other countries in Europe, where researchers were prepared to look, for example Trisomy 21 Downs births in Germany, Sweden and the UK, changes in minisatellite DNA and thyroid diseases.

11.6 Thyroid cancer after Chernobyl

The remarkable and aggressive increases in thyroid cancer in territories most affected by the disaster were initially denied by the radiation risk establishment but later, owing to the fact that the disease is normally very rare, were conceded. Although no formal calculation was published, the increases appeared to show that there were two significant errors in the risk models of the ICRP, apart from the fact that the effect was orders of magnitude larger than that predicted by the ICRP risk factors. The first error concerned the belief that internal irradiation of the thyroid by radio-Iodine was less effective than external irradiation in causing cancer. The second was in the belief that there would be a time lag of more than ten years in the onset of the clinical symptoms. In the event, thyroid cancer increases began a few years after the doses were delivered.

The risk agency community, having had to concede the facts of the increase, promptly responded by adjusting the doses to as high a level as possible to try and fit the data to the model. The idea was to assume that the children who were affected had been Iodine-deficient and therefore their thyroid glands would take up more Iodine. This was unsuccessful since doses large enough to fit the cancer data would be so high that the children would have died of radiation sickness. Early data was presented in ECRR2003 but Table 11.4 shows the situation in Belarus up to 2004 (Malko 2009). In the most contaminated region, Gomel, the difference between the expected numbers of cases and those observed is 126. Other data from 1986 to 2007 presented by Malko 2009 shows that the effect peaked in Belarus in 1995 and by 2001 had fallen back to just around the baseline pre 1986 levels. It should be noted that the thyroid gland is very radiation sensitive due to its iodine content since iodine has a high atomic number Z = 53 and therefore living in an areas of high gamma radiation or being internally exposed to gamma emitters apart from I-131 will significantly add to the exposures through the secondary photoelectron effect. In parts of northern Finland where inhabitants were also exposed to internal uranium (though local geology) the Iodine exposure from Chernobyl had a much greater thyroid cancer producing effect than in areas where there

was no uranium (Slama 2009). The ICRP and UNSCEAR have been unable to respond credibly to the increase in thyroid cancer and its dissonance with their radiation models.

Table 11.4 Thyroid cancer incidence and Relative Risk (based on ICRP model) in children in Belarussian regions from 1986 to 2004 (Malko 2009)

Regions	**Observed**	**Expected**	**O-E**	**RR**
Brest	165	3	162	55
Vitebsk	11	2	9	5.5
Gomel	378	3	375	126
Grodno	43	2	41	21.5
City Minsk	62	3	59	20.7
Region Minsk	42	3	39	14
Mogilev	43	2	41	21.5
All	744	18	726	41.3

The error in both the absolute magnitude of the effect and also the rapid onset may be a consequence of the fact that the extremely active Tellurium-132/Iodine-132 Second Event couple was a major exposure hazard in the early days. In addition, the basis of the radio-Iodine risk model is a series of studies by Holm on hospital thyroid patients in which any cancers which developed within the first five years of exposure were discarded from the study as being due to pre-existing lesions on the basis that the Hiroshima LSS had shown a significant time lag for thyroid cancer. Lars Erik Holm has been mentioned earlier in connection with the ICRP.

11.5 Leukemia after Chernobyl

Since Hiroshima, observations of high leukemia yields following the A-bomb, leukemia and especially childhood leukemia has become the first symptom to be investigated in any irradiated population. For this reason, leukemia incidence is likely to be the first data that would be addressed by establishments wishing to control the perception of harm following a nuclear accident. Recalling that the accident occurred during a period when state control of data by the former USSR was significant, the Committee interprets the confusion over increases in leukemia rates in Chernobyl affected territories as due partly to this factor. The problems in interpreting leukemia data and studies of leukemia following Chernobyl are listed in Table 11.5.

Table 11.5 Problems interpreting data on leukemia after Chernobyl

Problems in interpreting data on leukemia after Chernobyl
1. Soviet cover-up at the diagnosis stage, so no leukemia appears on medical forms.
2. Soviet cover-up at the registry/report stage so figures are adjusted to fit controls.
3. Later researchers use database containing incorrect totals.
4. Assumption of linear response means that controls may have higher rate than exposed.
5. Regression method assume linear response: coefficients will include Type II error.
6. Small numbers make result critically dependent on removal or exclusion of few cases.
7. Pooled data will give confused results owing to dose response variation.

There have been reports of leukemia increases in the main Chernobyl-affected territories of the ex-Soviet Union (listed in Table 11.3); reviews assert that no increases are predicted and that any increases found are due to better ascertainment or cannot be caused by radiation owing to lack of a positive dose response coefficient (also listed). The Committee takes the view that leukemia data from the Chernobyl affected territories are difficult to analyse in such a way as to develop useful models owing to lack of accurate data for internal and external doses, the insecurity of the databases and other problems listed in Table 11.5

There have been two main sets of studies which inform on leukemia risk in Europe, the series of studies undertaken by IARC in Lyon and the infant leukemia reports. In the IARC series, childhood leukemia incidence data were pooled from most of the cancer registries in Europe and the ex-Soviet territories, and analysed as a time series and using regression methods to examine the hypothesis that the exposure period was followed by a significant step in childhood leukemia. Although an increase was observed, it did not show as a step change and in addition, the highest doses did not correlate with the highest incidence. This resulted in the authors concluding that the accident had no significant effect. The Committee views this study as essentially flawed owing to variation in dose and in genetic susceptibility across the pooled dataset and considers that examination of individual time series from each country might reveal an effect, as it did in data from Scotland and Wales.

The second set of studies involved examining the increase in infant leukemia 0-1 in the cohort who were *in utero* over the period of maximum exposure to internal irradiation from Caesium-137 or other isotopes. Examination of this phenomenon, which was reported from six separate countries, forms part of an analysis which the Committee accepts as unequivocal evidence for a significant error of 100-fold or greater in the ICRP risk factors for internal irradiation. This will be considered separately.

11.6 Cancer in Northern Sweden after Chernobyl

Weapons fallout studies of Wales and England followed differential exposures of populations in two countries. Tondel *et al* 2004 published a sophisticated epidemiological analysis of differential Chernobyl fallout effects in small areas of one country, northern Sweden. They correlated Cs-137 precipitation in small areas (communities) with cancer incidence from 1984 to 1996 in order to examine the effects of exposure. Cs-137 data were obtained from the Swedish Radiological Protection agency SSI and cancer incidence from the Swedish Cancer Registry. The study found a statistically significant 11% increased risk per 100kBq/m^2. Using external exposure data only this translates to a 650-fold error in the ICRP model, but if internal exposure is added in, one the basis of Cs-137 alone, this number will probably reduce to a 400-fold error. It should be noted that the real dose is unknown; the radiation covariate was Cs-137 area contamination. The Chernobyl fallout was not the same as the weapons fallout in terms of the spectrum of radionuclides. There will have been other isotopes and the material will have included Uranium fuel particles. However, in terms of ICRP analyses, it is the external Cs-137 dose that would be the quantity employed for radiation protection. Nevertheless we see good agreement with the earlier study of Wales and England which gave a 300-fold error.

11.7 Nuclear workers and their children

Nuclear workers and their children are an obvious category for the analysis of radiation induced disease and the Committee has examined the main studies which have looked at cancer and leukemia rates in this group. Most studies have shown that the group (with some exceptions) has a lower rate of incidence of these diseases than controls from the general population. This is conceded by the authors of these studies as due to the fact that nuclear workers have in general better health than the general population owing to their higher socioeconomic status, the ‘healthy worker effect’. The magnitude of this effect has been hard to assess from published data. However, a very large recent study gave information which enabled the Committee to reanalyse the data and to show a trend in cancer risk with length of employment in the nuclear industry. The results are given in Table 11.6

Table 11.6 making allowance for the healthy worker effect in data from the 'Second Analysis of the National Registry of (UK) Radiation Workers'

Years in industry	All deaths	SMR all causes	All cancers	SMR all cancers	SMR all cancer corrected a
0-1	281	64	67	64	112
2-4	623	72	159	73	128
5-9	1466	79	443	89	156
10-14	1863	81	508	80	140
15-19	2162	87	589	85	149
20-25	4194	85	1186	82	143
30+	2176	83	646	80	140

[a] *Based on extrapolation of trend in cancer SMR to zero time to give SMR = 57*

The method used to obtain a value for the 'healthy worker effect' is based on the extrapolation of the trend in standardised mortality ratio to the moment the worker enters the nuclear industry. Using the resultant zero dose, zero time SMR as a control, it is clear that although nuclear workers may have lower age specific mortality than the general population, they die at a greater rate than they would if they worked not in the nuclear industry but in some other employment which conferred the same economic and social benefits. The results in Table 11.6 show that this effect occurs within the first five years of employment and by 5-9 years working in the industry their risk of death from cancer is more than 50% higher than it would be if they had not been employed in this way.

A problem with the nuclear industry workers' studies is that the doses are measured by film badges and therefore are external. No real data exist for internal doses although there is considerable indicative evidence that it is the low internal doses that are responsible for the slightly increased rates of cancer and leukemia that are found among nuclear workers and their children. These increases are usually discounted on the basis that the dose-response relationship is not linear, and that the groups with highest cancer risk are not the high dose group, but are usually the intermediate dose group. This effect, the Burlakova type response, was found in recent studies of UK workers as Table 11.7 shows.

Table 11.7 Trends with increasing external doses for mortality risks from all cancers and from leukemia derived from the Second Analysis of Nuclear Industry Workers (UK) and adjusted for healthy worker effect.

Film badge dose (mSv)	**SMR All cancers**	**Corrected SMR all cancers[a]**	**SMR leukemia**	**Corrected SMR leukemia[a]**
0 (zero time)	0.57	1.00	0.57	1.00
<10	0.97	1.7	1.06	1.86
10-	1.01	1.8	0.7	1.22
20-	0.97	1.7	0.77	1.4
50-	1.10	1.9	1.24	2.2
>100[b]	1.01	1.8	1.19	2.1

[a] *Corrected on the basis of a healthy worker cancer mortality risk of 0.57 relative to the general public*

[b] *Averaged over the dose groups 100-200, 200-300 and 300+ due to small numbers in these groups*

No real attempt has been made by the authors of the various studies of nuclear workers and their families to establish the magnitude of the healthy worker effect and the Committee feels that this is an important issue which must be addressed. The use of internal comparisons using groups within different external radiation dose ranges is not helpful since the dose–response linearity assumptions are built in to the interpretation of the results. In addition, it is not clear with pooled studies that such a stratification is epidemiologically homogeneous, and may compare individuals from different sites or with different internal doses from internal isotopes. The main nuclear industry studies considered by the Committee are given in Table 11.8.

Table 11.8 Main studies of nuclear workers considered by the Committee

Study	Notes
1. Hanford, USA	External: 10-fold error in the external risk factor found; doubling dose for all cancers 340mSv; leukemia excess not dose related.
2. UKAEA	External: increased mortality from various kinds of cancer. Clear excess from prostate cancer.
3. UKAEA Prostate	Case control study: prostate cancer associated with monitoring for internal exposure with relative risk up to 20-fold. Defined error in ICRP model from internal isotope exposure risk at about 1000-times.
4. Sellafield, UK	External: excess cancer risk found with wide confidence intervals. Central estimate about 0.1 per Sv in 10mSv region.
5. AWE, UK	Average external dose 8mSv. Evidence of increased risk with period of employment.
6. All workers UK	Analysis of pooled data; Burlakova type response; excess risk from all cancers based on healthy worker effect (see text).
7. Oak Ridge US	Increased risk in older workers reported.
8. Nuclear Industry Family Study UK	Leukemia in <25-year-old offspring of nuclear workers in UK found significant excess risk of leukemia with relative incidence risk in offspring of fathers with >100mSv of 5.8. Biphasic dose response; doubling risk from internal monitoring.
9. Offspring Record Linkage Study UK	After exclusion of Sellafield fathers, there was a significant excess risk of leukemia or non Hodgkin lymphoma in the offspring of radiation workers (fathers RR=1.77, mothers RR = 5) with evidence for Burlakova response and highest risk if monitored for internal isotopes (RR = 2.91 vs. 1.61 not monitored). Authors use non-linear response as evidence that radiation was not the cause.

11.8 Unequivocal evidence

All the evidence which associates low level internal exposure with cancer and leukemia suffers from the problem that other causes for the effects may be advanced, however implausible they may be. Kinlen *et al.*'s population mixing (discussed above) is a good example of this. There is also the problem that with low level radiation, cause and effect are separated by the lag period between initial genetic damage and final clinical expression of a cancer which can be confirmed by histopathology, and during such a period other possible causes

may be found. However, in the last few years, advances in technology and the existence of well defined populations who were exposed after the Chernobyl accident, together with a slight easing of the situation with regard to access to small cancer incidence and mortality data have made possible two study situations where there is now unequivocal evidence of error in the ICRP model as it relates to internal exposure. The two sets of such studies providing unequivocal evidence of risk factor error are listed in Table 11.9

Table 11.9 Recent studies which the Committee takes to show unequivocal evidence of error in the ICRP models.

Study	**Shows**
1. Minisatellite DNA mutation after Chernobyl	Objective scientific indicator in children born after Chernobyl accident shows 7-fold increase in mutation relative to siblings born before. Error in ICRP 700- to 2000- fold for this end-point.
2. Infant leukemia in five countries	Increases in infant leukemia in children who were *in utero* over the exposure period for internal radiation define error in ICRP risk factor from 100- to 2000-fold for this end-point.

11.9 Studies representing unequivocal evidence of errors in ICRP model

11.9.1 Minisatellite DNA

The ICRP model of genetic mutation after irradiation is based, like ICRP's cancer risk model, on the Hiroshima LSS yield of gross genetic effects and studies of radiation effects in mice.

Although subtle genetic effects on sex ratio were apparent in the LSS offspring, the RERF researchers excluded them from the study because they did not accord with their notions of the expected direction of such an effect [Padmanabhan, 1997, Busby 2006]. Neels's exclusion of the sex ratio effects resulted in the belief that the genetic effects of 10mSv in the first generation would be unmeasurable. Thus BEIR V gives the incidence of total genetic effects including chromosomal effects (unbalanced translocations and trisomies) at 6 per million offspring compared with the natural rate of 4,200. It predicts a 10mSv excess risk of 10 cases of congenital malformation in a natural rate of 25,000 per million offspring and similar vanishingly small increases are given for autosomal dominant, X-linked and recessive disorders. Using a combination of mouse studies and the epidemiology of the LSS, the doubling dose for spontaneous genetic burden has been estimated to be 1 Sievert. [e.g.BEIR V, 1990 p 70]

However, the development of molecular techniques has enabled objective measurements of the consequences of irradiation to be investigated in

human populations. There have been several studies of minisatellite DNA mutation in children living in parts of the ex-Soviet Union and exposed to radiation from Chernobyl. Using the technological development of 'DNA testing' in which minisatellite DNA is separated into bands which are characteristic of its genetic identity, it has been possible to show that children living in Belarus and exposed to radiation from fission-product isotopes which contaminated their environment suffered a doubling in genetic mutation. [Dubrova, 1996, 1997]. Similar work with barn swallows exposed in Belarus showed that these genetic changes were also present and were associated with phenotypic changes in plumage patterns as well as reduced survival, therefore underlining the potential importance of such mutations. [Ellegren *et al.* 1997].

