Position paper on radiation risk prepared by LLRC for SAFEGROUNDS January 2010

Perspectives on the health risks from low levels of ionising radiation – Background information for DEBATE PAPERS

SAFEGROUNDS documents generally represent consensus guidance on best practice in on the management of radioactively and chemically contaminated land on nuclear and defence sites in the UK. However, SAFEGROUNDS is not a scientific committee; it is a forum where individual stakeholders express their views and agreement will not be reached on every topic. The aim is to build consensus around common needs and concerns but no one stakeholder's views take precedence over others' legitimate needs or concerns in the consensus building process, provided that the process has been properly conducted.

Where consensus cannot be achieved, the role of SAFEGROUNDS is therefore to raise awareness of the differences of view and encourage resolution through appropriate channels rather than make its own judgements. One of these topics is the health risks from low levels of ionising radiation and so SAFEGROUNDS invited four authors to contribute debate papers for publication on the website as part of the awareness raising process.

Unlike SAFEGROUNDS guidance documents, the purpose of these four debate papers is to explore differences in view rather than areas of agreement. They were not intended as consensus papers; they have not been endorsed by the Steering Group; and in each case individual Members may well disagree with some of their contents.

The first three debate papers were independently written by members of the SAFEGROUNDS Project Steering Group. Although there are naturally conflicts between papers, each can be taken as fully representing the views of the author's organisation.

- Shelly Mobbs of the Health Protection Agency.
- Richard Bramhall of the Low Level Radiation Campaign
- Paul Dorfman of Warwick University, on behalf of the Nuclear Consultation Group

This fourth paper was written by David Collier, an Independent Consultant. Its purpose is to offer a framework for understanding different perspectives on the potential impact on human health of levels of ionising radiation below current regulatory limits. It attempts to summarise the key points from the three position papers and the main differences in perspective, but is not a substitute for them. SAFEGROUNDS therefore encourages all those seeking an understanding to also read the source documents, which are concise and written to be accessible to a wide audience, and are supported by detailed references to the literature.

Although drafts of this paper were reviewed by the other debate paper authors to help ensure the positions being expressed had been properly understood, the subsequent analysis of the competing arguments is that of the author alone. It was commissioned by CIRIA (as managers of SAFEGROUNDS) but cannot be taken as representing the views of CIRIA or any SAFEGROUNDS member organisation.

It was also the author's decision to set the arguments out side by side without commenting explicitly or implicitly on their validity, on the basis that it is a guide to the arguments and not an assessment of them. This approach has value but means that consensus support for the publication of the paper from all sides of the argument could not be obtained. CIRIA recommends reading the comments from the other three debate paper authors in the Foreword of the Overview Framework paper before reading the individual perspectives.

Abstract
On the basis of radiobiological theory and epidemiological evidence, the position held by LLRC (and, increasingly by others[i]) is that ICRP's current dose/risk estimates are in error for some types of exposure. We believe that the exposures of interest are those characterised by high ionisation density in or close to sensitive tissues. We hold that the mechanisms of harm are poorly understood and that radioactive contamination causes many more conditions than are accommodated within ICRP advice. These health outcomes and new discoveries such as epigenetic effects have not been incorporated into ICRP's risk modelling, partly because of an inappropriate epidemiological basis,[ii] partly because the concept of absorbed dose has been extended into exposure regimes for which it is inappropriate, and partly because of mistaken assumptions about linear extrapolation from high dose to low.
The scale of the errors varies because of the large number of different radionuclides involved and the different physical and chemical forms in which they affect populations. Tissue location and varying radiosensitivity in subpopulations of cells and people add further uncertainty about the scale of variance with ICRP estimates. The range of error is between 100 for post-Chernobyl cancer increases in Sweden [iii] and 1000 for prostate cancer in internally contaminated nuclear industry workers.[iv] Up to 10,000-fold has been cited in respect of the KiKK study [v] and, in the mid-range, COMARE offers 200- or 300-fold in respect of the Seascale leukaemia cluster [vi] and up to 1000-fold for other studies.[vii]
In our opinion and in the opinion of the European Committee on Radiation Risk (ECRR) weighting factors published by ECRR [viii] provide a means of modifying current dose/risk estimates so that regulation of exposures can continue uninterrupted on a precautionary and more rational basis.

As a quantity for radiological protection purposes "Absorbed dose" has severe limitations

> "The growth of cancers is … the unchecked development of a single family of cells, derived originally from only one."[ix]
>
> "… one single track of ionising particles may be sufficient for the initiation process"[x]
>
> "… There are important concerns with respect to the heterogeneity of dose delivery within tissues and cells from short-range charged particle emissions, the extent to which current models adequately represent such interactions with biological targets, and the specification of target cells at risk. Indeed, the actual concepts of absorbed dose become questionable, and sometimes meaningless, when considering interactions at the cellular and molecular levels".[xi]

How we got here
Until the 1920s the main focus of radiation protection was external X-rays, but the radium dial painters' scandal made it obvious that internal effects needed specific investigation. The new trend led to standards determined by looking at the actual effects of internal contamination seen in the dissected tissues of people. In 1944 this was reversed, starting with Herbert Parker's arrival at the Manhattan Project and continuing through the 1952 decision of the NCRP to close down Karl Morgan's

internal radiation subcommittee, the decision to use the Japanese A-bomb survivors as an epidemiological baseline and the hijacking of ICRP by American influence. All this was done for reasons of administrative convenience and global politics so that all types of external radiation and internal radiation from incorporated radioactivity could be summed simplistically to give risk figures that, so far as internal radioactivity was concerned, had no basis in reality because in the process many extremely complex issues had been swept aside.[xii]

Criticisms of ICRP

ICRP and the other bodies from which ICRP draws information have been subject to a range of criticisms over many years. One criticism is the lack of independence, as there are significant overlaps of personnel.[xiii] Other explicit or implicit criticisms concern the scientific basis of ICRP's approach; examples are a recent paper on infant leukaemia (cxx below), statements by IRSN in 2005 (xxi below), statements by ECRR in 2009 (xxxviii below), a wide-ranging review from 1994, [xiv] various books, [xv] and a letter signed in 1999 by 133 organizations and individuals from 13 countries worldwide.[xvi] In February 1998 the European Parliament convened a Scientific and Technical Options workshop entitled *Criticisms of the ICRP Risk Model.* [xvii] Some of the most outspoken critics attended and spoke. The biased and deficient reporting of the proceedings has been described by Busby.[xviii]

RERF failings

It is widely accepted that the A-bomb survivors' data are an unsatisfactory basis for estimating the effects of internal contamination.[xix, xx, xxi, xxii, xxiii]. As early as 1953, data were available to falsify assumptions that there is a 7-year time lag between exposure and the onset of leukaemia, that there was no fallout or residual radiation at Hiroshima and Nagasaki and that there were no heritable defects in those who were exposed [xxiv] and hence to falsify a risk model based on those assumptions. (Interestingly, the "Atomic Bomb Injuries" data were cited by the BMJ in 1955.[xxv])

Since the Radiation Effects Research Foundation controls were as contaminated as the study group it has been possible to reanalyze RERF data to show whether there are health effects in the controls attributable to fallout.[xxvi, xxvii]. Busby has shown [xxviii] that UNSCEAR reported [xxix] high leukaemia rates in the Hiroshima controls relative to all Japan. Sternglass [xxx] attributed to fallout the dramatic increase in cancer rates in children which was recorded all over Japan between three and five years after the A-bombings. RERF data have been reanalyzed [xxxi] to show disturbances of sex ratio in live births.

