ECRR European Committee on Radiation Risk

Baltic Sea Regional Office

Preliminary formal Response to the SKB Environmental Impact Statement of December 2009 relating to the proposed radioactive waste repository at Forsmark, Sweden.

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Part I Summary

The ECRR set up a Baltic Sea Regional Office in 2009 since the committee was concerned about the proposed development of a nuclear waste repository at Forsmark in Sweden. If such a project were permitted, the consequences would inevitably be that additional radioactive pollution of the Baltic sea would occur. Measurements of radioactivity available from HELCOM and other sources already show the sea to be the most radioactively polluted salt water sea in the world; the effects of this contamination on people living on the shores of the Baltic are the subject of current ECRR research being directed from the ECRR office in Riga Latvia. The SKB Environmental Impact Statement (in Swedish) and a number of related documents were obtained from SKB and from the website of SKB. ECRR had already been consulted by the UK Committee on Radioactive Waste management (CoRWM) for an assessment of the various proposals advanced by that organisation for dealing with the UK's waste and therefore there was some expectation that the SKB proposals would be amenable to a similar analysis. The ECRR's concern was to examine the environmental transfer and also the radiation risk models. ECRR's position in this case of Forsmark, as it was in the CoRWM case, was to employ the ECRR model to estimate risk to health following exposures to releases for the waste operation, both the transfer of the radioactive material and its inevitable eventual leakage intro the environment. There are therefore two stages to be carried out in any modelling of radiation risk from nuclear waste disposal. The first is to model the movement of the radioactivity from its origin and establish concentrations in environmental material with time. The second is to model the exposures to humans and biota and calculate the risk of illness e.g. cancer, genetic damage, species loss etc.

Currently, the second of these models, the one which is employed by governments to set limits to exposure is that of the International Commission on Radiological Protection ICRP. It has been shown to be seriously in error for modelling the effects of internal fission product radionuclides and Uranium. For Uranium, the error resulting from employing the ICRP exposure risk model is upwards of 500-fold. This matter is discussed at length in the main text. However, as a result of this, any assessment of risk carried out in relation to the EIS would be wrong by a very large amount. Astonishingly, the EIS barely mentions radiation risk. There is one section (3.4, page 37) where the document refers to the ICRP model: however no modelling of dose or exposure is to be found anywhere in any of the documents examined. Even where the radiation exposures are discussed, the EIS makes very erroneous statements and gives misleading information. For example, on p 37 we are told that after 100,000 years all that will remain is natural uranium minerals. This is not true: there will be massively enhanced levels of both U-238 and also the more radioactive U-235 and U-234. The bar graph on p 38 appears to show that the radioactivity will decay to 0.0005% of its initial value after 100,000 years; however, most of the material is uranium. Since this has a half life of billions of years, there will be virtually no change in its quantity over the 100,000 years of the graph on p 38.

The EIS is disappointingly empty of any real information which can be used to assess the real fears of people concerned about the development. It is, however, redolent with images which are clearly placed there to mislead: photographs of ducks, eagles, frogs, beautiful rivers sparkling in the sun with a small girl standing on a bridge. This is serious discourse manipulation: it is saying - this is what we are about. In reality, of course, the project is about bringing the refined uranium contents of many Uranium mines in the world and placing the huge quantity of uranium, together with its dangerous fission and activation products under the Baltic Sea in copper cans. The material will inevitably leak into the sea, over the significant timescales involved, and will make the sea contaminated, radioactive and poisoned forever. And if there is an accident, or there is some error in the dispersion modelling (which has not even been done, or is not reported) then the people living on the shores on the Baltic near the repository will be exposed to this material and will suffer genetic damage and cancer.

It is proposed that SKB presents credible dispersion and risk models that can be examined independently and that the risk modelling carried out employs the system of the ECRR published in 2003 and currently being updated to be published in 2010.

Chris Busby

Feb 5th 2010

Part II The ICRP and current radiation risk assessments

A Low Level Radiation Campaign briefing (www.llrc.org)

Abstract

On the basis of radiobiological theory and epidemiological evidence, it is believed that ICRP's current dose/risk estimates are significantly in error for some types of exposure. 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,[i] 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 [ii] and 1000 for prostate cancer in internally contaminated nuclear industry workers.[iii] Up to 10,000-fold has been cited in respect of the KiKK study [iv] and, in the mid-range, COMARE offers 200- or 300-fold in respect of the Seascale leukaemia cluster [v] and up to 1000-fold for other studies.[vi] In our opinion and in the opinion of the European Committee on Radiation Risk (ECRR) weighting factors published by ECRR [vii] 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."[viii]
>
> "… one single track of ionising particles may be sufficient for the initiation process"[ix]
>
> "… 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".[x]

The origin of the problem

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. [xi] Continuing the new trend, in 1951 the American National Committee on Radiation Protection, dominated by the Atomic Energy Commission, closed down the work of Karl Morgan's internal radiation subcommittee. American influence dominated the decision to use the Japanese A-bomb survivors as an epidemiological baseline for determining risk, although the "exposed" cohorts and the controls were more or less equally likely to be internally contaminated. In 1950 American influence resuscitated the International X-ray and Radium Protection Committee, which had been since before the war. It was renamed as the International Commission on Radiological Protection but operated as "little more than the overseas branch of the NCRP"[xii] whose first act was to adopt the NCRP's own standards in their entirety.