Most recently, the minisatellite DNA tests have been applied to the children of Chernobyl liquidators who were born after the accident compared with siblings born before the accident. [Weinberg *et al.* 2001] There was a seven-fold increase in genetic damage found in the post-exposure children. By comparison with mutation rates for the loci measured, this finding defined an error of between 700-fold and 2000-fold in the ICRP model for heritable genetic damage. In addition, the research results could be stratified by dose range and this resulted in a biphasic or Burlakova type response. It is remarkable that studies of the children of those exposed to external radiation at Hiroshima show little or no such effect, suggesting a fundamental difference in mechanism between the exposures. [Satoh and Kodaira, 1996]. The most likely difference is that it was the internal exposure to the Chernobyl liquidators that caused the effects.

This evidence of a substantial error in the ICRP model may have been accepted by the Chairman of the UK Committee on Medical Aspects of Radiation in the Environment, Professor B. A. Bridges, who conceded that the time may have come for a paradigm shift. In his outline of concerns, Bridges focused on the bystander effect whereby intercellular communication between cells traversed by an ionising track causes nearby cells to exhibit genomic instability resulting in genetic mutation in a large number of cells which were not subject to the initial ionisation injury [Azzam *et al.* 1998, Hei 2001]. It remains to determine a model in which external and internal irradiation may result in significant differences in such an end point since, in principle, genomic instability and bystander effects are applicable to internal and external irradiation and to natural and novel sources equally.

11.9.2 Chernobyl infants

Following the Chernobyl accident in 1986, the cohort of children who were exposed in their mother's womb to radioisotopes from the releases suffered an excess risk of developing leukemia in their first year of life. This 'infant leukemia' cohort effect was observed in six different countries. It was first reported in Scotland [Gibson *et al.*, 1988], and then in Greece [Petridou *et al.*,

1996], in the United States [Mangano, 1997] and in Germany [Michaelis, *et al.* 1997].

Busby and Scott Cato examined the relationship between the observed numbers of cases and those predicted by the ICRP model. For the first time, the specificity of the cohort enabled them to argue that the effect could only be a consequence of exposure to the Chernobyl fallout. There could be no alternative explanation.

Because the National Radiological Protection Board had measured and assessed the doses to the populations of Wales and Scotland and because they themselves had also published risk factors for radiogenic leukemia based on ICRP models it was a simple matter to compare their predictions with the observations and test the contemporary risk model. The method simply assumed that infants born in the periods 1980-85 and 1990-92 were unexposed and defined the Poisson expectation of numbers of infant leukemia cases in the children who were *in utero* over the 18 month period following the Chernobyl fallout. This 18 month period was chosen because it was shown that the *in utero* dose was due to radioactive isotopes which were ingested or inhaled by the mothers. Whole-body monitoring had shown that this material remained in the bodies of the mothers until Spring 1987 because silage cut in the Summer of 1986 had been fed to cattle in the following winter. The result showed a statistically significant 3.8-fold excess of infant leukemia in the combined Wales and Scotland cohort ($p = 0.0002$). The leukemia yield in the exposed *in utero* cohort was about 100 times the yield predicted by the ICRP model. Table 11.10 compares the effect in the three main studies. In this table, the B cohort were those children exposed to the internal exposure from Chernobyl *in utero* in the 18 month period following the event and born between June 1987 and January 1988. These exposure periods were defined by the whole body monitoring results. The control periods A and C were the ten years before (1975-85) and the four years after 1988 for which data was available.

Since the World Health Organisation has given approximate exposure levels in Greece, Germany and the United States, it was also possible to examine the leukemia yield in the infant ‘exposed cohort’ reported by the several other studies and establish a dose response relationship. It was found that a biphasic or Burlakova type relationship existed for the data from the separate countries.

The importance of this study as evidence of falsification of ICRP caused it to be included in the CERRIE Committee agenda. Members of the Committee from both sides of the argument analysed a new dataset which was provided by the Oxford based Childhood Cancer Research Group. This gave infant leukemia numbers in the same exposure cohorts employed by the study carried out in Greece and that in Germany. This was a cohort of those who were *in utero* over the period of the fallout as defined by Petridou *et al*. 1996. Analysis by Muirhead of NRPB and by Wakeford of British Nuclear Fuels showed an excess of infant leukemia in the various countries with errors

ranging from about 100 to about 1000. Nevertheless, the final CERRIE report marginalised these results and ignored their significance, despite displaying the actual numbers and confidence intervals in a table in an Appendix. Later the UK data was aggregated with that from Germany and Greece to show that there was a highly statistically significant 43% excess infant leukemia risk in the combined cohort in the UK, Germany and Greece (Busby 2009).

The Committee accepts that the infant leukemia results represent unequivocal evidence that the ICRP risk model is in error by a factor of between 100-fold and 1000-fold for the type of exposure and dose, the latter figure allowing for a continued excess risk in the cohort being studied. The Committee notes that it will be necessary to follow the cohort as it ages. The Scottish Cancer registry has refused to release data to allow this to be done.

Table 11.10 Unequivocal evidence of ICRP risk factor errors: comparison between infant leukemia rates after Chernobyl in Wales and Scotland and similar data from Greece and from the former Federal Republic of Germany

Group	**[a]Wales and Scotland**	**[b]Greece**	**[c]Germany**
Exposed cohort B			
Cohort size	156,600	163,337	928,649
Number of cases	12	12	35
Rate	7.67	7.34	3.77
Unexposed cohort A + C			
Cohort size	835,200	1,112,566	5,630,789
Number of cases	18	31	143
Rate	2.15	2.79	2.54
Risk Ratio	3.6	2.6	1.5
Cumulative Poisson Probability	0.0002	0.0025	0.02

[a] See text for A B and C periods [b] Petridou et al.(1996) [c] Michaelis et al.(1997)

12
Uranium
Depleted Uranium Weapons

For there is nothing hid that shall not be manifested;
neither was anything kept secret, but that it should come abroad

Mark 4,22

12.1 Introduction

The element Uranium is the basis of and parent of almost all releases of radioactivity to the environment, yet curiously, until it began to be employed as a weapon, it had been quite neglected as a hazardous component. It is not measured routinely near nuclear power stations or reprocessing sites. It is treated as if it were natural which of course it is, but its concentration in these places, and the form it is released in is not.

The intense and increasing interest in the health of the troops who participated in the first Persian Gulf War in Iraq, and later those who served in the Balkans, where Uranium weapons were also used, and of course the civilian populations of those areas have resulted in evidence that the genotoxicity of Uranium is far greater than the military who used it, and the states which sanctioned this, believed. Despite the increasing evidence of its anomalous propensity for harm, from epidemiology and from laboratory and theory, the ICRP risk model, here as in everywhere else in radiation protection, is used to deny the evidence and to sanction its continued use as a weapon of war. As with the fallout from bomb tests, Chernobyl and the child leukemias near power stations, clear evidence of harm from exposure to Uranium is denied on the basis of deductive logic, that the absorbed doses are too low to cause any measurable effect. By 2006, when massive population-based evidence that the exposures to so-called Depleted Uranium, DU were causing harm, and evidence from laboratory studies and theoretical research had also emerged, UNSCEAR, in their 2006 report allowed 11 lines on one page in their 400 page report to the consideration of DU effects. UNSCEAR based its dismissal of any problem with Uranium exposures on three citations, desktop reviews, the RAND corporation 1999 report (Harley *et al* 1999), the US Institute of Medicine 2001 report and that of the Royal Society in 2001. None of these reports were peer-reviewed, and the RAND corporation is believed to be closely associated with the US Pentagon. All were selective in their references. And all were out of date. None of these could deal with the particulate nanoparticle inhaled Uranium from weapons fallout, since no-one had studied it. Y*et al*l three (and also countless reports from agencies like WHO) employed the ICRP model to show that the doses were too low.

Despite the many studies which will be reviewed below and which were accessible to UNSCEAR, its 2006 report (which appeared in 2008) states (p53):

There appear to be several possible reasons why Uranium is not. . . considered a human carcinogen (by the Institute of Health): Uranium is not very radioactive (having such a long half life of billions of years, 238U decays very slowly) and its chemical properties are often such that any inhaled or ingested Uranium is excreted rather quickly from the body.

By 2004, the way that official agency reports ignored the increasing peer reviewed evidence that Uranium was much more genotoxic than its radioactivity suggested became so embarrassing that the senior radiation health advisor to the WHO, Keith Baverstock wrote a paper with Carmel Mothershill on the issue to the Director General. He was sacked but the paper was later published (Baverstock 2005).

The scientific investigation of DU gives a curious condensed echo of the earlier investigations into the nuclear site child leukemias. This is not surprising given the political consequences of having to concede that the low doses of DU, conventionally assessed, were capable of causing such graphic and appalling genetic effects on populations exposed to the dust. For if this could happen with Uranium, it means that all of the basic equations and assumptions of the risk model are wrong. The matter has been painstakingly researched and reviewed recently by an American academic, Paul Zimmerman whose conclusions, independently gained by an academic, closely agree with the ECRR thesis developed in 2003 and in the present 2010 report (Zimmerman 2008).

It is an interesting fact that the military and the nuclear industry internally take Uranium exposure very seriously as far as handling the material is concerned. Spills, even small ones have to be dealt with, with all the rigours associated with contamination by radioactive material. The same is true for the military, who publish internal documents warning of the health effects. However, as soon as the Uranium is shot from the gun and has contaminated the theatre of war, it suddenly becomes benign, in all the reports of the issue, and in the denials of the military and its risk agencies and those of the governments involved.

The effects of exposure to Uranium are not, of course, restricted to DU and passive weapons fallout. Uranium is increasingly contaminating the environment, near nuclear sites, near isotope separation plants, near fuel manufactories, near Uranium mines and in atomic and thermonuclear weapons fission fallout, near and remote from the test sites. Uranium is increasingly found in food and drinking water as it is a significant component of agricultural fertilizer. It is therefore also found near fertilizer factories and phosphate mines and in the transportation of phosphate ore and its agricultural products

(Eisenbud and Gesell 2000, Busby and Schnug 2008). The mining of Uranium began at the beginning of the last century. Also beginning at the same time was a new disease: childhood leukemia, which is believed to result from a mutation *in utero*. The temporal correlation between the incidence of this disease and the production of Uranium (modeled as Radium) is startling, and is shown in Fig 12.1 below. Despite this, Uranium seems to have been forgotten in investigations into contamination near nuclear sites, diseases associated with weapons fallout, Chernobyl effects. It is the invisible substance. Measurements made new nuclear sites will show concentrations of exotic isotopes, vanishing concentrations of Plutonium in fish, but few measurements are made of the Uranium emerging from the nuclear sites. In the COMARE analysis into the Sellafield child leukemias, it was concluded that although the doses from Plutonium to the tracheobronchial lymph nodes of the children were high, the doses from natural radionuclides were higher, and so the nuclear site could not be responsible, even if these were the source of the disease. After Chernobyl, large amounts of Uranium were released as fuel particles, but no measurement of Uranium is to be found in any of the reports on Chernobyl fallout.

ECRR set up a sub-Committee in 2001 to examine the issue of Uranium weapons. This chapter will present a brief account of the findings, will review the evidence for DU and Uranium effects and will make recommendations.
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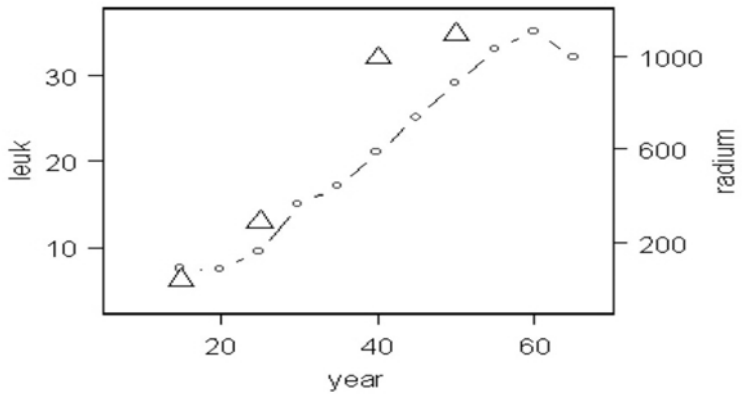
Fig 12.1 Trend in child leukemia mortality (line) and world Radium production (g) (*Source: Busby 2002 acknowledging Bramhall R)*



12.2 Depleted Uranium: Uranium weapons.

Depleted Uranium is a by product of the nuclear industry where the fissile isotope U-235 in natural Uranium ore is concentrated to produce reactor fuel consisting of 'enriched Uranium'. The isotope discarded by this process is Uranium 238 which is generally classed by the risk agencies as a low radiation hazard material owing to its long half life (4.5×10^9 y) and its weak gamma emission of 48keV. However, it is an alpha emitter and thus poses an ingestion risk owing to the high ionization density of alpha tracks and their high biological effectiveness in inducing mutation. In addition, there is a risk from the beta-emitting daughter isotopes Thorium 234 (β 0.26MeV, half life 24 days) and Protoactinium-234m (β; 0.23MeV, half life 6.75 hours) which decay through one another to Uranium-234, also an alpha emitter with a half life of 2.47×10^5 years. The overall activity of Uranium 238 therefore increases as soon as it is produced due to ingrowth of the beta daughters and by 30 weeks these are in total secular equilibrium. The activities per kilogram are given in Table 12.1 below. Uranium-238, because of its long half life, has a low specific activity, $12MBqkg^{-1}$ which means that, unlike most radionuclides which are considered in risk analyses, at environmental concentrations which represent a radiological exposure, the chemical concentration is significant. 1Bq is 83μg and $1Bqg^{-1}$ in tissue represents a concentration of 3.5×10^{-4} M which is a significant physiological concentration.

Over centuries, the specific activity of U-234 should be the same as the parent U-238, and thus the environmental concentrations of these isotopes are generally the same if the source is natural. The specific total activity is thus about 37MBq/Kg. It should be pointed out that DU material recently found in battlefields in Europe contains small quantities of isotopes of Plutonium, Neptunium and other fission products: thus the source of this DU is refinement of nuclear reactor waste. However, the quantities are very small and are not considered by the Committee to be of serious radiological significance. More curious are reports of weapons which have isotopic signatures showing enriched Uranium, first reported in Lebanon, then Gaza, and most recently in analysis of biological materials from a veteran of the Bosnia theatre in 1996 (Busby and Williams 2006, 2008, Ballardie *et al* 2008). Indeed, tables of isotope ratios in environmental post conflict samples published by the United Nations Environment program UNEP show clear evidence of enriched Uranium usage in Bosnia (UNEP Bosnia report 2002). (UNEP have consistently denied finding enriched Uranium, and this mistake was quickly covered up when pointed out: the table has been taken off the UNEP website). For this reason, the ECRR prefers the term Weapons Derived Uranium (WDU) to describe the issue.

Table 12.1 Specific Activity (MBq/kg) in decay of U-238 in Depleted Uranium to U-234 and ingrowth of daughters

Weeks	**U-238 (α,γ)**	**Th-234 (β)**	**Pa-234 (β)**	**U-234 (α,γ)**
0	12.43	0	0	0
5	12.43	7.89	7.84	0.001
10	12.43	10.77	10.75	0.004
20	12.43	12.21	12.21	0.01
30	12.43	12.4	12.4	0.017

Owing to the high density of Uranium, (19 $g.cm^{-3}$ metal and 10.96 $g. cm^{-3}$ for the dioxide) and the fact that the metal is pyrophoric (burns in air) the substance is used in the manufacture of armour piercing shells, missile nose cones and penetrators. It is also employed in certain ballast materials in some aircraft (e.g. helicopter rotors, commercial aircraft counterweights). As a weapon, on impact, the DU burns to a fine aerosol of ceramic Uranium oxide particles of mean diameters from about 1000nm (1μ) down to below 100nm depending on different study results and distances from targets. These particles are long lived in the environment (and in tissue), and can travel significant distances from the point of impact up to thousands of miles (Busby and Morgan 2005). They become resuspended in air, are found in air filters in cars at some distance from the attacks, and are respirable. Because their diameters are so small, below 1000nm, they are able to pass through the lung into the lymphatic system and in principle can lodge anywhere in the body. Here they may remain for several years in the same place. The biological half life of such particulate Uranium is unknown but is very long. According to research with animals it can be greater than 13 years (Royal Society 2001).