Nonlinear dose response

The authors of a study [xxxii] of fetal damage state:

> "it is clear that the dose-effect relationship for the early fetus is unlikely to be linear, because beyond a certain level of radiation injury to any tissue which is critical to the survival of the fetus, there will be a reduction in the end point being considered, even though the exposure is increasing, due to death of the fetus and loss as a miscarriage. This is the biphasic dose response. Therefore, to argue that effects seen in countries where the dose is low cannot be caused by radiation because such effects are not seen in countries or areas

> where the doses are high is an invalid argument because in the high dose regions early fetal death may have removed potential cases."

It is commonly observed [xxxiii] that radiation-induced epigenetic effects saturate at low dose. We take this as suggestive of a non-linear dose response; on the same logic as in the above paragraph it is likely to be part of a biphasic or poly-modal response. Experimental results from Russia [xxxiv] indicate that the dose dependency of radiation effects may be non-linear, non-monotonic, and poly-modal, and that over certain dose ranges low level exposures are more effective with regard to their impact on an organism or on a population than acute high level exposures. Such observations are repeated in individual studies of infant leukaemia after Chernobyl (e.g. cxiii below) and in meta-analyses.(cxx below)

European Committee on Radiation Risk (ECRR)

The European Committee on Radiation Risk (ECRR) has developed weighting factors [ECRR 2003] to compensate for some of the shortcomings of the ICRP. In response the Institut de Radioprotection et de Sûreté Nucléaire has issued a report:[xxxv]

> "Various questions raised by the ECRR are quite pertinent and led IRSN to analyze this document with a pluralistic approach.
>
> a. Besides natural and medical exposures, populations are basically undergoing low dose and low dose rate prolonged internal exposures. But the possible health consequences under such exposure conditions are ill-known. Failing statistically significant observations, the health consequences of low dose exposures are extrapolated from data concerning exposures that involve higher dose rates and doses. Also, few epidemiologic data could be analyzed for assessing inner exposure effects. The risks were thus assessed from health consequences observed after external exposure, considering that effects were identical, whether the exposure source is located outside or inside the human body. However, the intensity, or even the type of effects might be different.
>
> b. The pertinence of dosimetric values used for quantifying doses may be questioned. Indeed, the factors applied for risk management values are basically relying on the results from the Hiroshima and Nagasaki survivors' monitoring. It is thus not ensured that the numerical values of these factors translate the actual risk, regardless of exposure conditions, and especially after low dose internal exposure.
>
> c. Furthermore, since the preparation of the ICRP 60 publication, improvements in radiobiology and radiopathology, or even in general biology, might finally impair the radiation cell and tissue response model applied to justify radioprotection recommendations. It was thus justified to contemplate the impact of such recent observations on the assessment of risk induced by an exposure to ionizing radiation."

IRSN's report concludes:

> "The phenomena concerning internal contamination by radionuclides are complex because they involve numerous physico-chemical, biochemical and physiological mechanisms, still ill-known and thus difficult to model. Due to this complexity, the behaviour of radionuclides in the organism is often ill described and it is difficult to accurately define a relationship between the dose delivered by radionuclides and the observed consequences on health. This led the

> radioprotection specialists to mostly use the dose/risk relationships derived from the study of the Hiroshima/Nagasaki survivors, exposed in conditions very different from those met in the cases of internal contaminations.
> This fact raises numerous questions, which should be considered with caution because a wide part of the public exposure in some areas of the world is due to chronic internal contaminations and very few data concern these situations.
> […] the questions raised by the ECRR are fully acceptable, … "

and

> "… we do not possess, in the current state of knowledge, the elements required to improve the existing radioprotection system."

The Committee has broadly welcomed the IRSN's critique: [xxxvi]

> "In summary, the IRSN report is a pretty complete validation of the things members of the Committee have been saying for many years about internal irradiation."

The two documents show good agreement between ECRR and IRSN on the nature and significance of the problems inherent in ICRP's approach. The adverse criticisms of ECRR that may be read into the IRSN report clearly arose because IRSN did not appreciate that the ECRR Recommendations (although they are subtitled "Regulators' Edition") are a pragmatic solution to allow exposures to be regulated in the vacuum left by ICRP's failure. ECRR notes:

> "Its only divergence is in its disagreement with the way the Committee has dealt with the issue, which IRSN sees as rather ad hoc and insecure. We reply that the semi-empirical epidemiology/ biochemistry approach was predicated on our need to provide some system of modelling in the absence of any other secure system …"

The ECRR agrees with IRSN that further research is needed but does not agree that ICRP's approach is adequate pending the results of that research. We hold that the ECRR position conforms with a properly precautionary approach.

Department of Health radiation research

The 2006 Department of Health radiation research programme [xxxvii] identified fundamental gaps in knowledge, including the role of micro-distribution and whether radiation damage might be non-linear at low dose and low dose rate.

ECRR 2009

In 2009 the ECRR underlined [xxxviii] its view that ICRP radiation risk coefficients are out of date and that using them leads to risks being significantly underestimated. The Committee repeated its call for regulators to adopt its own model.

Challenges to linearity and Absorbed Dose averaging

1. Heterogeneity With massive understatement, Annex B of Publication 103 ICRP acknowledges the complexities and challenges of internal contamination under conditions where energy deposition is extremely heterogeneous. It discusses radionuclides emitting alpha particles, soft beta particles, low-energy photons and Auger electrons. Paragraph B55 states:

> "… the heterogeneous distribution of energy deposition is of concern with respect to the averaging procedure in the low dose range and especially with radionuclides which are heterogeneously distributed in an organ or tissues and which emit particles with short ranges. However, no established approaches are presently available for practical protection practice which take into account microdosimetric considerations or the three-dimensional track structure in tissues and the related energy deposition. Considering the stochastic nature of the induction of cancer and of hereditary disease and the assumptions that one single track of ionising particles may be sufficient for the initiation process, it appears that the present approach is pragmatic for radiological protection with a justified scientific basis. The uncertainty associated with such an approach should be kept in mind."

We agree that the ICRP approach is "pragmatic"[xxxix] but ascribing "a justified scientific basis" to it is one of ICRP's value judgements. It is obvious that the exposures discussed above cannot validly be modelled using absorbed dose and anomalous health effects cannot be dismissed on the basis that they fail to conform with expectations based on that criterion.

2. Particles

Micron sized radioactive particles are widely dispersed in the environment. The conventional view is that the risk from particles is not significantly greater than is assumed by the ICRP averaging model. However, this may be a result of a trading balance between cell killing close to the particle and an enhanced mutagenic effect in cells further away which are subject to lower doses. At the top end of the range cell killing is likely to predominate, and at the bottom end the effects would be indistinguishable from the effects of external radiation. In other words, since a good proportion of the effect of the radiation from the particle is wasted in cell killing, the mutagenic efficiency of the unwasted portion may be considerably greater than assumed by the ICRP model. If this is the case then particles of lower activity, where cell killing does not predominate, may represent an enhanced health risk. This may be because the mid range will be in the quadratic region.