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.

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 (cvii 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. Official reporting of the proceedings has been criticised.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. Padmanabhan's analysis of RERF data reveals disturbances of sex ratio in live births.[xxxi]

Nonlinear dose response

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

> "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. cii below) and in meta-analyses.(cvii below)

European Committee on Radiation Risk (ECRR)

The European Committee on Radiation Risk (ECRR) has developed weighting factors (vii above) 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 There is reason to believe that the exposures of interest are those characterised by high ionisation density in or close to sensitive tissues. 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, stating: xxxix

> "… 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"xl 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.

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.[xli]

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".[xlii] This represents an extreme anomaly between actual risks and those expected on the basis of ICRP recommendations.

It appears improbable that the reported effects depend on the intrinsic radioactivity of Uranium. The hazard is more likely to be mediated by a mechanism known as the Secondary Photoelectron effect (SPE) in combination with the affinity between atomic Uranium and the DNA molecule. Particulates are also likely agents of harm, with implications for the deployment of weapons containing Uranium. In principle, the Secondary Photoelectron effect may provide a mechanism to explain the observed toxicity of heavy metals.

Quantifying the discrepancy between ICRP and a new model that takes account of the Secondary Photoelectron effect. 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 [xliii] 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. 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 quantity of DNA in a cell is about 7 picograms. The cell has a mass of 270 picograms, assuming an 8 micron diameter cell. So the DNA represents roughly 1/40th by mass on the basis of these BEIRV figures.[xliv] 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 - the principal target for radiation effects - 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. [xlv]
The photoelectron idea was presented by Busby at the CERRIE international workshop at St Catherine's College in 2003.[xlvi] The UK Committee on Radioactive Waste Management commissioned work on the relevance of SPE on public exposure to Uranium.[xlvii], [xlviii] and the argument outlined above was formally presented to the MoD Depleted Uranium Oversight Board in 2004. Papers have been published. [xlix] [l] [li] The UK HPA's treatment of SPE, Bonfield, and the Pattison paper have been cut to 3 new papers to be posted on www.llrc.org

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

The UK Health Protection Agency has engaged in dialogue on SPE with the LLRC but has used wrong methods and has not fulfilled various undertakings, thus obstructing further discussion. The scientific issues discussed by HPA and LLRC have not been resolved. A paper by Pattison et al. On this issue has been criticised for inappropriate criteria on particulate Uranium, for inappropriate methodology, and for failing to address those aspects of the SPE hypothesis which involve atomic Uranium.

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.[liii],[liv],[lv] Excess risks are associated with nuclear sites,[lvi],[lvii],[lviii],[lix],[lx],[lxi],[lxii],[lxiii],[lxiv],[lxv] and with contaminated coasts and estuaries,[lxvi],[lxvii],[lxviii],[lxix] phenomena which are probably 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.[lxx] Studies said to falsify earlier positive results may be confounded by Chernobyl fallout.[lxxi] 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. 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,[lxxii] the Strahlenschuztkommission response to KiKK,[lxxiii] and UKAEA's response to reports of prostate cancer.[lxxiv] ICRP routinely fails to cite such anomalous studies. The recently retired Scientific Secretary of ICRP has admitted [lxxv] 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.[lxxvi]

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.[lxxvii] 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 lxxi 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.[lxxviii],[lxxix]

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 [lxxx] is frequently cited as evidence that the Chernobyl disaster has had no observable effect on health. The report 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 [lxxxi],[lxxxii] 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 [lxxxiii] 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.[lxxxiv] 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.[lxxxv] 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.[lxxxvi] 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.[lxxxvii] 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.[lxxxviii] The data have been reanalysed,[lxxxix],[xc] 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 [xci] 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.[xcii]

Any assertion that Bithell *et al.*. [xciii] and Laurier *et al.*.[*xciv*] 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.[xcv]

The COMARE 10th Report has not falsified the studies summarised above. The method employed by COMARE [xcvi] 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.[xcvii] It should also be noted that less than 5% of the externalities of nuclear power are associated with the nuclear plant itself.[xcviii] 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.

Infant leukaemia after Chernobyl

After Chernobyl a significant increase in infant leukaemia between 20% and 330% was observed.[xcix, c, ci, cii, ciii civ, cv, cvi] It has been proposed that this phenomenon stands as unequivocal evidence of a large error in ICRP risk factors.[cvii] 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 (cvii above) 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 post-Chernobyl increase in infant leukaemia was a major factor influencing the establishment of the CERRIE committee in 2001. The treatment of this topic by the CERRIE Majority report was contradictory.[cviii] COMARE was fully aware of its importance in CERRIE's discussions but does not mention it.[cix] COMARE Chairman Professor Bryn Bridges defended the 9th report by citing *"the forthcoming" European Childhood Leukaemia/ Lymphoma Incidence Study (ECLIS) which 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.

Infant Mortality

No-one has refuted the proposal [cx] 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.[cxi],[cxii],[cxiii],[cxiv]. 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 [cxv] the doubling dose for congenital abnormalities is 21.3 Gy. However, Scherb [cxvi] [cxvii] and other workers [cxviii] 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 [cxix] 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 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. [cxx] The authors show that the findings summarised were not an artefact of increased surveillance after Chernobyl. They cite [cxxi] 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 [cxxii] associated with high levels of natural background radiation.

[i] 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. viii.

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- 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?

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