A single Abrams 120mm tank shell contains about 3kg of DU (111MBq of radioactivity) and there is 275g in a 30mm GAU3A A-10 Thunderbolt Gatling Gun round. These munitions were used in Gulf War 1. More recently evidence has emerged that hard target warheads have been deployed on cruise missiles and bunker busting bombs, each containing up to one tonne of Uranium. Estimates of the quantity of Uranium used in Gulf War 2 in 2003 are as high as 1700 tonnes (Al Ani and Baker 2009).

Military penetrators explode on impact with hard targets with about 80% conversion to micron diameter Uranium Oxide particles of a ‘ceramic’ nature. These particles are highly mobile and extremely long lived in the environment, owing to the very high degree of insolubility of Uranium Oxides UO_2 and U_3O_8. They can be inhaled and the sub-micron diameter particles are translocated from the lung to the lymphatic system, building up in the tracheobronchial lymph nodes and potentially able to circulate everywhere in the body since they incapacitate macrophages (Kalinich *et al.* 2002). They can pass through the skin and through most gas-mask filters. Alpha and beta

disintegrations from these particles cause very high and repetitive doses to cells local to the range of the disintegration i.e. about 30 microns for the alpha and 450 microns for the beta tracks. The instantaneous (t = 0) dispersion (spectrum) of particle size from DU impacts was obtained using special cascade impactor collectors at the US Aberdeen proving grounds by Glissmeyer *et al.* (1979). The mean geometric diameter for collected behind the target were found to be 0.8μ. More recently, the EU SHER (2010) report states that 31% of the particles have mean diameters below 0.18μ. Particles of this size are effectively gaseous and can pass through the skin and penetrate to any part of the body. They are therefore not comparable to historic studies of Uranium exposures since such concentrated forms have never existed and have never been studied: they are completely new exposures that have to be assessed *de novo*.

The reason that DU is employed is that the weapons are astoundingly successful and have revolutionised warfare, rendering the tank and its armour useless. In addition, its use represents a route for the nuclear industry to rid itself of a waste product which would otherwise be expensive to dispose of. But the downside is that the material clearly represents a radiation hazard which is indiscriminate: battlefields are going to be contaminated and civilian populations are going to be exposed.

Apart from the evidence that Uranium is far more genotoxic than is modeled, which will be reviewed below, there is an immediate argument from quantity of radioactivity. The average Natural Uranium content of soil is about 10-20 Becquerels per kilogram, including all the Uranium isotopes. The average excretion of Uranium in urine is less than 10nBq l^{-1} (in the UK) as a result of absorption of natural Uranium in food and water. Pure Depleted Uranium contains about 12.4MBq of U-238 per kilogram and in Kosovo, some soil samples analysed by the United Nations Environment Program (UNEP) contained 250,000Bq/kg (UNEP 2001, Annex). The 350 tonnes of DU used in the first Gulf War represent 4.3 TBq (4.3×10^{12} Bq) of Uranium alpha activity (13.0×10^{12} if the radioactive beta emitting daughter isotopes are included). The 1700 tonnes that were used in the 2003 war, represents 63 TBq of activity dispersed mainly into a populated area of perhaps 100km^2. This gives a mean density of deposition of radioactivity of 630,000Bq/m^2. These sums are instructive and are collected together in Table 12.2.

It is possible to find a comparison to illustrate the overall radiological situation. As an alpha emitter and long-lived environmental particle Uranium can be compared with Plutonium-239 a radionuclide released by Sellafield and a major contaminant of the Irish Sea. Plutonium in the environment is also in the form of sub-micron sized oxide particles. The comparison is made in Table 12.3.

Like DU, these Plutonium Oxide particles are also long lived and mobile. Plutonium from Sellafield has been measured in autopsy specimens across the UK, in sheep droppings on the east coast of England 100 km from Sellafield at the same latitude and even in the teeth of children up to 200 km

from the site in south east England. U-238 has a very long half life, 4500 million years, so owing to its much shorter half life of 24,100 years, the specific activity of Pu-239 is far greater. It is 2.3TBq/kg. But this means that 350 tons of DU (or 4.30TBq of U-238) is equivalent in activity (quantity of radiation) to about 2 kg of Plutonium-239. The ethical dimensions of the intentional scattering of 2kg of Plutonium-239 over a populated area are easy to imagine.

Table 12.2 Mean density of deposition of radioactivity from DU in the two Gulf Wars and Kosovo including decays from U-238 and beta daughters Pa-234m and Th-234 compared with other radioactive contamination.

Event	**Activity released or estimated deposited**	**Mean activity density Bq per square metre (area)**
10 tons of DU in Kosovo	0.37TBq	3700
350 tons of DU in Iraq 1	13 TBq	130,000 (into 100 km^2)
1700 tons of DU in Iraq 2	63TBq	630,000 (into 100 km^2)
Global weapons fallout Strontium-90 (Sr-90) Northern Hemisphere lat. 50-60deg (UNSCEAR, 2000)	73.9PBq	460
Chernobyl 30km Exclusion Zone *measured* Sr-90 (IAEA)		37,000 to more than 111,000
UK North Wales Radioactive Sheep restrictions *measured* Caesium-137 (Cs-137)		15,000 to 30,000
UNSCEAR definition of contaminated area. (Cs-137)		> 37,000
Irish Sea cumulative Plutonium from Sellafield 1952-1996 [Busby, 1995]	1350TBq	20,000

Table 12.3 Comparing Plutonium-239 and Uranium-238 in the environment

	Uranium-238	**Plutonium-239**
Environmental form	0.2-2μ oxide particles	0.2-2μ oxide particles
Density of material $g.cm^{-3}$	(UO_2) 10.9;(U_3O_8) 8.3	(PuO_2) 11.46
Solubility	Insoluble	Insoluble
Environmental Longevity	Long lived	Long lived
Main radioactive emissions	Alpha + beta + beta	Alpha
Alpha particle energy	4.19MeV	5.15MeV
Half life	4.51 billion y	24400y
Specific activity	37.2MBq/kg (α + β)	2.3TBq/kg (α)
Main present contamination source	DU	Fuel reprocessing e.g. Sellafield
Mass for equal activity	175 tons	1kg

12.3 The evidence of harm from Uranium exposures

Uranium oxide nanoparticle exposure from weapons does not represent the same kind of hazard as Uranium exposures in people living in high background Uranium areas, nor those who work as Uranium miners and machinists. The exposures are quite different in quality and type. Comparisons of miners exposed to Uranium ore dusts will compare those who inhale particles which have very low concentrations of Uranium and which are fairly large. In Gulf war veterans and civilian populations the Uranium is almost pure. The local doses to and concentrations in tissue will be thousand of times greater in the case of the weapons exposures and the much smaller particle sizes will ensure the rapid internalisation of the uranium, through completely evolutionarily novel routes, though the lungs or directly through the nose to the mid brain or even through the skin. The nanoparticles will penetrate individual cells. Thus the highest concentrations will begin in the cells, where the DNA is, and concentration will fall towards the blood supply reservoir, the opposite of what happens with those who ingest uranium contaminated solutions of food. Comparing Uranium urine excretions or blood concentrations to get an idea of similar levels of exposure and making calculation on the basis of average dose conversion coefficients will also be invalid for this reason. It is an averaging problem, like all the others associated with comparing external and internal irradiation. Nevertheless, because there are overlaps, the effects of exposure to Weapons Derived Uranium will be discussed in parallel with other Uranium exposures. We should expect many of the effects found but at much lower apparent doses in the case of WDU. The above *caveat* should be borne in mind.

12.3.1 Health effects: epidemiology

Uranium is primarily genotoxic. Exposure to Uranium causes genetic and genomic changes and therefore impacts most organs in mammals. Particularly targeted are the kidney, the brain and the reproductive system. A list of reported conditions associated with Uranium exposure is given in Abu Quare and Abou-Donia 2002 and Craft *et al* 2004. Bertell 2005 has reviewed the area and drawn attention to significant gaps in knowledge and recently a number of authors have discussed the problem in a UN report (UNIDIR 2008).

The teratogenicity of exposure to Uranium weapons aerosols is reviewed by Hindin *et al* (2005). Many reports of congenital defects in children born in Iraq following the first and 2nd Gulf wars (e.g. Hamburg 2003) have not been followed up by any studies by WHO or any responsible authorities. The main reported illnesses and conditions associated with exposure to Uranium are listed in Table 12.4

It will be apparent that Uranium exposure will have a profound effect on the health of any population, and that the range of effects covers the entire spectrum of disease.

Table 12.4 Illnesses and conditions reported in the literature to be associated with exposure to Uranium.

Mutagen: Reproduction: teratogenic and genotoxic; causes lower fertility, miscarriages, heritable defects in children, stillbirths, childhood cancer and leukemia. Oestrogenic mimic with responses in humans and animals.
Mutagen: Cancer and leukemia increases in those exposed and their offspring in humans and animals.
Kidney disease generally, problems below 100ng/g contamination, glomerular and tubular lesions, tumorigenic changes, creatinine levels alter with dose, glomerular structures altered, IgE and IgG nephropathy, persistent structural and functional and functional damage.
Blood; cytotoxic and leukemogenic; reduction in red blood cells.
Brain; targets the brain and causes wide range of effects associated with damage to deep brain and brainstem fuction, effects shown by objective tests. Basis of the Gulf War syndrome. Weapons Uranium particles enter the mid brain directly from the nose.
Concentration: circulates as uranyl ion which has the same affinity as Calcium, therefore binds to and targets DNA, nervous tissue, bone, sperm. For this reason most organs will be affected (mitochondrial DNA affecting energy conversions in cells).
Chromosome aberrations found in those exposed to Uranium; the effect is out of proportion to the ICRP calculated dose for external radiation.
Mutagen: retinoblastoma rates highest in Navajo tribes living on Uranium

tailings; rates also high in offspring of Sellafield workers and near Rocketdyne site near Los Angeles contaminated with Uranium.
Mutagen: Sex ratio effects in offspring of male Uranium miners Inflammation: associated with oxidative stress at site of Uranium
Carcinogen: cancer increases in BNFL Uranium fuel-element workers

Despite this, there have been virtually no epidemiological studies carried out of populations exposed to weapons Uranium. The one exception is a study carried out at the request of the Italian military into cancer in the Balkans peacekeepers. The first report showed a significant excess of lymphoma (equivalent to 8-fold) in peacekeepers stationed in Bosnia and Kosovo (Italian report 2001). More recent investigation of the data shows that the cancers were mainly from those who served in Bosnia, making the relative risk more like 14-fold. A recent update on the situation seems to have been kept confidential; reports are that levels of cancer in this cohort are startlingly high and checks are being carried out. No credible study of cancer or birth defects in UK or US veterans has been published although parliamentary questions have elicited data which show an increase in lymphoma in UK veterans of the 1st Gulf War. Recently, a coroner's jury in the UK found that a British Gulf war veteran, Stuart Dyson, died of colon cancer because of exposure to Depleted Uranium in Iraq (Dyson 2009) and the Minister was informed under Section 43 of the UK Coroners Act. Evidence was taken from ECRR and from scientists from the UK Ministry of Defence but clearly the jury believed that the cancer was caused by the exposure.

Cancer data from Sarajevo in Bosnia has been reported, and show remarkable increases (up to 20-fold) in the incidence at many sites (Hamburg 2003). A cohort study of cervical cancer in Greece concluded that exposure to Uranium aerosols was the cause of a statistically significant increase in the disease those exposed as shown by cervical smear screening results (Papathanasiuo *et al* 2005). There have also been many reported of high levels of cancer in Iraq following the bombing both in 1991 and later in 2003, but no systematic study has been published. An early study by McDiarmid *et al* (2002) found no evidence of increased risk of cancer in US veterans of the first Gulf war, though ill health from many conditions (generally, Gulf War syndrome) was reported.

Gulf war syndrome itself was examined in a sophisticated Factor Analysis by Haley *et al* (2000) in the USA, funded by Ross Perot. The syndrome encompasses many conditions, problems which the military and their advisors in the UK blamed on stress, but which Haley identified as having in common that they resulted from damage to the brainstem and lower brain housekeeping functions. Haley went on to show that this was the case by carrying out a magnetic resonance imaging case control study of US veterans. The P32 and H1 studies identified significant loss of viability in cells in the brain associated with the housekeeping functions of the brain which were

manifesting themselves as Gulf War syndrome. Haley was not aware of the targeting of the brain and lower brain by Uranium and blamed the effects he found on exposures to organophosphates. However, research which was carried out some years after Haley's work showed the profound targeting of this area of the brain by Uranium, and the fact that inhaled Uranium has a direct access to these parts of the brain through the olfactory lobe (see below).

The situation in Iraq has become serious: genotoxicity of Uranium exposures has resulted in a catastrophic increase in cancer and congenital disease. This was reported at the September 1998 General Conference of the IAEA and has been comprehensively reviewed by Al Ani and Baker (2009).
In the same volume, these authors review other evidence of increases in genetic and genomic based disease in those parts of Iraq contaminated with Uranium and cite the many studies that report the levels of contamination and also the health indicators. However, none of these reports have been considered by the risk agencies and in addition no western based study has been carried out on the populations of Iraq in order to investigate the concerns. The Committee is currently engaged in a study of cancer and congenital birth defects in Iraq.

Statistically significant Uranium effects have been reported at the Springfields fuel fabrication plant in the UK (McGeoghegan and Binks 2000). A strong association, related to Uranium exposure, was reported for Hodgkin's lymphoma and Non Hodgkin lymphoma, though the authors did not believe the relation was a causal one since the absorbed doses were too low.

12.3.2 Genetic damage: chromosome aberrations

Chromosome aberration analysis can be used as a flag for earlier exposure to ionizing radiation. Indeed, it is possible to reconstruct the doses and make some assumptions (on the basis of the types of chromosome damage, dicentrics and centric rings) on the type of exposure, whether low or high LET (Hoffman and Schmitz Feuerhake 1999).

Unexpectedly high levels of chromosome aberrations in Uranium miners in Namibia were reported by Zaire *et al* 1997. Studies of chromosome aberrations in a set of Gulf War veterans suffering from Gulf War syndrome were also examined by Schroeder *et al*, 1999. Results showed levels of damage consistent with earlier exposures of about 150mSv although clearly these veterans could not have been exposed to more depleted Uranium than would account for a committed dose of 100μSv. Both these studies identify an error in the calculation of dose from the Uranium exposures of approximately 1000-fold. It should be noted that chromosome damage leaves the body with a half life of about 2 years, yet these Gulf veterans were showing this damage some ten years after the exposures, suggesting some depot of Uranium which was long lived. The Royal Society (2001) cite references to support the view that the half life of some types of Uranium in the body is longer than 10 years and may be considered to be perhaps indefinite. Chromosome aberrations have

been found in a case control study of New Zealand Atomic test veterans (also exposed to Uranium at the test sites) some 40 years after the exposures.