2a. Particles and the Bragg effect

At the 3-day international CERRIE Workshop in 2003 Professor Bryn Bridges pointed out that as a result of the Bragg effect dead cells would tend to be concentrated in a shell at a radial distance equal to the decay range of the alpha particle. This zone of dead cells would effectively insulate a community of potentially damaged cells preventing communication with healthy cells outside the range of the decays. These considerations may have significant implications for the development of clonal damage, and warrant further research.[xl]

3. The Secondary Photoelectron effect (SPE)

Releases of Uranium giving rise to its incorporation in body tissue appear to be genotoxic despite Uranium's low radioactivity. For example, a wide-ranging review of the teratogenicity of parental prenatal exposure to DU aerosols has concluded that "the evidence, albeit imperfect, indicates a high probability of substantial risk".[xli] This represents an extreme anomaly between actual risks and those expected on the basis of ICRP recommendations.

The hazard is likely to be mediated by a mechanism known as the Secondary Photoelectron effect (SPE) in combination with the affinity between Uranium and the DNA molecule. It does not depend on the intrinsic radioactivity of Uranium. Particulates as well as atomic Uranium are implicated.

The absorption of gamma rays by any element is proportional to at least the fourth power of the element's atomic number *Z*. ICRP, in considering gamma ray absorption, models the human body as water, H_2O. It has been proposed [xlii] that the baseline of absorption in uncontaminated tissue should be established using Oxygen - the most massive of the atoms in the water molecules in the ICRP phantom. The atomic number of Oxygen is 8. $8^4 = 4096$. The atomic number of Uranium is 92. $92^4 = 71639296$. $71639296/4096 = 17490$. This is the enhanced ability of an atom of Uranium to absorb incident gamma or X-rays, relative to an atom of oxygen.[xliii]

Energy absorbed in this way is re-emitted in the form of photoelectrons indistinguishable from beta radiation, potentially causing tissue damage.

The enhancement of external radiation by high atomic number materials was described as early as 1947 when Spiers calculated the enhancement of X-rays in bones, showing a ten-fold increase in radiation damage at the edge of bones due to photoelectrons induced in Calcium (Z=20). Others had tried to use Iodine (Z = 53) to enhance X-ray therapy for brain tumours. Experiments in the USA on the photoelectron enhancement of X-rays by gold nanoparticles (Z =79) have been shown to cure breast cancer in mice.

Uranium binds strongly to DNA. This is well known and has been described in the peer review literature since 1962. The affinity constant for UO_2^{++} and DNA is 10^{10} This means that at very low concentrations of Uranium, the DNA is fairly well saturated with it. The reason for the affinity is that the ion UO_2^{++}, the uranyl ion, follows Calcium in its chemical properties in the body. Calcium is the element which stabilises the DNA through neutralising the negative charges on the phosphate backbone.

The actual amount of DNA in a cell has been measured and is reported in the 1990 recommendations of the US National Academy of Sciences BEIRV committee. BEIRV state that the principal target for radiation effects is the DNA, something that all now agree on. The quantity of DNA in the cell, according to BEIRV, is about 7 picograms total (phosphate and sugar and bases; a picogram is 1×10^{-12} grams). But the cell (assuming an 8 micron diameter cell) has a mass of 270 picograms. So the DNA represents roughly 1/40th by mass on the basis of these BEIRV figures.

It is thus shown that at quite modest levels of Uranium in tissue, it is the Uranium that is the predominant absorbing material for natural background gamma radiation, and that the absorbed energy is converted into photoelectrons which attack the DNA both directly and indirectly though ionization of water. This argument is simple and immediate. The base line is that Uranium health effects are not mainly due to its intrinsic radioactivity, but to its high atomic number. Counter-intuitively, it is low energy incident radiation and the smallest particles that represent the greatest divergence from expectations based on LNT. [xliv]

The photoelectron idea was presented by Busby at the CERRIE international workshop at St Catherine's College in 2003 but was omitted from the CERRIE Majority Report. During the life of the first CoRWM, following discussions with LLRC, the Committee commissioned work on public exposure to Uranium. This can be found in its Report. The argument outlined above was formally presented to the MoD Depleted Uranium Oversight Board in 2004.

In June 2007 Busby submitted two papers on this topic [xlv] [xlvi] to the Royal Society journal *Proceedings of the Royal Society B*. They were both immediately 'unsubmitted' by the editors who, after Busby complained, advised him to submit them to the *Journal of the Royal Society Interface*. The *Interface* editor, Professor William Bonfield, sent the papers to three referees. All of them are known to Busby and all told him they had recommended publication but Prof. Bonfield rejected them "for lack of space". The USA-based Science Oversight Board, including 41 scientists and experts, complained in an open letter to the President of the Royal Society. There was no reply.

This work has been presented to an international conference held by the Federal German Agricultural Laboratories in Brunswick. The German Federal Radiation Protection Department agreed to study the new evidence. The FAL Director, Professor Dr. Ewald Schnug, contributed some evidence from optimum concentrations of elements in plants and a joint paper has been published.[xlvii]

In 2008 LLRC submitted information to the HPA's consultation on radiological criteria for the on-land disposal of solid waste. Among other issues LLRC drew attention to work on SPE. HPA summarily diverted LLRC's submission into a separate consultation on application of ICRP's 2007 Recommendations (ICRP Publication 103) in the United Kingdom. HPA went on to publish its advice on waste disposal without acknowledging the scientific issues presented by LLRC, stating [xlviii] that they would be addressed at the time of publishing advice on ICRP Publication 103. In the interim, an unpublished response from HPA discussed the Secondary Photoelectron effect.[xlix] It applied an inappropriate method which underestimated the potential impact of incorporated Uranium (see below for discussion). LLRC issued a short response pointing out the error.[l] HPA attempted a rebuttal [li] but refused to discuss the matter further, stating that a paper would be submitted to a peer reviewed journal and that LLRC would be able to respond to that. To our knowledge no such paper has been published. HPA went on to publish its advice on ICRP Publication 103. The *Summary of Comments Received* [lii] did not acknowledge the unresolved status of the dialogue relating to SPE.

The specific error in HPA's treatment of SPE is the use of Mass Energy Absorption Coefficients. These are an inappropriate quantity for investigating the evidence submitted because they give a ratio for the two media (Uranium and tissue) without accounting for their differing densities. The lower part of HPA's Figure 1[liii] uses the spurious ratio μ_{en}/ρ for the y axis.

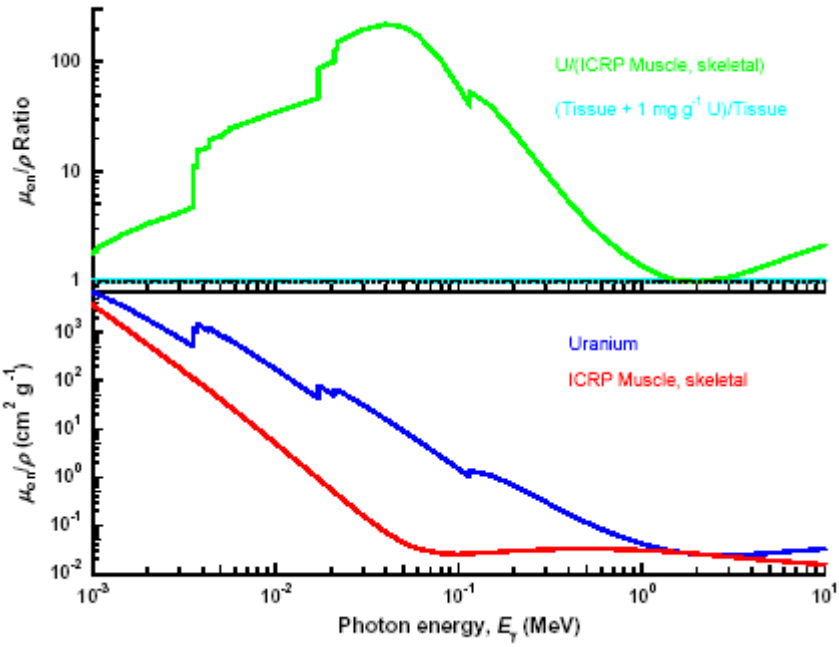

Figure 1. Comparison of the mass energy absorption coefficients for uranium and ICRP skeletal muscle. The lower part of the figure shows the direct comparison between uranium and skeletal muscle. The upper part of the figure gives the ratios of uranium to skeletal muscle and also with the addition of uranium to skeletal muscle: 1 mg g-1 of uranium has been added to exaggerate the difference, but the change in the absorption coefficient still peaks at only +1.1%.

The value for 10^{-1} MeV on the x axis, for example, hence produces values of 10^{0} for Uranium and 10^{-2} for tissue. The ratio of these is 1:100. Using the more relevant quantity Linear Energy Dissipation or Linear Attenuation Coefficients (μ_{en}) for the same 10^{-1} MeV gives 10^{0} x 19.6 (Uranium) / 10^{-2} x 1 (tissue) = 1960.
The difference in opinion can be put another way as, in fact, HPA has;

> "When considering absorbed dose, this density-normalised parameter is the quantity of interest … " [liv] (emphasis added)

and

> "… increasing the density of a medium increases the energy imparted to it but leaves the absorbed dose unchanged, since absorbed dose is defined as the energy imparted divided by the mass.".[lv]

HPA accept [lvi] that LLRC showed correct ratios for Uranium and tissue using Linear Attenuation Coefficients. They accept that "this ratio is … useful since it can be used to calculate the number of interactions." The number of interactions is, in fact, the quantity of interest. It cannot be stressed too strongly that considering absorbed dose in this context is inappropriate, because this is one of the situations where, as the CERRIE Majority reported,

> "the actual concepts of absorbed dose become questionable, and sometimes meaningless, when considering interactions at the cellular and molecular levels."[lvii]

As we observe above, with quotations, ICRP itself has acknowledged the complexities and challenges of internal contamination under conditions where energy deposition is extremely heterogeneous.
The HPA graphs only work if the thought experiment is dealing with a homogeneous Uranium soup while in reality it is highly probable that the Uranium, and hence the interactions, will be on the DNA molecule itself. The energy absorbed by the Uranium is dissipated within the DNA or in tissue close by. A high proportion of the energy dissipated will be in the form of low energy electrons; one authority observed

> "[with reference to] Auger electrons, the point, as I'm sure you are well aware, is that low energy electrons give high density of ionisations along their track, comparable with the diameter of DNA (hence a greater possibility of double strand breaks). Also, because the shower is released instantaneously, there are many more ionisations (i.e. OH radicals) simultaneously in the vicinity of the emitting atom, hence an enhanced probability of DS breaks. […] This area of dosimetry is completely ignored in the "standard" models, but could be very important."[lviii]

The upper part of HPA's Figure 1 is derived from the lower, showing the result of dividing the Mass Energy Absorption Coefficients of Uranium at different energies by those of tissue. The peak difference is about 200:1. If Linear Attenuation Coefficients are used instead, the peak difference is about 4000:1. (The roughly 20-fold difference between these figures is equal to the ratio of the densities of the media involved.) However, we are discussing the Secondary Photoelectron effect which does not depend on ratios of density but on the ratios of the fourth powers of the atomic numbers (*Z*) of the elements. We repeat that an atom of Uranium absorbs incident gamma or X rays 17,490 times as efficiently as an atom of Oxygen.

The foregoing calculations have been based on the conservative assumption that the absorption of gamma photons is proportional to the fourth power of the *Z* number. It has been pointed out [lix] that the fifth power may be applicable. Authorities differ and this question has yet be resolved. If the fifth power is the relevant value the effect will be larger than the 17,490-fold difference given above.

The potential numbers of interactions implied by these large differences, and the consequent ionisation density in sensitive tissues must be considered.

As for the location of the interactions, HPA's first paper [lx] says that "little is known of the *in vivo* distribution of Uranium in cells." Data are available for DNA *in vitro* and it cannot be assumed that DNA binding does not happen *in vivo*. We suggest that the precautionary principle applies — i.e. that until the degree of Uranium binding in vivo has been demonstrated one should assume that it is at least as great as *in vitro*.

A report on SPE and Uranium was published in *New Scientist* September 2008.[lxi] Hans Georg Menzel, chair of ICRP's dose assessments committee, was quoted as saying that committee members intended to conduct investigations. We have heard of none.

A paper by Pattison and others [lxii] models a 10 micron particle although 50% of the particles found after use of Uranium-based armour piercing weapons are <1 micron and very few are > 10 micron. Nevertheless Pattison finds the effect is significant, contrary to what HPA purported to have found in their dialogue with LLRC as reported above. It should be noted that Pattison *et al.* have not addressed atomic Uranium nor its DNA affinity. These considerations would apply strongly to many exposures involving Uranium (e.g. mining).

Criticisms of the Pattison *et al.* approach to modelling particles were sent to Prof. Bonfield for publication in *The Royal Society Interface*, but he rejected them. A Green Audit paper in press details the criticisms:

- the hypothetical particles used by Pattison *et al.* are far larger than those found in the battlefield which are spherical and smaller than 1 micron in diameter;
- the hypothetical particles are cylindrical, rather than spherical, and thus reduce the enhancement;
- the choice of the same 5 micron target volume for large and small particles results in incorrect and misleading conclusions about the effect of particle size;
- the input data removed a significant proportion of low energy photons from the natural background radiation spectrum, thus reducing the enhancement, since it is the low energy photons that contribute to the short range photoelectrons;
- the results were not supported by real measurements and biological effects reported by different groups.