Chromosome aberration analysis in Bosnia has shown significant Uranium exposure effects in an ecological study by Ibrulj *et al* (2007). The study evaluated peripheral lymphocytes from 84 individuals split between inhabitants of Hadzici where NATO strikes involved Uranium (and UNEP measurements showed presence of Uranium in 2002) and a control area where there was little exposure. Results showed a statistically significant increase in chromosome aberration frequencies in the exposed group in 2007, some ten years after the attacks. Micronuclei were also increased in peripheral lymphocytes in the same populations exposed to Uranium (Ibrulj *et al* 2004).

Hadzici in Bosnia was also studied by Krunic *et al* (2005) to evaluate the genetic damage to those who were exposed to Uranium weapons. The authors were able to show excess micronuclei in peripheral lymphocytes compared with controls from west Herzegovina.

In cell culture experiments, Miller *et al* 2002 were able to induce dicentric chromosome changes and neoplastic transformation in human cells exposed to depleted Uranium at 50μM (i.e.200ng/l) for 24hrs. This is a very low concentration and the presence of alpha emissions per cell is stochastically absent. Using different Uranium isotopes the study showed that there was a specific activity related effect and the conclusion was that radioactivity can play a role in the neoplastic transformation frequency. Nevertheless, the exposure was so low that this result supports the argument for secondary photoelectron enhancement outlined in Chapter 6 and reviewed below.

From these studies it can be concluded that Uranium exposure causes chromosome damage and micronuclei formation in human populations at levels of radiation exposure (conventionally assessed) which are more than 1000 times too low to explain these effects. Similar results have been reported from laboratory research on cell cultures (Darolles *et al* 2010). The authors find differences between enriched and depleted Uranium in cell culture studies at quite low concentration levels. Reduced to basics, depleted Uranium causes aneuploidy and micronuclei formation whereas the enriched Uranium causes chromosome aberrations. This would, of course, be an expectation of the two types of action implicit in the discussions of mechanism above. Owing to the higher activity of U-235 the main chemical species in solution is uranyl U-238 in both the enriched and the depleted Uranium experiments. Therefore there will be significant binding of the U-238 uranyl to the chromosomes resulting in destruction of whole chromosomes through the photoelectrons emitted throughout their length at the binding sites of the U-238 atoms along the phosphate backbone. The U-235 effects are then the normal alpha track high ionisation effect where a chromosome is cut (double strand break) and recombines anomalously to give the aberrations which are found. The authors (who are associated with the French IRSN) point out that the aneuploidy

produced by U-238 is associated in other reports with cancer induction and they call for a reassessment of the carcinogenicity of Uranium.

12.3.3. Reproductive and transgenerational genetic effects

The teratogenic effects of Uranium exposures have been reviewed by Hindin *et al* (2005) who concluded from the evidence that Uranium represented a teratogenic hazard. Certainly many reports have emerged from areas where Uranium weapons have been employed showing that there follow major increases in stillbirth, and congenital malformations of a particularly alarming and unusual kind. Despite these, no credible western studies have been commissioned or carried out. A case control study of UK Atomic Test Veterans children and grandchildren identified a 9-fold excess of congenital conditions in the children and an 8-fold excess in the grandchildren relative to national controls (Busby and de Messieres 2007). These veterans were exposed mainly to Uranium since their gamma film badge doses were in general known and analysis showed the existence of significant quantities of Uranium on the test sites.

A review of the reproductive toxicity of natural and depleted Uranium by Domingo (2001) concluded that Uranium was a development toxicant when given orally or subcutaneously to mice. Decreased fertility, embryo toxicity, teratogenicity and reduced growth were shown to occur. Paternain *et al* (1989) had already showed developmental and birth outcome effects in mice at doses as low as 5mg/kg with no zero effect dose. A study of the effects of Uranium on the hatching success, development and survival in early stages of zebrafish (*danio rerio)* was reported by Bourrachot *et al* (2008). The authors used levels of depleted Uranium in the water of 200-500μg/l (about $3Bql^{-1}$) but also employed a higher specific activity Uranium isotope U-233 to examine the effects of what they believed to be chemical rather than radiological stress. Both regimes showed significant developmental effects at the lowest exposures. $250\mu gl^{-1}$ showed a 43% reduction in median hatching times relative to a control. A 15 day exposure to this concentration of depleted Uranium gave a 100% mortality at the pro-larval stage. The more radioactive U-233 was more effective, but both isotopes showed the effects at this very low concentration. The radiation doses at which this was occurring are vanishingly small and would not be considered harmful on the basis of current risk models.

Raymond-Whish *et al* (2007) found that drinking water below the US EPA standard caused estrogen receptor dependent responses in female mice. The authors exposed pregnant female mice to drinking water containing from $0.5\ \mu gl^{-1}$ to $28mgl^{-1}$ and found estrogen receptor effects including selective reduction of primary follicles, increased uterine weight, greater uterine luminal epithelial cell height and other conditions. Mouse dams that drank the Uranium containing water had morphologically normal pups but these had fewer primary follicles than pups from dams that drank normal water.

12.3.4 Kidney

The kidney has been identified as a target for Uranium toxicity by many studies; the early research is reviewed in the Royal Society reports (RS2001, 2002). More recently interest has followed concerns relating to weapons exposures and research has focused on the levels needed to produce nephrotoxic effects. A number of relevant studies are listed in Table 12.5.

A most relevant and interesting report by Ballardie *et al* 2008 presents the results of a comprehensive medical and physical analysis of a veteran of the Balkans who presented with a range of kidney conditions and many Gulf war syndrome conditions. Rather than assuming that this man's spectrum of conditions was a result of stress, a team of doctors and scientists at the Manchester Royal Infirmary and the University of Sheffield set about analyzing everything they could in order to try and discover the cause. By biopsy analysis they discovered that his kidney was contaminated with enriched Uranium, which was uniformly disseminated throughout the mitochondrial tissue. Treatment with heavy metal chelating agents effected a cure. This is a major piece of evidence in the arguments which the Gulf War and Balkans veterans have regarding the origin of their ill health and was significant in persuading the jury about causality in the above-mentioned coroner's inquest on Stuart Dyson who also suffered from Gulf War syndrome before dying prematurely from colon cancer.

Table 12.5 Recent studies of relevance to the effects of Uranium on kidney structure and function

Study	**Results**
Prat *et al* 2005	Identified a set of 18 genes which were deregulated following exposure to Uranium; the Calcium pathway is heavily implicated; nephroblastoma genes implicated
Berradi *et al* 2008	Rats exposed to 40mg/l DU in water for 9 months. Kidney deterioration and lower red blood cell counts (renal anemia).
Goldman *et al* 2006	Investigated effects of DU on rat kidney brush border vesicles. Uranyl at 140μg /mg protein reduced ability to transport glucose.
McClain *et al* 2002	Effects of embedded fragments of DU (shrapnel) in rodents. Uranium from implanted fragments found in bone, kidney, muscle and liver distant from the site of implant. Alters neurophysiological parameters in rat hippocampus, crosses the placental barrier, enters foetal tissue. Decreased rodent litter size when animals bred 6 months

	after implantation. No kidney effects found suggesting adaptation.
Fukuda *et al* 2006	Toxicity and biochemical markers in rats exposed to Uranium at 0.2, 1 or 2μg/g animal. Measurable changes in many markers in bone and kidney at the lowest doses.
Zhu *et al* 2008	Renal dysfunction after long term chronic exposure to Uranium pieces surgically implanted in rats.
Zimmerman *et al* 2007	Clinical chemistry and microscopic renal effects in rats exposed to single injection IM of 0.1, 0.3 and 1.0 2μg/g animal. Nephrotoxocity seen at all doses.

12.3.5 Brain

The effects of Uranium on the brain have only recently emerged. As already outlined above, the studies by Haley demonstrated a link between lower brain function and the spectrum of conditions which make up Gulf War syndrome. Inhalation of Uranium nanoparticles from the weaponised aerosols provides a direct route to the lower brain through the physiological connections with the nasal passages and olfactory bulb. The French (IRSN and other) studies were perhaps the first to show the accumulation of Uranium in nervous tissue, to which it seems to have an affinity, probably because of the similarity of the uranyl ion to Ca^{++}. Monleau *et al* (2005) of the IRSN laboratory in France showed that Uranium concentrations in the brains of rats exposed by inhalation were as follows: olfactory bulb> hippocampus> frontal cortex> cerebellum. Uranium is normally excluded from the overall system by a low gut transfer factor. Evolutionarily there will never have been a period when aerosols of pure Uranium existed in the environment and even Uranium miners will not be exposed to the same extent since the dusts in the mines have very low Uranium content. A list of recent studies is given in Table 12.6.

It is clear from the results of Lestaeval *et al* 2005 that at levels where there is no nephrotoxicity, there are measurable changes in behaviour in rats exposed to 144μg/kg. by injection. Taken together, these studies strongly suggest that Gulf War syndrome is an effect of inhalation of micrograms of Uranium and draw attention to the extraordinary neurotoxicity of the material.

Table 12.6 Recent studies of neurological effects of Uranium

Study	**Results**
Monleau *et al* 2005 IRSN, France	Inhalation of Uranium by rats. Uranium concentration in brain: Olfactory bulb> hippocampus> frontal cortex> cerebellum. Behavioural changes shown
Barillet *et al* 2007	Oxidative stress and neurotoxicity in adult male zebrafish exposed to U-238 and U-233 in water. Oxidative stress and

IRSN, France	neurophysiological changes (increase in ACh) in exposures to both isotopes
Pellmar *et al* 1999	Depleted Uranium fragments implanted in rats and caused electrophysiological changes in hippocampal slices
McDiarmid *et al* 1999	Gulf war veterans studied found subtle effects on reproductive and central nervous system function
Briner and Murray 2005	Rats exposed to drinking water containing 75 or 150mg/l DU. Behavioural changes after 2 weeks; increased lipid oxidation
Lestaeval *et al* 2005 IRSN France	The brain is a target organ after depleted Uranium exposure. 144μg/kg injection in rats caused at kidney levels of 2.6 μg/g. This level would be normally seen as a sub toxic dose to the kidney. However, this was associated with decrease in food intake and sleep wake cycle disturbance.
Barber *et al* 2005	Short term kinetics of Uranium in rat brain after intraperitoneal injection 1μg/g animal. Uranium entered the brain rapidly and was initially concentrated in the hippocampus and striatum. Clearance was slow; contents of hippocampus, cerebellum and cortex was still high after 7 days

12.4 Animal studies, cell cultures and mechanisms

The ICRP-based desk analyses (Royal Society, WHO, SHER, RAND, ATSDR etc.) which employ absorbed dose and use risk factors for cancer culled from the Japanese A-Bomb cohorts do not predict the observations and must now be abandoned. Clearly Uranium exposure is much more hazardous. Cell culture and animal experiments have provided useful information for developing and understanding of the mechanism involved. What all these studies seem to show, is that internal Uranium exposure, to particles but also to ionic forms, seems to be acting as if it were considerably more radioactive than it is on the basis of its intrinsic radioactivity. Thus U-238 exposure causes oxidative stress, genomic instability, chromosome damage, micronuclei formation, all consequences of ionizing radiation exposure, yet in some experiments the concentration is so low that there is stochastically no radiation exposure because there are too few decays. This finding has been variously interpreted as suggesting a chemical mutagenic effect, a heavy metal effect, or a synergy between radiation and chemistry. Of course, one re-discovery is the affinity of Uranium for DNA phosphate. The affinity of the uranyl ion, UO_2^{++} for Calcium Ca^{++} sites was known in the 1960s when the substance began to be employed as an electron microscope stain. The affinity constant was measured in an elegant flow experiment by Nielsen *et al* in 1992 and was of the order of $10^{10}M^{-1}$. This would suggest, in mass-action equilibrium terms, that at quite

low concentrations (100ng/l) there is a significant amount of Uranium bound to the phosphate backbone of the DNA. This seems to agree with the experimental observations of biological effects reviewed here. The ECRR model is particularly concerned with radionuclides which bind to DNA (Strontium-90, Barium-140) since these beta emitters decay into the DNA and also change their charge and transmute into a radioactive daughter producing an ion and perhaps Auger electrons. The charge change alone will cause an ionization on the DNA. It seems that Uranium is therefore in this category, which would result in a weighting (see Chapter 6).

But there is also the fact that Uranium has a high atomic number and would therefore amplify natural background gamma radiation (and also the photon radiation which it, itself, produces, in addition to any photon radiation from other Uranium isotopes present in any mixture. The conclusion of the Committee is that such a mechanism is capable *on its own* of explaining the many anomalous findings reviewed in this chapter and in this section. The extent of the enhancement must await experimental investigation, but these experiments are straightforward, involving simultaneous exposure to Uranium and to X-rays of various energies. The use of dilute uranyl salts as an enhancing agent for X-ray targeted radiotherapy for cancer was suggested in a British Patent Application in 2007 (Busby 2008). It is clear from the studies that significant binding in vitro occurs at 200μM or 84ng/l. This concentration is not currently considered toxic but is in the same range as that found in many drinking waters and in the urine of Gulf veterans.

A list of some studies which bear on the issue of the mechanism for the anomalous enhancement of Uranium both as ionic and as particulate is given in Table 12.7.

Table 12.7. Studies of Uranium effects in cell culture and in animals which reveal information on possible mechanisms for its anomalous hazard.

Study	**Result**
Gueguen *et al* 2007	Drug metabolism is altered following exposure of DU to rats; induces expression of CYP enzymes
Miller *et al* 2005	Leukemic transformation of haematopoietic cells in mice internally exposed to DU pellets.
Miller *et al* 1998	Transformation of human osteoblast cells to tumorigenic type after exposure to DU; 0.0014% cells were hit by alpha particles. Suggests no radiation effect.
Miller *et al* 2002	Showed both Uranium and tungsten capable of causing micronuclei in human osteoblast system and tumorigenic transformations.
Yang *et al* 2002	Malignant transformation of human bronchial epithelial cell by exposure to Uranium; DU shows carcinogenesis *in vitro*

Kalinich *et al* 2002	Depleted Uranium induces apoptosis in mouse macrophages
Gueguen *et al* 2006	Hepatic effects of Uranium on liver metabolism enzymes
Pariyakaruppan *et al* 2006	Uranium causes oxidative stress in lung epithelial cells
Grignard *et al* 2008	Contamination with depleted or enriched Uranium differently affects steroid metabolism in rats
Tissandie *et al* 2006	Short term DU exposure affects vitamin D metabolism in rats
Yazzie *et al* 2003	Uranyl acetate causes DNA single strand breaks *in vitro* in the presence of ascorbate. Suggests that affinity for DNA is greater than affinity for ascorbate.
Busby 2005a	Suggests and attempts to quantify secondary photoelectron effect for Uranium bound to DNA phosphate. Draws attention to affinity of Uranyl for DNA.
Busby 2005b	As above for Uranium particles
Stearns *et al* 2005	Induction of hprt mutations and DNA adducts in Chinese Hamster ovary cells at 200 μM (80ng/l).
Busby and Schnug 2008	Discusses SPE for Uranium in ionic form as explanation for observed effects
Elsaesser *et al* 2007	Monte Carlo simulations of Uranium, Gold and water nanoparticles of different sizes confirm the enhancements due to SPE
Wan *et al* 2006	In vitro immune toxicity of depleted Uranium: effects on mouse macrophages. At 50 and 100μM. Macrophage activity altered at 200μM for 2 h.
Pattison *et al* 2008	Monte Carlo simulation of Uranium particles in tissue confirm SPE effect is 'significant' but lower than suggested by Busby.
Hahn *et al* 2002	Implanted DU fragments cause soft tissue sarcomas in the muscles of rats.
Darolles *et al* 2010	Different toxicological profiles of depleted and enriched uranium: U235 causes chromosome aberrations (alpha) U238 causes aneuploidy (photoelectron toxicity explains this spectrum)

12.5 Conclusions

It is necessary to conclude that Uranium represents a perfect example of the problem resulting from the physics-based approach to radiation risk which

ECRR2003 drew attention to. When doses are calculated in terms of absorbed dose following ICRP, the quantities of Uranium usually found in the environment confer very small doses compared with natural background gamma radiation, and even smaller when compared with the levels of dose which correlated with cancer in the A-Bomb groups. But it is clear that this approach is massively in error, since it has avoided or, more accurately, knows nothing about, chemistry, biology, physiology and pharmacology. These sciences were historically considered of less importance than physics and mathematics, in some deeply felt (by the physicists anyway) philosophical and emotional way. This is the flaw in rational analysis: it is only as good as its data, and if, in order to solve a problem, it has to be reduced to the level where a solution can be claimed, the answer is often wrong.