The Green Audit paper employs results from Monte Carlo mathematical modelling by Elsaesser *et al. 2007* to develop semi-empirical calculations of dose enhancement near a 400nm uranium particle embedded in ICRU tissue. Results show increasing enhancement close to the particle surface with a maximum value of 50-fold within 100nm of the surface. However, the paper finds that there is a critical dependence of modelling results on the low energy photon spectra in tissue. It suggests that mathematical modelling of these small particle, low energy photon interactions is unsafe and that results should be obtained from experiment rather than modelling. Experiments by e.g. Regulla *et al.* 1998 and Hainfeld *et al.* 2004 suggest that enhancements of greater than 2000-fold would exist close to uranium particles.

In this section on SPE we have outlined large knowledge gaps, uncertainties, and challenges which the risk agencies have declined to address. They represent an urgent matter for further research. Two achievable and affordable research directions which could be illuminating are: 1) determination of the *in vivo* affinity between Uranium and DNA; 2) irradiation of laboratory animals dosed with soluble Uranium, checking for genetic damage against a control. At this stage, pending such research, the theory provides a sufficiently powerful mechanistic basis for taking a precautionary approach to discharges, particularly since there is also sufficient epidemiological and other data indicating that Uranium has anomalous radiological toxicity. Eminent among these data is a review of the teratogenicity of parental prenatal exposure to depleted uranium aerosols which concludes that "the evidence, albeit imperfect, indicates a high probability of substantial risk."[lxiii]
We have dealt with this issue at some length to demonstrate that it is a rapidly developing field of considerable importance for SAFEGROUNDS which will have to be revisited.

Evidence of somatic disease
There is a vast body of evidence from Chernobyl, representing possibly the greatest chance so far available to study the effects of wide-spread radioactive contamination.[lxiv,lxv,lxvi] Excess risks are associated with nuclear sites, [lxvii,lxviii,lxix,lxx,lxxi,lxxii,lxxiii,lxxiv,lxxv,lxxvi] and with contaminated coasts and estuaries, [lxxvii,lxxviii,lxxix,lxxx] phenomena which in LLRC's opinion are mediated by the accumulation and resuspension of fine-particle sediments contaminated with radioactivity, followed by inland migration and inhalation or ingestion.
Speculation on the cause of the disease being studied may be based on an invalid radiation risk model.[lxxxi] Studies said to falsify earlier positive results may be confounded by Chernobyl fallout.[lxxxii]
In sum, the evidence is that there are effects at low doses, as conventionally modelled following ICRP, which are greater than can be accommodated within that model. LLRC's position is that the flaws in the ICRP model do not allow it to be used as the basis of denying causation.
Various arguments are deployed to deny health effects which do not conform with expectations based on ICRP; we shall not consider them all here. The main technique is to rely on the ICRP paradigm itself and in particular to repeat the superficially plausible but misleading dogma of dose. Examples are COMARE 4th report on the 12-fold excess of childhood leukaemia at Seascale, the Swedish radiation protection institute's response to findings by Tondel of a 30% increase in cancer in parts of Sweden after Chernobyl,[lxxxiii] the Strahlenschuztkommission response to KiKK,[lxxxiv] and UKAEA's response to reports of prostate cancer.[lxxxv] ICRP routinely fails to cite such anomalous studies. The recently retired Scientific Secretary of ICRP has

admitted [lxxxvi] that this is a mistake. At the same time he acknowledged that ICRP's advice cannot be applied to post-accident exposures. Among the evidence ignored by ICRP in formulating its advice is the totality of the effects of the Chernobyl disaster.[lxxxvii]
Other arguments involve misuse of epidemiological method. An example concerning a reported excess risk of childhood leukaemia close to a Scottish coast contaminated by discharges from Sellafield is analysed in the literature, where it is shown that cancer registry officials had ignored the major confounder of Chernobyl fallout.[lxxxviii] When the data are reworked to exclude the period affected by Chernobyl the excess risk associated with residence near the sea is confirmed. (The same confounder operating in a different context was referred to at lxxxii above). Cancer registry officials in Wales have repeatedly made elementary errors about population data for areas contaminated by Sellafield discharges and other sources. This operated to diminish excess risks of cancer and leukaemia found by others. COMARE had failed to notice the errors and issued a retraction after they were pointed out.[lxxxix],[xc]

Chernobyl Forum Report (CFR)
In general the Chernobyl disaster caused doses (as conventionally modelled) around the same level as natural background. The Chernobyl Forum Report [xci] is frequently cited as evidence that the Chernobyl disaster has had no observable effect on health. This desk review in fact contains admissions that many diseases have increased; the caveat is that there was no consistent trend with dose. In this respect there is agreement between the Chernobyl Forum Report and the findings of the other overviews already cited; where they differ is that WHO and IAEA, the lead agencies in the Chernobyl Forum, adhere dogmatically to the conventional model of radiation risk and thus have to deny that radiation caused the disease.

Leukaemia: the KiKK and other studies
The German KiKK studies have reported [xcii],[xciii] significant increased risks of leukaemia and solid cancers among children under five years old in the vicinity of all German nuclear power stations. An independent team appointed by the German Government's Federal Office for Radiation Protection (BfS) reported [xciv] that the design and methodology of the KiKK study were sound. It disagreed with the authors' view that a radiobiological cause for the increased cancers could be ruled out. The BfS report stated that the dose and risk models assumed by the KiKK authors did not necessarily reflect the actual exposures and possible radiation risks and that it was necessary to investigate the radiobiological plausibility of the findings under different exposure scenarios. More work was needed on the exact radiation doses to nearby people. Also more research was required on the biological effects of ionizing radiation in the light of the paradigm shift caused by new findings from radiation epidemiology, genetic medicine and molecular biology. It further suggested that a combination of genetic polymorphisms for reduced DNA repair and/or genetic radiosensitivity might provide a possible biological explanation for the KiKK findings.
Any assertion that radiation doses were too low to have caused the excess leukaemia must be rejected on grounds of the insecurities in the risk model.
KiKK's use of proximity as a surrogate for exposure indicates a need for the same or a similar methodology to be applied in new studies of situations where it is possible to ascertain levels of exposure to radioactive discharges.

On the basis of the existing risk model Darby and Read have argued that there can be no causative association between the KiKK results and NPPs.[xcv] These authors also state that increased childhood leukaemia has been found in areas of Germany and the UK where NPPs were planned but not built. They suggest that "nuclear power plants tend to be built in areas where the risk of childhood leukaemia is already increased for some other, as yet unknown, reason." The argument is repeated by SSK.[xcvi] Neither report gives references for these studies. In their absence we assume the authors have in mind a study of sites considered but not used in the UK.[xcvii] We caution that the sites were in areas of high rainfall and that the study overlooks the higher weapons test fallout in such regions which correlates with childhood leukaemia.[xcviii] Similarly we assume that, for Germany, the authors have in mind a BfS study of childhood cancer and congenital malformation around NPPs in Bavaria which includes potential NPP sites.[xcix] The data have been reanalysed,[c],[ci] showing that the BfS paper reduced risks around operational sites by including very small reactors. In similar fashion, it inflated risks around planned but unused sites by including Rehling, the only place where risk was significantly higher than expected. Rehling is 30km downwind of Gundremmingen, the operational site with the highest risk. Without Rehling the results were not significant. Interestingly, risks at Rehling and Gundremmingen were almost identical, calling into question the BfS decision to limit its study to disease incidence within 15km of the NPPs. If Darby and Read intended a German study [cii] which similarly included planned but unused sites we would point out that the data do not support any claim that either leukaemia or all malignancies were elevated in the vicinity of the unused sites.[ciii]

Any assertion that Bithell *et al.*. [civ] and Laurier *et al.*.[cv] have not replicated the KiKK results must be questioned. Both found increased risks which did not reach statistical significance; this does not mean that they can be ignored. Scientific method and in particular Bradford Hill's cannon of consistency require that they be added to the sum of other studies. The same can be said of a recent meta-analysis.[cvi]

The COMARE 10th Report has not falsified the studies summarised above. The method employed by COMARE [cvii] employs population data aggregated to the level of local authority wards and assesses the proximity of those populations to nuclear installations according to population centroids. In the rural areas where nuclear installations tend to be sited, wards are generally very extensive and towns that may be in the vicinity are not close. Consequently, any health effects apparent in the population closest to the nuclear installations are diluted into the larger population that resides at a greater distance. This invalidates the conclusions of a study intended to inform on the risks of living close to nuclear installations. The KiKK methodology does not suffer from this weakness, yet it still uses proximity to the installation as a surrogate for exposure. It is instructive to consider that in 2001 the UK Small Area Health Statistics Unit (SAHSU), set up following the recommendations of the 1984 Black Report, acknowledged the limitations of such a study design, recognising that "it does not take into account the influence of weather conditions, water movements, occupations, lifestyles etc. which will all influence actual exposure." SAHSU also acknowledged that over any extensive study period potential associations between disease and exposure will be attenuated because exposed individuals migrate out of the area while those who move in are unexposed.[cviii] It should also be noted that less than 5% of the externalities of nuclear power are associated with the nuclear plant itself.[cix] The fact that significant excess risks near reactors have been reported makes it all the more urgent to investigate the upstream and downstream components where

the remaining >95% of the external costs reside. This will inevitably include SAFEGROUNDS' area of interest.

Infant leukaemia after Chernobyl

A significant increase in infant leukaemia between 20% and 330% after Chernobyl was proposed to CERRIE as unequivocal evidence of a large error in ICRP risk factors.[cx, cxi, cxii, cxiii, cxivcxv, cxvi, cxvii] This, in fact, was the primary reason for the Committee's establishment. The phenomenon is a crucial challenge to conventional radiation risk estimates because there is no known confounder for the hypothesis that the Chernobyl fallout caused the disease in this very precisely defined subset of the population. Leukaemia is recognised as an early indicator of radiation damage; more specifically, infant leukaemia (i.e. diagnosed before a baby's first birthday) signals damage acquired in the womb.

It has been pointed out (cxx below) that the post-Chernobyl infant leukaemia observations represent epidemiological confirmation of biphasic dose:response. Such behaviour is not remarkable for an *in utero* cause and endpoints in the living child, since above a certain dose some defence system may become overwhelmed. Increasing the dose of any foetal poison will generally result in foetal damage and ultimately in death of the foetus. In real-world situations concerning an endpoint that is registered after birth the highest doses will not necessarily produce the greatest effect.

The treatment of this topic by the CERRIE Majority report was farcical.[cxviii] COMARE was fully aware of its importance in CERRIE's discussions but does not mention it.[cxix] Chairman Prof Bryn Bridges defended the 9th report by citing the forthcoming European Childhood Leukaemia/ Lymphoma Incidence Study (ECLIS) which, the report says,

> *"will investigate trends in incidence rates of childhood leukaemia and lymphoma in 20 European countries, in relation to [...] Chernobyl [...] Such large studies are much more likely to produce firm results than those proposed in the CERRIE report."*

ECLIS is still unpublished, however. The proposition that the infant leukaemia falsifies ICRP has never been refuted, and a new paper [cxx] puts it explicitly into the literature.

Infant Mortality

No-one has refuted the proposal [cxxi] that the deceleration in the general, long-term reduction in infant mortality rates which was observed world-wide at the time of atmospheric weapons testing was due to fallout.

Epigenetic effects

Epigenetic effects ("non-targeted effects" - bystander signalling and genomic instability) define a process in which the effects of a single hit of radiation on a single cell are communicated to hundreds of cells which are then more prone to mutation. *A priori* this defines a mechanism for amplifying the impact of radiation and producing greater damage per unit dose.[cxxii, cxxiii, cxxiv, cxxv]. It will be of greater significance for internal contamination than for external irradiation on account of the potential for some radionuclides to become relatively immobilised, leading to chronic irradiation of local tissues.

ICRP advice does not include any analysis of how disease end-points are or may be associated with epigenetic effects. ICRP's position is that available data do not

provide good evidence of a robust causal association with cancer risk. This is confounded by non-cancer illnesses that kill the victims before they can be diagnosed with cancer (otherwise known as "confounding by deaths from competing causes"). It is sometimes stated that newly discovered phenomena (e.g. epigenetic effects) will already be included in cancer risk estimates since these are based on human epidemiological data and therefore encompass all relevant biological processes. This is falsified by the fact that ICRP do not address the full range of human epidemiological data available.

Non-linear dose/response

UNSCEAR states [cxxvi] the doubling dose for congenital abnormalities is 21.3 Gy. However, Scherb [cxxvii] [cxxviii] and other workers [cxxix] using data from the Bavarian congenital malformation dataset have shown the doubling dose is in the order of a few mSv for congenital malformations such as malformations of the heart, deformities and Down's Syndrome. This implies that UNSCEAR is in error at least at 3 orders of magnitude. Scherb [cxxx] shows alteration in sex ratio of live births generally greater in more contaminated countries and calculates the numbers of missing baby girls. Other authorities hold that epidemiological data that demonstrate ill-health effects can not be discounted on the basis of assumptions about absorbed dose and linear dose response. The ECRR states [2003 Recommendations p. 54] that "The health consequences of exposure to ionising radiation follow damage to somatic cells and germ cells and thus involve almost all illnesses." In a large literature review [cxxxi] of congenital malformation, fetal loss, stillbirth, infant death, infant leukaemia, genetic mutation, Down's Syndrome, and neural tube defects in many countries Busby ***et al.*** show that the ICRP assumption of a threshold for ***in utero*** effects is unsafe and that the A-bomb survivors' data are incomplete. The authors show that the findings summarised were not an artefact of increased surveillance after Chernobyl. They cite [cxxxii] several laboratory studies which falsify the ICRP assumption of a 100mSv threshold for effects after in utero exposure. Excess Down's Syndrome has also been found [cxxxiii] associated with high levels of natural background radiation.