The Committee has had to deal with this very real problem by presenting a real solution; in this case the solution is to weight Uranium exposures by a factor of 1000 at normal background gamma photon levels (100nGy/h). This will be modified when experimental results of Secondary Photoelectron effects become available. It is clear that the effects of Uranium are wide ranging, and so to consider only genetic effects from Uranium exposure would be quite wrong. In addition, different types of exposure will cause different spectra of conditions.

In the case of conventional estimates of risk from internal Uranium, which essentially compare it with external doses, the errors are arguably greater than for any other material. There is now sufficient evidence to treat Uranium aerosols as if they had infinite biological effectiveness. The Committee therefore believes that using a risk factor to assess causality in Uranium-exposed populations or individuals should be done with extreme caution, even if that risk factor has been modified by application of a weighting that approximates observation.. If a disease or condition or genetic heritable effect of any kind is seen to increase after exposure to Uranium, causality should not be ruled out whatever the dose differential between a population before and after the exposure, or between exposed populations relative to unexposed controls.

13
Risk of Exposure: Non-Cancer Risks

13.1 General health detriment

The Committee considers that the ICRP's concentration on cancer as the main outcome of radiation exposure is inadequate for the purposes of public protection. This is clear from all the data and is particularly true for Uranium exposures. The fundamental biological mechanisms of radiation action are now well established, and these clearly predict general harm to the organism at all doses. DNA damage to cells, which occurs at the lowest doses and which may become enhanced through a number of mechanisms not addressed by ICRP, must cause general and specific health detriment to the organism, even if this is not measurable epidemiologically. Thus the Committee notes the reports which argue both for and against non-cancer effects in human populations but feel that arguments from the cellular level demand that the general harm of exposure to ionising radiation be broadly assessed.

Arguments relating to natural background exposures have been shown to be quantitatively in error for cancer and there is evidence that they are also in error for other more general health indicators. However, general health detriment suffered throughout a lifetime is difficult to quantify within a system where other factors are confounding the analysis. For example it is very likely that weapons fallout exposures will be the cause or a main cause of general ill-health and non-specific life shortening in the cohort who were exposed at or around birth, although little work has been done on this problem and as far as mortality rates are concerned it is too soon to say if the early elevated age specific mortality rates will continue. This problem has already been addressed for cancer, and indeed the contemporary breast cancer epidemic has been correlated with this exposure by Sternglass in 1994 and Busby in 1997. However, it may be difficult to resolve the issue since data informing non-specific ageing and more general health detriment are confounded by advances in health care and improvements in social conditions and therefore the effects of radiation are very difficult to establish. This does not mean that there are none. Therefore, the approach taken by the Committee is to decide on risk factors for those categories of harm which can be measured and, in the absence of any hard data, to extrapolate the data on infant mortality and other indicators to a mean life quality reduction factor which would operate on a broad spectrum of morbidity and would feed through to premature death in a system where other factors remained constant. One change that has taken place since the ECRR2003 report is to advise on a specific risk factor of $0.05Sv^{-1}$ for heart disease.

13.2 Foetal development and infant mortality

Global weapons fallout caused infant mortality, largely through the operation of foetal development defects of the heart and circulatory system. It may be assumed also that there was an increase in early foetal death, although figures for this effect are not available.

The ground-breaking work of Luning *et al.* in 1963 on foetal development in the offspring of Strontium-90 injured male mice has never been adequately or credibly followed up. The Committee finds it unacceptable that these critical findings have been ignored despite their applicability to human populations. In a very large study Luning *et al.* injected small quantities of Strontium-90, a major component of fallout, into male mice and mated them within an hour to females. The pregnant females were killed just before term to establish the extent of foetal death in the offspring *in utero*. Controls were injected with sodium chloride or the other fallout isotope Caesium-137. Results showed a significant increase in foetal death in the Strontium-90 group but no effect in either control. In a further series of experiments Luning went on to mate surviving males with untreated females to show that there was also a significant foetal death rate in the second generation. There are only two other published works which examine the genetic effects of Strontium-90 in mammals. The first, a Russian study by Smirnova *et al.*, employed rats in the same system and confirmed the effect; pathology of the dead foetuses showed that the deaths were caused by heart development defects. The second study, by Satsuda, showed increases in leukemia in live survivors. This is less relevant to the present chapter, but pointed to a transgenerational effect.

Increased infant mortality over the period of the peak period of global weapons fallout (1959-63) was first reported by Sternglass, using time-series analysis, for the USA and then for England and Wales. Since then the effect has been confirmed by Whyte, Busby and most recently by Koerblein, who examined the effect in Germany. In a separate study Busby was able to show a very high degree of correlation of infant mortality from heart and circulatory system defects with Strontium-90 contamination. The effects were mainly in early neonatal and stillbirth mortality and in the UK they were sufficiently alarming for the government to order a confidential inquiry by the Medical Research Council in 1966. This was finally published in the mid 1980s and was unable to find a cause for the effect although no attempt was made to connect it with radiation exposure.

The level of the effect in England and Wales, together with the known doses to the parents, enables the Committee to establish a risk factor for infant mortality following exposure to Strontium-90. The risk factor for foetal death is not easy to establish since 1959-63 was a period when the birthrate was fluctuating rapidly due to the effects of the large population peak from the Second World War baby boom. However, the Chernobyl accident exposures caused sharp depression in the birthrate in many countries and this was

established by Bentham for Wales and Cumbria, parts of the United Kingdom where doses were well established (though the doses of Strontium-90 were quite low). The Committee has therefore used this data to decide on a risk factor for early foetal death which is presented as an approximation in the absence of better data.

The risk factors chosen by the Committee for infant mortality and foetal death effects are given in Table 13.1 The Committee recognises that infant and foetal mortality effects are unlikely to follow a linear dose response, owing to the death of the foetus at many stages in its development and its probable response to different biochemical and biophysical (particulate) aspects of the exposures (see Fucic *et al* 2007). Thus the risk factors are based on percentage excess rates per mSv (ECRR) annual exposure to parents and are for the exposure range 0-5mSv. The exercise is intended to make clear the cost of radiation exposure of the foetus and parent and to add this into the general health cost of exposed populations.

In support of the risk factors adopted by the Committee, recent correspondence from Yablokov has revealed data which are relevant to the assessment of the infant mortality yield following exposure to fission isotopes. These support the estimate of 20-40% per mSv (ICRP). Two nuclear cities in the Soviet Union, Snezhinsk and Ozersk, are of the Mayak nuclear site in the South Urals. They have exactly the same population types, weather patterns and natural background radiation but suffer exposure to different doses of largely the same fission isotopes.

Table 13.1 Risk factors for infant, neonatal, stillbirth and birth rate depression

Birth effect	**Percentage increase in baseline rate per mSv (ECRR)[c] parental exposure in year of conception**	**Observed excess number per thousand live births 1963 per mSv (ICRP)[d] parental exposure**
Infant (0-1year) mortality	0.05 %	21 increase to 24 = 3
Neonatal (0-28 days) mortality[a]	0.07%	13 increase to 16 = 3
Stillbirth[a]	0.04%	13 increased to 17 = 4
Birth rate depression[b]	0.05%	-

[a] Based on Sr-90 exposure to parents in 1963 in England and Wales; [b] Based on fall in birth rate in Finland and parts of the UK after Chernobyl; [c] Dose calculated according to ECRR model and including new weighting factors W_j and W_k ; [d] Dose calculated at the time using ICRP model

Infant mortality was reported by Petrushinka *et al.*. (1999) for the period 1974-1995. Table 13.2 shows the rates which suggest an infant mortality increase of about 45% per mSv (ICRP) dose to the foetus.

Table 13.2 Infant mortality and stillbirth at the two Soviet Mayak cities Ozersk and Snezhinsk (1974-1995)

	Ozersk (n=20983)	**Snezhinsk (n=11994)**
Average effective dose mSv	1.6 (0.05-3.36)	0.98 (0.04-2.04)
Infant mortality/1000	14.9	11.7
Stillbirth/1000	7.0	5.8

13.3 Heritable Genetic Effects

Although infant mortality effects reviewed in 13.2 probably represent heritable genetic effects the ICRP only considers heritable effects which are measurable in phenotype after birth e.g. congenital defects and perhaps increases in clinically diagnosed heritable genetic diseases. Thus foetal death and infant mortality are not addressed as radiation exposure outcomes by ICRP. The ICRP risk factor for heritable genetic effects is based on the Hiroshima LSS and the Committee therefore concludes that this risk factor is deficient for the consequences of internal exposure. It is also deficient since it fails to include effects which were reported for the interim period between 1945 and 1953 by the Japanese (Kusano 1953). This Committee decision is supported by recent work in which minisatellite DNA examination of genetic damage has been applied to the offspring of those who were exposed at Hiroshima, with the result that no significant excess DNA mutation was found. Although this was presented as an opposition to the findings of minisatellite DNA damage in the children of Chernobyl, the Committee takes the contrary view that the Chernobyl findings were a consequence of internal exposure whilst the Hiroshima findings were from external exposure. Padmanabhan has shown that there were significant genetic effects following Hiroshima, but these were manifested as sex ratio changes in the study group and were discarded by the US led team because they could not explain them (see Busby 2006).

The ICRP risk factor for total Mendelian, chromosomal and multifactorial heritable genetic damage is now 2.4% per Gy in the reproducing population. This falls to 0.38% per Gy in the first generation. The Committee chooses the same value for the exposed reproductive generation but owing to the effects of transgenerational genomic instability considers the value of 0.38% too low for effects in the first generation. It notes that the calculation of dose for internal exposures will generally result in an adjustment of the dose to a value that will accurately reflect the increased risk of genetic damage found in the Chernobyl minisatellite studies. Thus a dose of 1mSv calculated by the ICRP models from Strontium-90 will be increased by the Committee's hazard

weighting factors to 300mSv by applying the values for W_k and W_j from Tables 6.2 and 6.3. This would effectively increase the numbers affected by a 1mSv exposure to Sr-90 from 0.01 per 1000 births to 5, a figure which roughly reflects both the predicted and observed infant effects and also the 7-fold increase in minisatellite mutation rates in the offspring of the Chernobyl liquidators.

13.4 Broad spectrum health detriment following low-level radiation exposure

The Committee has examined data relating to populations exposed to low level internal radiation from fission products released from Chernobyl, following Hiroshima, and also following exposures to Uranium weapons particles in the war zones of Iraq and the Balkans. It is clear that the general health detriment predicted by the cell-damage models is found in such populations. The Committee has chosen to model such general health deficit as a percentage decline in mean life quality, although in reality the effects are seen as both a reduction of lifespan and also throughout the life of the exposed individuals. They may be expressed probabilistically as well defined clinical or physiologically measurable effects in individuals who have been examined and as ill defined conditions which result in reduced quality of life. A list of conditions which are found in populations exposed after Chernobyl and in Hiroshima inhabitants following the A-bombing was provided Malko in 1997. The conditions are very similar in spectrum to those presented by Ammash in 2000 for populations exposed to Depleted Uranium particles in Iraq. Malko's findings for adults and adolescents are given in Table 13.3, for children in 13.4.

Bandashevsky has reported significant associations between measured Caesium-137 contamination in children as measured by whole body monitoring, and cardiac arrythmias in contaminated regions of Belarus near Gomel. The non-specific effects of radiation reported for populations living in the regions of Hiroshima and Nagasaki are also of interest here; for example, in a study of morbidity in victims of the A-Bombing that has not been cited or considered by ICRP, Furitsu reports non cancer somatic effects very similar to those found in the Chernobyl affected territories. Results are shown in Table 13.5. Morbidity rates for 1232 victims of the A-Bombing were examined in the Hannan Chuo Hospital, Osaka between 1985 and 1990. Results are shown in Table 13.5.

Global weapons fallout effects on IQ and attainment scores have been studied by Oftedal for Scandinavian countries and by Sternglass for the USA. Both show a significant reduction in performance scores in children born during the peaks in weapons fallout.

The Committee has chosen a value of 0.1% per mSv exposure calculated according to ECRR models to express the reduction in general health due to exposures. This represents a 0.1% excess probability of any person suffering loss of life quality due to developing one or more somatic

illness or life-quality affecting condition during their lifetime as a consequence of exposure to 1mSv. The Committee has chosen to make the approximation that the effect will be linear with dose in the range 0-500mSv and have based this on Eyring and Stover's considerations of equilibrium harm but believe that this may be a conservative estimate and recommend research to establish a more accurate figure.

Table 13.3 Indices of somatic illness per 100,000 in adults and adolescents of 3 contaminated and 5 control regions of the Brest region in Belarus in 1990 (from Malko 1997).

Non cancer diseases	**3 contaminated districts**	**5 control districts**	**P-value**
Altogether	62,023	48,479	<.0001
Infections and parasites	3251	2119	<.0001
Endocrine, metabolism, immunity	2340	1506	<.001
Psychic disorders	2936	2604	<.01
Chronic Otitis	250	166	<.01
Circulatory system, hypertension, ischaemic heart disease	12060	9300	<.001
Of which (above) stenocardia	1327	594	<.01
Cerebrovascular	1981	1363	<.001
Respiratory	2670	1789	<.001
Digestive organs, e.g. ulcers, chololelitic, cholecystitis	7074	5108	<.001
Urogenital, nephritis, nephroses, kidney infections	3415	1995	<.001
Female infertility	84	56	<.01
Skin diseases, dermatitis, eczema	3377	2060	<.001
Osteomuscular, osteoarthritis	5399	4191	<.001

13.5 Accelerated ageing

The non-specific effects discussed in the previous section may be seen as a general accelerated ageing effect. Indeed, the accumulation of somatic genetic damage which is the inevitable consequence of exposure would be indistinguishable from the similar accumulation of somatic genetic damage associated with natural ageing. Both are associated with evidence of somatic genetic damage, e.g. chromosome aberrations. The focus of research into the effects of ionising radiation has historically been cancer incidence and mortality. However, it has been known for many years that the trend in incidence of most cancers and the incidence of age-related processes including death are both best modelled as a logarithmic survival function. For ageing this function is named after Gompertz. A mathematical description of radiation damage given by Eyring and Stover for beagle dogs exposed to Plutonium

involved opposing rate processes where damage and repair are balanced until the repair systems are overwhelmed over time by accumulated damage. The functions are equally applicable, with different coefficients, to natural ageing processes and the Committee believes that this effect of radiation exposure must be included in any discussions of policy.