Richard Bramhall
January 26th 2010

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[i] see, e.g. "An unbiased study of the consequences of Chernobyl is needed" Guardian Letters 18 Jan. 2010; http://www.guardian.co.uk/environment/2010/jan/18/nuclear-radiation-risk-chernobyl-cancer, and "The risks of nuclear energy are not exaggerated" Guardian "Response" 20 Jan. 2010; http://www.guardian.co.uk/commentisfree/2010/jan/20/evidence-nuclear-risks-not-overrated

[ii] Studies of Japanese A-bomb survivors at Hiroshima and Nagasaki are exclusively of acute high dose external gamma, X and neutrons. The controls inhabited the cities and were exposed to internal radioactivity to the same extent as the study groups. Study group exposures were characterised by well-averaged energy deposition throughout all tissues. Using such data to predict the effects of chronic internal contamination with beta and alpha emitters is problematic yet they provide the largest body of data informing ICRP's radiation risk coefficients. Other, smaller studies informing ICRP's coefficients suffer various weaknesses which have been analysed by Busby in "Wings of Death" ref. ix.

[iii] TONDEL M, HJALMARSSON P, HARDELL L, CARLSSON G, AXELSON O "Increase of regional total cancer incidence in north Sweden due to the Chernobyl accident?" *Journal of Epidemiology and Community Health* 2004;58:1011-1016 (abstract at http://jech.bmjjournals.com/cgi/content/abstract/58/12/1011. Radioactive Times May 2006 calculates a 125-fold error based on the assumption that the effect is transient and that there will be no excess after

1996. If excess cancer rates continue throughout life, the implied error in ICRP's modelling will be 600-fold or more. http//:www.llrc.org/rat/subrat/rat61.pdf page 17

TONDEL M "Malignancies in Sweden after the Chernobyl Accident in 1986" Linköping University Medical Dissertations No, 1001 Linköping University Faculty of Health Sciences Division of Occupational; and Environmental Medicine Dept of Molecular and Clinical Medicine SE-581 85 Sweden 2007. ISBN 978-91-85715-17-6

[iv] BMJ. 1994 January 22; 308(6923): 268–269. PMCID: PMC2539344 Prostatic cancer and radionuclides. Cancer risk has no effect on mortality. W. D. Atkinson, M. Marshall, and B. O. Wade http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2539344/pdf/bmj00424-0058c.pdf

[v] Assessment of the Epidemiological Study on Childhood Cancer in the Vicinity of Nuclear Power Plants: Statement of the Commission on Radiological Protection (SSK), September 2008

[vi] COMARE 4th Report

[vii] COMARE 10th Report

[viii] *ECRR 2003 Recommendations of the ECRR The Health Effects of Ionising Radiation Exposure at Low Doses and Low Dose Rates for Radiation Protection Purposes: Regulators' Edition* Edited by Chris Busby with Rosalie Bertell, Inge Schmitz-Feuerhake, Molly Scott Cato and Alexei Yablokov Published on Behalf of the European Committee on Radiation Risk by Green Audit, 2003. ISBN: 1 897761 24 4 http://www.euradcom.org/2003/ecrr2003.htm

[ix] "Wings of Death: Nuclear Pollution and Human Health": Chris Busby. Green Audit, Aberystwyth 1995 ISBN: 1-897761-03-1

[x] Annex B of ICRP Publication 103 para. B55

[xi] CERRIE Majority Report Chapter 2 Part 2 paragraph 11.

[xii] See switcheroo.doc attached - an account of how empiricism was sacrificed to theory. This is at www.llrc.org/switcheroo.htm to be a reference out.

[xiii] The illness of Stuart Raymond Dyson, Deceased and his previous exposure to Uranium weapons in Gulf War I. Supplementary report on probability of causation for HM Coroner Black Country Coroners District Smethwick, W. Midlands 2009 Paragraph 4 at http://www.llrc.org/du/subtopic/dysonrept.pdf.

[xiv] Review: "Inconsistencies and Open Questions Regarding Low-Dose Health Effects of Ionizing Radiation" Rudi H. Nussbaum and Wolfgang Köhnlein. Environmental Health Perspectives Volume 102, Number 8, August 1994 (http://www.ehponline.org/members/1994/102-8/nussbaum-full.html)

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Tamplin, AR. Gofman, JW. The Radiation Effects Controversy, Bulletin of Atomic Scientists, 26/2: 5-8 (1970)

[xvi] Letter to the BEIR VII Committee in 1999, initiated by the Institute for Energy and Environmental Research. See http://www.ieer.org/comments/beir/ltr0999.html

[xvii] Workshop Report "Survey and Evaluation of Criticism of Basic Safety Standards for the Protection of Workers and the Public Against Ionising Radiation" European Parliament, STOA Unit, Brussels. 1998. (This report apparently cannot be found in Brussels and no copy of the final version is known to us. The abstracts have been recovered; see http://www.llrc.org/health/subtopic/stoaabstracts.pdf

[xviii] Wolves of Water, Chris Busby. Published by Green Audit, Aberystwyth, UK ISBN 1 897761 26 0 pp. 344-6 (http://www.llrc.org/wings/subtopic/stoapp344346.pdf)

[xix] Schmitz-Feuerhake I. Dose revision for A-bomb survivors and the question of fallout contribution. Health Phys (Letter) 1983;44:693–5.

[xx] Stewart AM, Kneale GW. A-bomb survivors: factors that may lead to a re-assessment of the radiation hazard. Int J. Epidemiol. 2000;29: 708-714,

[xxi] IRSN "Health consequences of chronic internal contaminations by radionuclides: Comments on the ECRR report "The health effects of ionizing radiation exposure at low doses for radiation protection purposes" and IRSN recommendations. Direction de la Radioprotection de l'Homme." Report DRPH/No. 2005-20. http://www.euradcom.org/2005/irsn%20rapport%20ecrr-en.pdf "*Conclusions and IRSN recommendations: The phenomena concerning internal contamination by radionuclides are complex because they involve numerous physico-chemical, biochemical and physiological mechanisms, still ill-known and thus difficult to model. Due to this complexity, the behaviour of radionuclides in the organism is often ill described and it is difficult to accurately define a relationship between the dose delivered by radionuclides and the observed consequences on health. This led the radioprotection specialists to mostly use the dose/risk relationships derived from the study of the Hiroshima/Nagasaki survivors, exposed in conditions very different from those met in the cases of internal contaminations. This fact raises numerous questions, which should be considered with caution because a wide part of the public exposure in some areas of the world is due to chronic internal contaminations and very few data concern these situations."*

[xxii] Gofman J.W. *Radiation-Induced Cancer from Low-Dose Exposure: An Independent Analysis.* San Francisco: Committee for Nuclear Responsibility; 1990. www.ratical.org/radiation/CNR/RIC. DOI 10.1007/s12199-008-0039-8

[xxiii] Ref. i above.

[xxiv] "Atomic Bomb Injuries"; Eds. Dr Nobio Kusano *et al.*. of the Japanese Preparatory Committee for Le Congrès Mondial des Médicins (*sic)* pour l'Étude des conditions Actuelles de Vie Eds. Tsukiji Shokan, Tokyo, 1953. Relevant extracts at http://www.llrc.org/1953datafromhiroshima.htm

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[xxvii] Shoji Sawada, Estimation of residual radiation effects on survivors of Hiroshima atomic bombing, from incidence of acute radiation disease. Submitted to Radiation Protection Dosimetry, and in press in Proceedings of ECRR Conference, Lesvos, May 2009.