Table 13.4 Indices of somatic illness per 100,000 in children of 3 contaminated and 5 control regions of the Brest region in Belarus in 1990 (from Malko 1997).

Non cancer diseases	**3-contaminated districts**	**5-control districts**	**p-value**
Altogether	68725	59974	<.01
Infectious and parasitic	7096	4010	<.01
Endocrine, metabolism	1752	1389	<.01
Psychic	2219	1109	<.01
Nervous system and sense organs	4783	3173	<.01
Chronic rheumatism	126	87	<.01
Chronic pharyngitis, sinusitis	117	83	<.01
Digestive organs Includes chronic gastritis And cholelitic, cholecyctitis	3350 129 208	2355 40 61	<.01 <.01 <.01
Atopic dermatitis	1011	672	<.01
Osteomuscular and connective	737	492	<.01
Congenital malformations Includes heart and circulation	679 306	482 242	<.01 <.01

Bertell has addressed the issue of accelerated ageing epidemiologically. She studied the effects of low-dose, high dose-rate medical X-rays compared with the natural background component of ageing, and found, among other things, that there is no acceptable dose rate factor reduction for low-dose rate. She suggested that the effect was due to breakdown of intercellular communication through small mutations accrued over time due to background radiation, or quickly through medical X-rays. The mutations are not perceived by the individual until they accumulate. As a yardstick, Bertell used the natural rate of increase of non-lymphatic leukemia in a large population (3 million followed over three years in the US Tri-State leukemia survey), which followed a compound interest type increase from age 15 years, at about 3% per year, as a yardstick. Her question was: how much medical X-ray exposure would increase the non-lymphatic leukemia rate by the same amount as one year of natural ageing does? The two turned out to be equal, although the dose rate from background is very much slower.

The concept of accelerated ageing is supported by the discovery of the epigenetic phenomena associated with genomic instability.

Table 12.5 Comparison of morbidity rates (%) of the A-Bomb victims and of the general Japanese population (Furitsu, 1994)

Non cancer disease	**A-Bomb victim sample morbidity rate %**	**Japanese population morbidity rate %**
Lumbago	29	8
Hypertension	24	15
Ocular disease	18	3
Neuralgia, myalgia	12	2.5
Anaemia, leukopenia	12	1
Dental disease	10	<1
Gastro-duodenal ulcer	9	2
Ischaemic heart disease	9	2
Liver disease	8	1
Diabetes mellitus	7	3
Nephritis, urethral infection	5	1
Skin disease	5	2
Bronchitis, pneumonia	5	0.8
Cardiac arrhythmia	5	<0.1
Cholethiasis, pancreatitis	4	1

13.6 Effects of radiation on the general environment

The Committee emphasises that even from the most anthropocentric viewpoint (as discussed in the chapter on ethics) people cannot be considered to be independent of the environment that supports them. Harmful effects of exposures on living creatures, plants and ecosystems must be prevented if only through human selfishness. Environmental discharges from nuclear processes result in much higher doses to creatures which are in contact with them: thus discharges to the sea result in very large doses to marine creatures many of which concentrate radionuclides and thus receive very high doses. If such doses are known to cause health detriment in people and in animal and cell studies, marine organisms must suffer similar effects since they are composed of similar cells which operate their living processes in very similar ways (see e.g. Jha 2006)

At the gross level there are reports of increases in skin and other cancers in fish caught in the Irish Sea where they are exposed to discharges from the Sellafield plant. Because these reports have become widely known the Irish Sea fishing industry has suffered enormous economic damage - an effect known in Ireland as ‘Sellafield Blight’. In County Louth, the nearest part of Ireland to the Sellafield plant, ‘Sellafield Blight’ affects the ability of farmers to sell produce and the use of beaches for recreational purposes. Beaches in

Cumbria are now no longer used by people for recreation and indeed on occasion have been ‘red flagged’ by the nuclear industry.

In addition to such obvious and socially significant effects, the Committee is aware of research which indicates major effects of exposure which have been largely ignored. Some examples will be given. For example, the sharp decline in fish stocks in the northern hemisphere in the late 1960s has been conventionally ascribed to over-fishing. Sternglass has suggested that some if not all of it may be a consequence of radiation exposures from weapons fallout. If even part of this suggestion is true, the resulting consequences of the testing and by implication, of discharging materials to the sea, are not accounted for in the cost-benefit analyses supporting nuclear projects. They may be massive. The post-war period also saw a very large reduction in bird populations, an effect which was seen also after the Chernobyl accident. One of the most startling examples of this has been the entire loss of the black-headed gull population from the Ravenglass estuary near Sellafield. Research suggested that the egg shells were affected by the discharges from Sellafield but experimental work showed that the effect was not due to external radiation but must be some consequence of one of the internal nuclides, perhaps Sr-90 or Ba-140 exposure in the shell. Lobster carapaces in the Irish sea have been recently shown to concentrate the isotope Technetium-99 to a very large extent. Some lobsters with over 100,000Bq/kg of this isotope have been tested. The isotope Strontium-90 has been shown to cause genetic effects in many animal and plant systems. For example, Ehrenberg showed genetic mutations in wheat at very low doses of Sr-90.

Results of research into contamination of the Irish Sea carried out in the late 1990s showed significant affects on coastal communities of the radionuclides which contaminated that shallow and constrained water body. Nevertheless, Caesium does flow out of the northern and southern channels and coastal levels are now falling, and are less than 20Bq/kg in sediment at the highest points. The situation is much worse in the Baltic Sea where there is virtually no exit and radioactive material from weapons and Chernobyl fallout combines with radionuclides from the various nuclear plants in Sweden, Finland and Russia to aggregate in the sediment and biota. The HELCOM reports show levels of Cs-137 in sediment at 1000Bq/kg, more than ten times higher than levels found in the Irish sea at the peak of Sellafield discharges. ECRR has opened an office in Sweden to begin researching the effects of these levels of contamination.

There are meteorological implications of radioactive discharges also. An interesting suggestion has been that the large quantities of the radioactive gas Krypton-85 released following fission may be a contributing factor in the thinning of the ozone layer, since its ionisations will result in more rapid breakdown of molecules in the stratosphere which absorb the far ultraviolet solar radiation. Krypton-85 has also been cited as an agent which will alter the

normal conductivity of the atmosphere and thus alter the processes which affect weather patterns.

These examples, involving social, psychological and physical damage, suggest that discharges may have effects which must be included in any assessment of the consequences of any release. However, such research as has been done to establish the health consequences in non-human species suffers, as Pentreath has pointed out (Pentreath 2002), from lack of scientific credibility and of common terminology at even the most basic level.

The Committee believes that arriving at an understanding of the impact of environmental discharges is a massive project which is beyond the scope of the present recommendations. However, two general points must be made:

1) People are part of the environment, and human mortality and morbidity at low dose has been studied more closely and more consistently than any other species. The existing evidence described in these recommendations indicates that releases currently thought to be trivial do in fact confer unacceptable risks. It follows that, as far as regulation is concerned, it may be unnecessary to determine the effects on non-human species. The exception to this is practices which cannot be avoided, such as dealing with waste and contaminated land. The types of exposure associated with such practices should determine the emphasis and direction of research.

2) Present indications are (ICRP 2002) that research on non-human impact will be dominated by the assumption of a linear dose response and mathematical modelling. The alternative is to look in the real world using ecological studies comparing populations in contaminated areas with those less contaminated. If the mistakes of the past are to be avoided these studies will have to be undertaken using protocols which will render their results scientifically credible. Since there is no dispute about the similarity of effects in human and non-human species the responsible agencies should come to a more coherent position on the ability of epidemiological studies to inform on low doses than ICRP currently has (see ICRP 1999) and resolve their problems with ecological/ correlation studies, in which, according to the UK NRPB's Colin Muirhead (NRPB 2001) *it is not possible to tell from the data available whether the infants who developed leukemia received higher radiation doses than healthy infants in the same areas.*

14
Examples of Application

14.1 Introduction

The ECRR model partitions internal and external doses. For internal doses, it discriminates between specific isotopes and between distributions of those doses depending on whether they arise from atomic (molecular) forms of the isotopes as against sub-micron and micron sized particles.

In Table 10.6, asterisks denote isotopes where the route of exposure or exposure type will carry excess weightings under W_j and W_k due to their ability to cause mutation through mechanisms not considered by ICRP. Some of these isotopes and their weightings are given in Table 14.1

Table 14.1 ECRR weightings for weapons fallout internal exposure isotopes

Isotope	**Weighting**	**Note**
H-3	10	Transmutation/ Hydrogen bonding amplification
C-14	5	Transmutation and enzyme amplification
Sr-90	300	DNA binding (10) and Atomic Second Event (30)
Pu, Am	300	Insoluble particles
Ce-144	50	Insoluble particles
Ru-106	50	Insoluble particles
U-238	1000	Secondary Photoelectrons/ DNA

The purpose of this chapter is to give an idea of the process by which the ECRR's new risk model may be used to assess the human health impact of any given exposure. Disease yields from three different exposure episodes are calculated approximately using both the ICRP and ECRR models. These episodes are:

- Mortality from global weapons fallout, mortality, morbidity and loss of life quality from the entire nuclear project up to 2000
- Chernobyl and increases in cancer in Belarus
- Chernobyl effects in Northern Sweden studied by Tondel *et al* 2004

Internal doses from specific isotopes to the various exposed populations discussed here may be unknown. Some approximations have therefore been necessary.

14.2 Global mortality yield of atmospheric weapons testing

The difference between the ECRR and ICRP mortality predictions for world populations is shown in Table 14.2. The dose figures for global fallout given by UNSCEAR were averaged over the whole planet. The true distribution of

fallout was not uniform: the northern hemisphere in general received more fallout, and levels in parts of Europe were higher still, owing to the effect of rainfall. An understanding of the real effect in Europe may be approached by taking England and Wales as an example. The measurements made by the UK authorities allowed quite accurate assessments of the fallout isotope doses. Cumulative Sr-90 doses over the period 1950-63 to England and Wales (population 46,000,000) were about 0.6mSv as estimated by the Agricultural Research Council using ICRP models.

Table 14.2 mortality and morbidity yield of weapons fallout using figures from UNSCEAR 1993 and comparing the models of the ICRP and the ECRR.

Effect	**ICRP dose mSv**	**ICRP yield Deaths**	**ECRR dose mSv**	**ECRR yield Deaths**
Cancer deaths	4.464	[a]1,116,000	104	[b]52,000,000
[c]Infant deaths	1	0	24	857,000
Life quality loss	4.464	0	104	[d]10%
Early foetal death + stillbirth	1	0	24	1,660,000

[a] *Using the risk factor 0.05/Sv due to inclusion of a DDREF of 2*

[b] *Using the risk factor 0.1 /Sv and avoidance of DDREF*

[c] *In the cohort exposed in the five years 1959-1963 assuming a 5-yr dose of 1mSv*

[d] *Averaged over the world population of 5 Bn in the lifetime of the peak 5-year exposed cohort*

Using the ECRR weightings, this global fallout figure becomes equivalent to 180mSv internal dose. In the 46 million population this translates into 46,000,000 x 0.1 x 0.18 = 828,000 extra cancer deaths over the lifetime of the exposed individuals, say 70 years. This is roughly 11,800 extra cancer deaths per year. In 1958, before the fallout exposure could have fed through to cancer mortality, there were 96,342 deaths from all cancers in England and Wales. In 1990, the figure was 144,577, a 50% increase in a population of roughly unaltered size. Thus, despite advances in cancer therapy, an extra 48,235 cancer deaths occurred. Many of these will have been a result of the increased average age of the population, however, standardisation for this shows that there has been at least a 20% increase in cancer incidence in England and Wales combined. This increase began in the 1980s in England and in the mid 1970s in Wales (where it was greater, about 35%, see chapter 10). Thus in the UK there has been an increase of about 18,000 cancer deaths per year over the 1958 number; it has a temporal spectrum consistent with the fallout trend, which is higher in Wales where the fallout was higher, and is ascribable to some cause apart from ageing of the population. That this cause is environmental was implicit in WHO statements in the 1960s relating to cancer causes and is

confirmed by the recent 2001 statement of the ASPIS conference on Kos Island. The predictions of the ECRR risk model of just under 12,000 extra deaths per year suggest that the fallout was the cause of this cancer epidemic. The higher cumulative dose for the population in Wales similarly accounts for the proportionately larger effect in that country. As pointed out earlier, this study is the basis for the ECRR approach to obtaining weighting factors for internal exposures. Its results are spectacularly confirmed by the intra-community Chernobyl exposure study of Tondel *et al* 2004 which is discussed below.

14.3 Total yield of mortality, morbidity and loss of life quality from the nuclear project up to 2000 from ICRP and ECRR.

Figures from UNSCEAR 1993 give total estimated ICRP collective dose equivalents to the world population up to 1989. Assuming that these doses, which are based on ICRP models, are accurate, this table gives a baseline for calculating the total fatal cancer yield. The sources are given in Table 14.3 and the calculation, based on ICRP and ECRR models in Table 14.4

Table 14.3 ICRP-based global effective dose commitment from nuclear project up to 1993 and approximate ECRR model effective dose commitment.
(*Source UNSCEAR 1993 Table 58)*

Source of exposure	**ICRP based collective effective dose commitment (person Sieverts)**	**[a]ECRR based collective effective dose commitment (person Sieverts)**
Global nuclear tests	22,300,000	579,800,000
Weapons fabrication	10,000	260,000
Nuclear power production	100,000	2,600,000
Radioisotope production	80,000	8,000,000
Accidents	602120	15,655,120
Local and regional doses	380000	9,880,000
Total	23,472,120 ([b]4.7mSv)	616,195,120 ([b]123mSv)

[a] *ECRR figures assume the same proportion of isotopes and internal radiation as calculated by UNSCEAR for fallout except for allowance for higher internal doses from radioisotope production.*

.[b] *based on world population of* 5×10^9 *assumed by UNSCEAR*

Table 14.4 Global consequences of exposures from nuclear project based on UNSCEAR figures up to 1989.

Effect	ICRP yield	ECRR yield
Cancer death	1,173,606	61,619,512
Cancer total	2,350,000	123,239,024
Infant death	0	1,600,000
Foetal death	0	1,880,000
Loss of life quality	0	10%

14.4 Predicted mortality yield of Chernobyl accident: caveats over linear models

UNSCEAR 1993 gave (Table 58) the total committed effective dose from the Chernobyl accident to the world population as 600,000 person Sieverts. The ICRP60 risk factor of 0.05/Sv would predict 30,000 fatal cancers in the world from this; as UNSCEAR 2000 points out, such an increase would be statistically invisible.

Gofman has used the area deposition of Cs-137 to calculate the external committed dose to the major countries of the world with significant exposure and has applied his own risk factor of 0.37 per Sievert (derived from his approach to the Hiroshima LSS data) to calculate the fatal cancer yield of 970,500 but in this calculation, no internal doses were used whatever. In any case, Gofman did not distinguish between external and internal doses (Gofman 2000).

In a report commissioned by the Belarus Ambassador to the UK, Busby (2002) has used the fallout yield of cancer in Wales to approximate an ultimate increase in fatal cancer rate in Belarus of 50%, or 25,000 extra fatal cancers a year in the population of 9,800,000 due to exposures in the first five years following the accident. In ECRR2003, for Belarus, the Committee partitioned the dose given by UNSCEAR 1993 amongst individual radioisotope exposures and applied the weightings for internal excess risk given in Chapter 6. The Committee made an approximate calculation as follows. The first year average committed effective dose to Belarus was given by Savchenko as 2mSv. If this is extrapolated to five years and one third of the dose is weighted as Sr-90 or hazardous particulates, the ECRR calculation results in an ECRR model cumulative dose of about 900mSv and a fatal cancer yield of 882,000 which the Committee assumes will express over 50 years which is 17,640 extra fatal cancers per year, roughly in line with Busby's calculation. The 70 year overall yield is 1,200,000 in Belarus alone. The same approach to the global figures estimated by UNSCEAR suggests that the overall 70 year global cancer mortality yield following Chernobyl is in excess of 6 million.

The predictions made by Busby in 2002 using the ECRR risk model, and followed by ECRR2003 were borne out by results presented by Okeanov *et*

al in Geneva in 2004. In November 2004 the *Swiss Medical Weekly* published findings by workers at the Clinical Institute of Radiation Medicine and Endocrinology Research in Minsk, Belarus. They showed that between 1990 and 2000 cancer rates had risen by 40% overall, compared with rates before the catastrophe in April 1986. Belarus has had a national Cancer Registry as long as anywhere in Britain, keeping a computer database of all new cases of malignant tumours. The Okeanov paper presented an overall comparison of changes in the incidence of cancer morbidity in Belarus. Increases in the various oblasts (regions) were:

- Brest 33%
- Vitebsk 38%
- Gomel 52%
- Grodno 44%
- Minsk 49%
- Mogilev 32%
- Minsk city 18%
- all Belarus 40%

The view of conventional radiation protection community however was predictable. On the basis of the ICRP model, based on absorbed dose, very little if any cancer had resulted or would result from the fallout. This was expressed, for example, in UNSCEAR 2000:

Apart from the substantial increase in thyroid cancer after childhood exposure observed in Belarus, the Russian Federation and Ukraine there is no evidence of a major public health impact related to ionising radiation 14 years after the Chernobyl accident. No increases in overall cancer incidence or mortality that could be associated with radiation exposure have been observed. The risk of leukemia, one of the most sensitive indicators of radiation exposure, has not been found to be elevated even in the accident recovery operation workers or in children. There is no scientific proof of an increase in non-malignant disorders related to ionising radiation. … For the most part [the public] were exposed to radiation levels comparable to or a few times higher than the natural background levels. Lives have been disrupted by the Chernobyl accident but from the radiological point of view, based on the assessment of this Annex, generally positive prospects for the future health of most individuals should prevail.

The increases in ill health reported from the Chernobyl-affected territories of the ex-Soviet Union were regularly ascribed by IAEA and WHO to ‘radiophobia’. However, publications began to emerge which showed measurable objective indicators of genetic harm in animals and in plants. The ECRR Chernobyl sub-Committee addressed the issue of lack of translation

(and consideration) of Russian language peer review publications and produced a report *Chernobyl-20 Years After* in 2006 where this evidence was presented (Busby and Yablokov 2006). This was reprinted and revised in 2009. Also in late 2009 Yablokov *et al* edited a book on the issue which was published by the US New York Academy of Sciences (Yablokov *et al* 2009). This presents a very large array of data showing increases in cancer, congenital disease and general health effects caused by the Chernobyl exposures. There is now no credible support for the assertions of the risk agencies that the Chernobyl fallout had no measurable effect. Interested researchers are referred to these volumes which present data which are largely in line with the predictions in ECRR2003.

The wide difference between the predictions of the Committee's model and those based on the ICRP approach shows the extent to which internal radiation with the enhanced ability to give high doses to individual cells can alter the expected health detriment. As in the case of global fallout, the two approaches should be easily testable by examining the observed increases in cancer in the exposed groups. However, it must be borne in mind that implicit in the modelling assumptions of the ICRP based method with weightings for the radionuclides is that there is a linear response. The Committee has made clear that this is unlikely to be the case, and therefore emphasise that the model which they apply to calculate the health detriments is only an approximate one and will be roughly linear over the narrow range it is applied. It is intended to be used to obtain a more rational and accurate assessment of risk in a population to which the collective dose model of ICRP has already been applied or where such doses are available. It is intended to represent the best rational correction currently available to historical ICRP collective dose models. Within these populations, the high dose group will have proportionately lower levels of cancer as a consequence of the biphasic or supralinear dose response relationships reviewed by the Committee. Studies of the effects of Chernobyl should therefore compare health data from before the accident with data from after the accident rather than using control groups and arguing from linear risk assumptions.

This success of the ECRR approach in Belarus was followed in the same year by a second piece of supporting evidence. A study in 2004 of cancer in northern Sweden also gave results which are largely predicted by the ECRR model.

14.5 Cancer in northern Sweden after Chernobyl.

In 2004 a report was published of work carried out by Martin Tondel as part of a PhD thesis. The report examined cancer rates small communities in northern Sweden for the period 1988-1996 in relation to measured levels of Caesium-137 area contamination from the Chernobyl fallout (Tondel *et al* 2004). The study used cancer incidence rates supplied for community areas by the Swedish

Cancer Registry and carried out various regression analyses allowing for confounding variables. The conclusion was that there was an effect of the radiation on cancer rates which was monotonic and approximately linear over the ten year period following the accident. The increase in cancer was 11% per 100kBqm^{-2} of Cs-137 contamination. Tables in HRP 1972 show that 100kBqm^{2} of Caesium-137 (photon energy 660keV) delivers a dose at 1m of 0.39μGyh^{-1}. Thus the first year annual dose from the gamma shine from this level would be 3.4mSv. Thus the 11% increase in cancer per 100kBqm^{-2} is equivalent to a 32.5-fold increase in cancer per Gy which is some 640 times greater than the lifetime risk factor of 0.05Sv^{-1} published by ICRP. It should be noted that this study does not include cancers in the exposed populations in their lifetime, but just in the 10 years after the exposures.

At first this is certainly strong support for the ECRR approach: ECRR2003 predicts such a finding. However there are two aspects which are interesting. The weapons fallout comparison based its own dose reconstruction on the UNSCEAR data which gave a dose dominated by the isotope Sr-90 which carried a weighting of 300. The fallout from the Chernobyl accident had a different spectrum of fission products. Both contained the easily measured Cs-137 as the major component, but in the case of Chernobyl, Sr-90 was not a major component at some distances from the site of the reactor. However Chernobyl fallout contained (1) Te-132/I-132 and (2) a high proportion of Uranium fuel particles although the amount that precipitated in Sweden does not seem to have been reported. It seems that the exposures in Sweden were more hazardous than the weapons fallout exposures in Wales. This would suggest that the increases reported by Tondel are already greater per dose than the weapons fallout exposures. The main increases were in the 5-year period following the exposures. They were therefore the first spike in the temporal spectrum of cancer increases following exposure (discussed earlier in this report) and it can be expected that there will be a large increase in cancer in Sweden beginning in about 2006 or earlier.

This is a prediction of the ECRR model which will be easy to follow up. This increase will probably not have the same area relation to the original contamination as rainfall mainly disperses these materials to the sea. As reported above, the Baltic Sea is extremely contaminated with radioactivity. Levels of Cs-137 in sediment are as high as 1000Bq/kg according to measurements reported by the Helsinki Commission (HELCOM). This can be compared with 100Bq/kg Cs-137 in Irish Sea intertidal coastal sediment in areas when cancer studies showed a 30% increased risk relative to inland populations between 1974 and 1990 (Busby 2002, 2004b, 2006). The Baltic Sea regional offices of ECRR in Stockholm Sweden and Riga Latvia are currently discussing a joint study to examine this issue.

15
Summary of risk assessment method, principles and recommendations

15.1 Risk assessment method

The model for establishing the health consequences of an exposure to ionising radiation broadly follows that of the ICRP except that the ECRR has recognised both theoretical and epidemiological grounds for introducing a system of enhanced hazard weighting for certain kinds of internal exposure. Therefore the basic units of dose developed and used by the ICRP for exposure are adjusted for those isotopes and exposures which carry the ECRR enhancement weighting factors. Following these adjustments, it is possible to obtain an approximate value for health detriment from fatal cancer if a linear dose response relationship is assumed for a restricted dose range approximating to the range 0-10mSv externally averaged.

The Committee emphasises that this model has been developed entirely in order to provide a convenient approximation and wishes to make clear that the dose response relationship is unlikely to be linear in most cases.

The basic method follows the following procedure:

1. Partition the doses into external and internal doses.
2. Use the biokinetic models of the ICRP to establish committed doses for the various organs and the whole body.
3. Weight the doses using the Quality Factor weightings (relative biological effectiveness) to obtain committed effective dose.
4. Partition these internal doses between the various isotopes and types of exposure (hot particle or by radionuclide).
5. Weight the doses using the ECRR weighting factors.
6. Add all the doses together, external, internal and weighted internal.
7. Multiply the result by the appropriate risk factor (e.g. for fatal cancer this is 0.1 per Sievert).
8. This gives the approximate absolute value for the risk considered over the lifetime of the exposed individual.

In many cases, the early parts of this exercise will have been carried out by one of the risk agencies and the resulting tables of doses from the various isotopes and exposures can then be adjusted according to 4-8 above. In the event that only the overall dose has been published, some approximation of the proportions of external and internal doses must be made. For the major isotopic exposures of interest, the Committee lists dose coefficients for adults, children (1-14) and infants (0-1) in Table A1.

15.2 Principles and Recommendations

1. **The Committee has developed its modelling to allow assessment of the effects of radiation exposure for the purposes of policy and regulation.**

2. **The method involves the calculation of collective doses from different types of exposure and different sources to exposed groups and the calculation, by simple rules and coefficients, of the collective averaged health detriment.**

3. **The Committee believes that the model may also be used to approximate the effects of natural background radiation and for Technologically Enhanced Naturally Occurring Radioactive Materials.**

4. **The Committee recommends that the total maximum permissible annual dose limit to members of the public involving releases of anthropogenic isotopes or natural isotopes delivered in a novel fashion should be kept below 0.1mSv as calculated using the ECRR model.**

5. **The Committee thus argues for a level of exposure much lower than the level recommended by ICRP and recognises that most undertakings associated with releases of radioactivity to the environment would be severely curtailed by the adoption of such a recommendation. However, the Committee feels that this is an area where political decisions must be made based on accurate knowledge of the consequences of those decisions.**

6. **The Committee recommends that annual exposure limits for nuclear workers should be 2mSv. Nuclear workers must be made fully aware of the likely harm to them and their offspring.**

7. **The Committee endorses the principle of justification contained in radiation safety legislation but does not believe that such justification can be made on a utilitarian basis where the costs may be borne by some whilst the benefits accrue to others: rather the rights of all individuals must be respected equally.**

8. **The Committee recommends that radiation exposures be kept as low as reasonably possible using best available technology.**

9. **The Committee recommends that all health deficits associated with exposure be included in any assessment of the policy implications of exposure and holds that the unborn should in this regard be considered as having equivalent rights to living people.**

10. **The Committee holds that environmental consequences of radioactive discharges including effects on all life forms be considered in assessing the overall deficit of any practice involving radiation exposure.**

11. **The Committee will continue to examine research on radiation exposure and health detriment and will adjust the models it has developed to reflect both radiobiological theory and observational epidemiology.**

12. **The Committee calls on all governments of the world to abandon the current ICRP based risk model as a matter of urgency and to substitute for it the ECRR2010 risk model.**

16

Members of the European Committee on Radiation Risk and Individuals whose Research and Advice contributed to the Present Report

At 1st January 2010 the following individuals are or had been members, advisers or consultees of the ECRR. Their inclusion in this list may not mean that they endorse all the contents of the report but does imply that they are convinced that the ICRP system of modelling seriously underestimates the risk from low level ionising radiation exposure from anthropogenic sources. It will be noted that many of the new members and supporters of the ECRR and its analytical model are scientists for the former Soviet Union. This is unsurprising since it is these individuals who have been in a position to see first-hand and to study the effects of low-dose exposure to internal radionuclides.

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ECRR
2010 Recommendations of the European Committee on Radiation Risk
The Health Effects of Ionising Radiation Exposure at Low Doses for Radiation Protection Purposes. Regulators' Edition.

Executive Summary

This report updates the model presented by the Committee in 2003. It outlines the Committee's findings regarding the effects on human health of exposure to ionising radiation and presents a new model for assessing these risks. It is intended for decision-makers and others who are interested in this area and aims to provide a concise description of the model developed by the Committee and the evidence on which it depends. The development of the model begins with an analysis of the present risk model of the International Commission on Radiological Protection (ICRP) which is the basis of and dominates all present radiation risk legislation. The Committee regards this ICRP model as essentially flawed as regards its application to exposure to internal radioisotopes but for pragmatic reasons to do with the existence of historical exposure data has agreed to adjust for the errors in the ICRP model by defining isotope and exposure specific weighting factors for internal exposures so that the calculation of effective dose (in Sieverts) remains. Thus, with the new system, the overall risk factors for fatal cancer published by ICRP and other risk agencies may be used largely unchanged and legislation based upon these may also be used unchanged. It is the calculation of the dose which is altered by the Committee's model.

1. The European Committee on Radiation Risk arose out of criticisms of the risk models of the ICRP which were explicitly identified at the European Parliament STOA workshop in February 1998; subsequently it was agreed that an alternative view should be sought regarding the health effects of low level radiation. The Committee consists of scientists and risk specialists from within Europe but takes evidence and advice from scientists and experts based in other countries.

2. The report begins by identifying the existence of a dissonance between the risk models of the ICRP and epidemiological evidence of increased risk of illness, particularly cancer and leukaemia, in populations exposed to internal radioactive isotopes from anthropogenic sources. The Committee addresses the basis in scientific philosophy of the ICRP risk model as applied to such risks and concludes that ICRP models have not arisen out of accepted scientific method. Specifically, ICRP has applied the results of external acute radiation exposure to internal chronic exposures from point sources and has relied mainly on physical models for radiation action to support this. However, these are averaging models and cannot apply to the

probabilistic exposures which occur at the cell level. A cell is either hit or not hit; minimum impact is that of a hit and impact increases in multiples of this minimum impact, spread over time. Thus the Committee concludes that the epidemiological evidence of internal exposures must take precedence over mechanistic theory-based models in assessing radiation risk from internal sources.

3. The Committee examines the ethical basis of principles implicit in the ICRP models and hence in legislation based on them. The Committee concludes that the ICRP justifications are based on outmoded philosophical reasoning, specifically the averaging cost-benefit calculations of utilitarianism. Utilitarianism has long been discarded as a foundation for ethical justification of practice owing to its inability to distinguish between just and unjust societies and conditions. It may, for example, be used to underpin a slave society, since it is only overall benefit which is calculated, and not individual benefit. The Committee suggests that rights-based philosophies such as Rawls's Theory of Justice or considerations based on the UN Declaration of Human Rights should be applied to the question of avoidable radiation exposures to members of the public resulting from practice. The Committee concludes that releases of radioactivity without consent can not be justified ethically since the smallest dose has a finite, if small, probability of fatal harm. In the event that such exposures are permitted, the Committee emphasises that the calculation of ‘collective dose’ should be employed for all practices and time scales of interest so that overall harm may be integrated over the populations.