[xxviii] *Wings of Death: op. cit.* Ch. 5 *Paradigm Deconstructed*

[xxix] UNSCEAR 1964 Report to the General Assembly; suppl. 14, A/5814 (New York, United Nations. p 100

[xxx] Sternglass EJ, *Secret Fallout;* New York, McGraw Hill 1981.

[xxxi] Padmanabhan VT. Deviation in Sex ratio among the offspring of Atomic bomb survivors of Hiroshima/Nagasaki, presentation to CERRIE International Workshop July 2003.

[xxxii] Busby C, Lengfelder E, Pflugbeil S, Schmitz-Feuerhake I: "The evidence of radiation effects in embryos and fetuses exposed to Chernobyl fallout and the question of dose response": Medicine, Conflict and Survival Vol. 25, No. 1 January-March 2009, 20-40 (passage quoted is from page 30)

[xxxiii] e.g. HPA-RPD-055 page 9 and references

[xxxiv] Burlakova, E.B; Goloshchapov, A.N; Zhizhina, G.P.; Konradov, A.A. New aspects of regularities in the action of low doses of low level irradiation. In *Low Doses of Radiation—Are They Dangerous?* Burlakova, E.B., Ed.; Nova Science Publishers: New York, NY, USA, 2000.

[xxxv] Health consequences of chronic internal contaminations by radionuclides: Comments on the ECRR report *"The health effects of ionizing radiation exposure at low doses for radiation protection purposes"* and IRSN recommendations. Direction de la Radioprotection de l'Homme Report DRPH/No. 2005-20

[xxxvi] http://www.euradcom.org/2005/irsn.htm

[xxxvii] Department of Health's Radiation Protection Research Strategy, July 2006: "… the main policy questions that need to be addressed":

- How can the characteristics of ionising radiation affect health risk? To what extent are radiation quality, dose rate, micro-distribution and energy important factors in determining the risk following exposure to a particular type of radiation such as alpha particles?
- What tissues are relevant to radiation-induced health detriment? Will these vary during the different stages of biological development and does the variation depend on radiation type?
- Can radiation-induced health effects be clearly distinguished?
- To what extent are health effects from internal radiation quantitatively or qualitatively different to those from external radiation?
- Can it be determined whether radiation damage is non-linear at low doses and low dose rates?

[xxxviii] http://www.euradcom.org/2009/lesvosdeclaration.htm

[xxxix] *pragmatic* adj. 1 "relating to the affairs of a state or community": Oxford English Dictionary. In other words, the ICRP approach is policy-based science.

[xl] CERRIE Minority report para 65. p 56

[xli] Hindin R, Brugge D, Panikkar B. "Teratogenicity of depleted uranium aerosols: a review from an epidemiological perspective." Environmental Health: A Global Access Science Source 2005 Aug 26;4:17 doi:10.1186/1476-069X-4-17 http://www.ehjournal.net/content/4/1/17

[xlii] *pers. comm.* the late Dr. Philip Day, 12th January 2009.

[xliii] For the avoidance of doubt, this use of Oxygen as the baseline for gamma absorption in uncontaminated tissue replaces LLRC's earlier formulations which used a *Z* value of 3.33, being a notional mean atomic number of the three atoms in a water molecule.

[xliv] C. V. Howard, A. Elsaesser & C. Busby (2009) The biological implications of radiation induced photoelectron production, as a function of particle size and composition. International Conference; Royal Society for Chemistry NanoParticles 2009 (http://www.soci.org/News/~/media/Files/Conference%20Downloads/Nanoparticles%20Sep%2009/Oral_18_32.ashx)

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[xlvii] Busby CC AND SCHNUG E (2007) 'Advanced Biochemical and Biophysical Aspects of Uranium Contamination' in *Loads and fate of Fertiliser Derived Uranium* ed. LJ de Kok and E. Schnug, Backhuys Publishers, Leiden

[xlviii] HPA-RPD-052: Response to Comments Received during the Consultation on Proposed HPA Advice on Radiological Protection Objectives for the Land-based Disposal of Solid Radioactive Waste. K A Jones, S F Mobbs and T Anderson. HPA April 2009 ISBN 978-0-85951-639-6

[xlix] *Scientific / technical response to questions from Richard Bramhall relating to the assessment of doses and risks from internal emitters and ICRP methodology*, pers. comm. HPA 9th December 2008.

[l] http://www.llrc.org/wobblyscience/subtopic/uraniumhowler.pdf

[li] *Response to e-mail form [sic] Richard Bramhall, dated 5th January 2009.* pers. comm. HPA 26th January 2009.

[lii] HPA-RPD-057: Response to Comments Received during the Consultation on Proposed HPA Advice on the Application of ICRP's 2007 Recommendations to the UK. J R Cooper, J Wellings and J R Simmonds HPA September 2009 ISBN 978-0-85951-649-5

[liii] in *Scientific / technical response to questions from Richard Bramhall … op.cit.* ref xlix.

[liv] Para 4.2.2 of ref. xlix.

[lv] We have paraphrased from HPA's para 4.2.2. *ibid.*

[lvi] in 4.2.2 *ibid.*

[lvii] from CERRIE (Government's Committee Examining Radiation Risks of Internal Emitters) Majority Report Chapter 2 *Risks from Internal Emitters Part 2* paragraph 11. Along with a number of extracts from other authorities, this paragraph from CERRIE was quoted in LLRC's paper to HPA's waste criteria dialogue. "*There are important concerns with respect to the heterogeneity of dose delivery within tissues and cells from short-range charged particle emissions, the extent to which current models adequately represent such interactions with biological targets, and the specification of target cells at risk. Indeed, the actual concepts of absorbed dose become questionable, and sometimes meaningless, when considering interactions at the cellular and molecular levels.*"

[lviii] the late Dr. Philip Day pers. comm. 12th Jan 2009.

[lix] Philip Day *op.cit.* and others.

[lx] HPA Ref xlix *op.cit.*

[lxi] *How war debris could cause cancer* by Oliver Tickell; *New Scientist* 07 September 2008.

[lxii] *Enhancement of natural background gamma-radiation dose around uranium micro-particles in the human body* John E. Pattison, Richard P. Hugtenburg and Stuart Green September 23, 2009, doi: 10.1098/rsif.2009.0300.
http://rsif.royalsocietypublishing.org/content/early/2009/09/23/rsif.2009.0300.abstract

[lxiii] Teratogenicity of depleted uranium aerosols a review from an epidemiological perspective.Environ Health. 2005 Aug 26;4:17.Hindin R, Brugge D, Panikkar B. "Teratogenicity of depleted uranium aerosols: a review from an epidemiological perspective." Environmental Health: A Global Access Science Source 2005 Aug 26;4:17 doi:10.1186/1476-069X-4-17
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[lxv] *Chernobyl: Consequences of the Catastrophe for People and Nature ed.* Alexey V. Yablokov, the late Vasilly B. Nesterenko, Alexy V. Nesterenko, Janette D. Sherman-Nevinger; *Annals of the New York Academy of Sciences;* http://www.nyas.org/Publications/Annals/Detail.aspx?cid=f3f3bd16-51ba-4d7b-a086-753f44b3bfc1

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