4. The Committee believes that it is not possible accurately to determine ‘radiation dose to populations’ owing to the problems of averaging over exposure types, cells and individuals and that each exposure should be addressed in terms of its effects at the cell or molecular level. However, in practice this is not possible and so the Committee has developed a model which extends that of the ICRP by the inclusion of two new weighting factors in the calculation of effective dose. These are biological and biophysical weighting factors and they address the problem of ionisation density or fractionation in time and space at the cell level arising from internal point sources. In effect, they are extensions of the ICRP’s radiation weighting factors employed to adjust for differences in ionisation density resulting from different quality radiations (e.g. alpha-, beta and gamma).

5. The Committee reviews sources of radiation exposure and recommends caution in attempting to gauge the effects of novel exposures by comparison with exposures to natural radiation. Novel exposures include internal exposures to artificial isotopes like Strontium-90 and Plutonium-239 but also include micrometer range aggregates of isotopes (hot

particles) which may consist of entirely man-made isotopes (e.g. Plutonium) or altered forms of natural isotopes (e.g. depleted Uranium). Such comparisons are presently made on the basis of the ICRP concept of ‘absorbed dose’ which does not accurately assess the consequence for harm at the cell level. Comparisons between external and internal radiation exposures may also result in underestimates of risk since the effects at the cell level may be quantitatively very different.

6. The Committee argues that recent discoveries in biology, genetics and cancer research suggest that the ICRP target model of cellular DNA is not a good basis for the analysis of risk and that such physical models of radiation action cannot take precedence over epidemiological studies of exposed populations. Recent results suggest that very little is known about the mechanisms leading from cell impact to clinical disease. The Committee reviews the basis of epidemiological studies of exposure and points out that many examples of clear evidence of harm following exposure have been discounted by ICRP on the basis of invalid physical models of radiation action. The Committee reinstates such studies as a basis for its estimates of radiation risk. Thus the 300-fold discrepancy between the ICRP model's predictions and the observed cases in the Sellafield childhood leukemia cluster becomes an estimator of risk for childhood leukemia following such exposure. The factor is thus incorporated by the Committee into the calculation of harm from internal exposure of specific types through its inclusion in the weighting factors used to calculate the ‘effective dose’ to the children in Sieverts.

7. The Committee reviews the models of radiation action at the cell level and conclude that the ‘linear no threshold’ model of the ICRP is unlikely to represent the response of the organism to increasing exposure except for external irradiation and for certain end points in the moderately high dose region. Extrapolations from the Hiroshima lifespan studies can only reflect risk for similar exposures i.e. high dose acute exposures. For low-dose exposures the Committee concludes, from a review of published work, that health effects relative to the radiation dose are proportionately higher at low doses and that there may be a biphasic dose response from many of these exposures owing to inducible cell repair and the existence of high-sensitivity phase (replicating) cells. Such dose-response relationships may confound the assessment of epidemiological data and the Committee points out that the lack of a linear response in the results of epidemiological studies should not be used as an argument against causation.

8. In further considering mechanisms of harm, the Committee concludes that the ICRP model of radiation risk and its averaging methods exclude effects which result from anisotropy of dose both in space and in time. Thus the

ICRP model ignores both high doses to local tissue caused by internal hot particles, and sequential hits to cells causing replication induction and interception (second event), and merely averages all these high risk situations over large tissue mass. For these reasons, the Committee concludes that the unadjusted ‘absorbed dose’ used by ICRP as a basis of risk calculations is flawed, and has replaced it with an adjusted ‘absorbed dose’ which uses enhancement weightings based on the biophysical and biological aspects of the specific exposure. In addition, the Committee draws attention to risks from transmutation from certain elements, notably Carbon-14 and Tritium, and has weighted such exposures accordingly. Weightings are also given to radioactive versions of elements which have a particular biochemical affinity for DNA e.g. Strontium and Barium and certain Auger emitters.

9. The Committee reviews the evidence which links radiation exposure to illness on the basis that similar exposures define the risks of such exposures. Thus the Committee considers all the reports of associations between exposure and ill health, from the A-bomb studies to weapons fallout exposures, through nuclear site downwinders, nuclear workers, reprocessing plants, natural background studies and nuclear accidents. The Committee draw particular attention to two recent sets of exposure studies which show unequivocal evidence of harm from internal irradiation at low dose. These are the studies of infant leukemia following Chernobyl, and the observation of increased minisatellite DNA mutations following Chernobyl. Both of these sets of studies falsify the ICRP risk models by factors of between 100 and 1000. The Committee uses evidence of risk from exposures to internal and external radiation to set the weightings for the calculation of dose in a model which may be applied across all exposure types to estimate health outcomes. Unlike the ICRP the Committee extends the analysis from fatal cancer to infant mortality and other causes of ill health including non-specific general health detriment.

10. The Committee concludes that the present cancer epidemic is a consequence of exposures to global atmospheric weapons fallout in the period 1959-63 and that more recent releases of radioisotopes to the environment from the operation of the nuclear fuel cycle will result in significant increases in cancer and other types of ill health.

11. Using both the ECRR's new model and that of the ICRP the Committee calculates the total number of deaths resulting from the nuclear project since 1945. The ICRP calculation, based on figures for doses to populations up to 1989 given by the United Nations, results in 1,173,600 deaths from cancer. The ECRR model predicts 61,600,000 deaths from cancer, 1,600,000 infant deaths and 1,900,000 foetal deaths. In addition,

the ECRR predicts a 10% loss of life quality integrated over all diseases and conditions in those who were exposed over the period of global weapons fallout.

12. The Committee refers to new research which demonstrates enhanced radiation hazards from internalized elements of high atomic number through enhanced absorption of natural background electromagnetic radiation and its conversion into photoelectrons. The Committee identifies this effect as a major cause of the health effects of exposure to the element Uranium and creates a weighting factor for such exposures. The Committee discusses the effects of Uranium weapons on populations exposed to Uranium fallout and asserts that the anomalous health effects observed following Uranium exposures are mechanistically explained by such processes.

13. The Committee notes that since the publication of its 2003 model there have been epidemiological observations that support the model's predictions, namely Chernobyl effects in Belarus reported by Okeanov 2004 and Chernobyl effects in Sweden reported by Tondel *et al* 2004.

14. The Committee lists its recommendations. The total maximum permissible dose to members of the public arising from all human practices should not be more than 0.1mSv, with a value of 2mSv for nuclear workers. This would severely curtail the operation of nuclear power stations and reprocessing plants, and this reflects the Committee's belief that nuclear power is a costly way of producing energy when human health deficits are included in the overall assessment. All new practices must be justified in such a way that the rights of all individuals are considered. Radiation exposures must be kept as low as reasonably achievable using best available technology. Finally, the environmental consequences of radioactive discharges must be assessed in relation to the total environment, including both direct and indirect effects on all living systems.

Annex A
Dose coefficients for the main isotopes of radiological interest

The rules for calculating effective doses have been described in Chapter 6. For the main isotopes of radiological interest, dose coefficients have been calculated by the Committee using these rules and assumptions. Table A1 gives dose coefficients for low-dose exposure through ingestion and inhalation. In general, for these isotopes, the effective dose E to an individual in age group a may be calculated according to the equation:

$$E_{total} = E_{external} + \Sigma_I k(a)_{I,\ ingest}\ I_{I,ingest} + \Sigma_I k(a)_{I,\ inhale}\ I_{I,\ inhale} \quad \ldots\ldots.(1)$$

Table A1 Dose coefficients of various isotopes for low-dose exposure following ingestion and inhalation.

Isotope (form)	**Half life**	**[a]k(0-1) Sv/Bq**	**k(1-14) Sv/Bq**	**k(adult) Sv/Bq**
H-3 (HTO)	12.3y	1.0 E-9	4.0 E-10	2.0 E-10
H-3 (CHT)	12.3y	5.0 E-9	2.0 E-9	1.0 E-9
C-14	5.7 E+3y	1.5 E-8	5.8 E-9	2.9 E-9
S-35 (inorganic)	87.4d	5.0 E-10	2.0 E-10	1.0 E-10
S-35 (NS, CS etc)	87.4d	5.0 E-9	2.0 E-9	1.0 E-9
Co-60	5.27y	1.75 E-7	7.0 E-8	3.5 E-8
Sr-89	50.5d	1.3 E-7	5.2 E-8	2.6 E-8
Sr-90/Y-90	29.1y/2.67d	4.5 E-5	1.8 E-5	9.0 E-6
Zr-95/Nb-95	64.0d/35.0d	2.4 E-7	9.5 E-8	4.7 E-8
Mo-99	2.75d	1.5 E-8	6.0 E-9	3.0 E-9
Tc-99m	6.02h	5.5 E-10	2.2 E-10	1.1 E-10
Tc-99	2.13 E+5y	1.6 E-8	6.4 E-9	3.2 E-9
Ru-106	1.01y	3.5 E-9	1.4 E-8	7.0 E-9
Ru-106 μ particle	1.01y	1.7 E-6	7.0 E-7	3.5 E-7
Te-132/ I-132	3.26d/2.3h	5.5 E-6	2.2 E-6	1.1 E-6
I-131	8.04d	5.5 E-7	2.2 E-7	1.1 E-7
Cs-134	2.06y	1.0 E-7	4.0 E-8	2.0 E-8
Cs-137	30.0y	3.2 E-7	1.3 E-7	6.5 E-8
Ba-140/La-140	12.7d/40h	3.9 E-6	1.6 E-6	7.8 E-7
Pb-210	22.3y	3.5 E-6	1.4 E-6	7.0 E-7
Bi-210	5.01d	6.5 E-9	2.6 E-9	1.3 E-9
Po-210	138d	6.0 E-6	2.4 E-6	1.2 E-6
Ra-226 ingestion	1.6 E+3y	1.4 E-5	5.6 E-6	2.8 E-6
U-238 inhalation	4.5 E+9	2.5 E-3	1.2 E-3	8.4 E-4
U-238 μ particle	4.5 E+9	2.5 E-2	1.2 E-2	8.4 E-3
U-238 ingestion	4.5 E+9	2.5E-4	1.2E-4	8.4E-5
Pu-239	2.41 E+4	1.0 E-5	5.0 E-6	2.5 E-6
Pu-239 μ particle	2.41 E+4	3.0 E-4	1.5 E-4	7.5 E-5
Am-241	4.32 E+2	1.0 E-6	4.0 E-7	2.0 E-7

[a] *coefficients to foetus multiply by 10*

In the expression for total dose, $E_{external}$ is the external dose, calculated according to the rules in Chapter 6. Internal doses are obtained by summation of the isotopic contributions from inhalation and ingestion using dose coefficients $k(a)_{I,\ ingest}$ and $k(a)_{I,\ inhale}$ given in Table A1 for different age groups (a).

The Committee will publish a complete list of dose coefficients for all isotopes of radiological interest.

Appendix

ECRR - CERI

European Committee on Radiation Risk
Comité Européenne sur le Risque de l'Irradiation

The Lesvos Declaration

6th May 2009

A. Whereas, the International Commission on Radiological Protection (ICRP) has promulgated certain risk coefficients for ionizing radiation exposure,

B. Whereas, the ICRP radiation risk coefficients are used worldwide by federal and state governmental bodies to promulgate radiation protection laws and standards for exposure to workers and the general public from waste disposal, nuclear weapons, management of contaminated land and materials, naturally occurring and technologically enhanced radioactive materials (NORM and TENORM), nuclear power plant and all stages of the nuclear fuel cycle, compensation and rehabilitation schemes, etc,

C. Whereas, the Chernobyl accident has provided the most important and indispensable opportunity to discover the yields of serious ill health following exposure to fission products and has demonstrated the inadequacy of the current ICRP risk model, especially as applied to foetal and early childhood exposures to radiation,

D. Whereas, by common consent the ICRP risk model cannot validly be applied to post-accident exposures, nor to incorporated radioactive material resulting in internal exposure,

E. Whereas, the ICRP risk model was developed before the discovery of the DNA structure and the discovery that certain radionuclides have chemical affinities for DNA, so that the concept of absorbed dose as used by ICRP cannot account for the effects of exposure to these radionuclides,

F. Whereas, the ICRP has not taken into consideration new discoveries of non-targeted effects such as genomic instability and bystander or secondary effects with regard to understanding radiation risk and particularly the spectrum of consequent illnesses,

G. Whereas, the non-cancer effects of radiation exposure may make it impossible to accurately determine the levels of cancer consequent upon exposure, because of confounding causes of death,

H. Whereas, the ICRP considers the status of its reports to be purely advisory,

I. Whereas, there is an immediate, urgent and continuing requirement for appropriate regulation of existing situations involving radioactivity, to protect the human population and the biosphere,

We the undersigned, acting in our individual capacities

1. assert that the ICRP risk coefficients are out of date and that use of these coefficients leads to radiation risks being significantly underestimated,

2. assert that employing the ICRP risk model to predict the health effects of radiation leads to errors which are at minimum 10 fold while we are aware of studies relating to certain types of exposure that suggest that the error is even greater,

3. assert that the yield of non-cancer illnesses from radiation exposure, in particular damage to the cardio-vascular, immune, central nervous and reproductive systems, is significant but as yet unquantified,

4. urge the responsible authorities, as well as all of those responsible for causing radiation exposures, to rely no longer upon the existing ICRP model in determining radiation protection standards and managing risks,

5. urge the responsible authorities and all those responsible for causing exposures, to adopt a generally precautionary approach, and in the absence of another workable and sufficiently precautionary risk model, to apply without undue delay the provisional ECRR 2003 risk model, which more accurately bounds the risks reflected by current observations,

6. demand immediate research into the health effects of incorporated radionuclides, particularly by revisiting the many historical epidemiological studies of exposed populations, including re-examination of the data from Japanese A-bomb survivors, Chernobyl and other affected territories and independent monitoring of incorporated radioactive substances in exposed populations,

7. consider it to be a human right for individuals to know the level of radiation to which they are exposed, and also to be correctly informed as to the potential consequences of that exposure,

8. are concerned by the escalating use of radiation for medical investigation and other general applications,

9. urge significant publicly funded research into medical techniques which do not involve radiation exposures to patients.

Statements contained herein reflect the opinions of the undersigned and are not meant to reflect the positions of any institution to which we are affiliated.

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The first report of the European Committee on Radiation Risk was published in 2003. It presented a rational model for calculating the health risks of exposure to ionizing radiation. Unlike the then-current framework of modelling radiation-risk, based on the effects of the external acute exposures of the US Atomic Bombs on the Japanese, the ECRR model employed evidence from those exposed to internal radioactivity. The publication made a significant impact on the radiation risk assessment landscape, has been reprinted three times and translated into Japanese, French, Spanish and Russian.

Since then a large amount of new data has emerged in this important area both from epidemiology and from radiobiology. A very large number of peer-reviewed reports and published studies, from Chernobyl effects in Sweden to cancer and Uranium related congenital disease in Iraq, confirm the accuracy of the Committee's 2003 radiation risk model for explaining and predicting the health effects of human exposure to internal radionuclides. As well as confirming the broad accuracy of the ECRR approach, the new evidence has required some modification to the model, in particular the inclusion of discoveries in the area of Uranium and high-Z element secondary photoelectron risks. The developments since 2003 have been incorporated into an expanded and largely re-written presentation of the ECRR's radiation risk model.

There is increasing concern over the embarrassing dissonance between the conventional modelling of health outcomes of radioactive releases to the environment and the observations. In this volume, the Committee explains how the current physics-based risk model came to be universally used, and points out its scientific and political shortcomings. In addition, the Committee addresses the ethical basis of releasing radioactive materials to the environment.

The volume is essential reading for anyone involved in legislation in this area and should also be of interest to members of the public who need to estimate the effects of nuclear discharges.

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