

RADIATION HEALTH EFFECTS

Dr Paul Dorfman

University of Warwick, member SAFEGROUNDS Team, member SAFEGROUNDS PSG, Chair of the Nuclear Consultation Group (NCG), co-Chair SD:SPUR, member of Steering Group (MoDSDPSG) and Advisory Group MoDSDPIAG) to MoD Submarine Dismantling Project (SDP), former co-Secretary to the Committee Examining Radiation Risks from Internal Emitters (CERRIE)

1. INTRODUCTION

Studies concerning the interaction of ionising radiation and the living environment (to determine differing pathways to, uptake of, and metabolism by differing soils, plants, and organisms) take many forms. The most direct primary negotiation is that between humans and radiation (radiation exposure to human populations). At present, the institutional knowledge construct of these primary negotiations determine the outcome of the LLR risk debate. In other words, the current institutional regulatory view of LLR is that: Although there exists no safe threshold for LLR, current radiation protection practices, disposals and dispersals do not present a significant risk to the UK population.

This determination directly effects risk distributions at the local regulatory level. Scientific LLR risk assessment is dependant on differing epidemiological, cellular, animal, and human experiments to determine whether current/historical operational/accidental emissions from nuclear plant significantly compromises the health of proximal populations. This negotiation is diagnosed via cause/effect (causation) in the context of proof of risk to critical groups and/or communities at risk via the assumed complete identification of:

- Concentration of quantity and quality of radioactive pollutant.
- Pathways through environment.
- Uptake and metabolism to receptor (e.g. human beings and other organisms).

In greater detail, the performance of the LLR risk may be seen as a nested set of inter-related debates concerning differing:

- Radio-isotopic species.
- Levels of radiation emissions (both authorised and accidental releases).
- Affected communities (e.g. differing radiation susceptibility of critical/age groups).
- Radiation pathways through the environment.
- Receptor uptake and metabolism (e.g. radiation translocation, residency and excretion rates).
- Radiation health effects (the aetiology of cancer, leukaemia, and other diseases).

These sets of debates are subject to differing fundamental scientific radio-biological (mechanistic) and epidemiological (direct effect) laboratory negotiations involving differing quantities and qualities of ionising radiation insult, including:

- High or low linear energy transfer (LET)¹.
- Single or continuous, periodic or episodic exposure.
- Anthropogenic (man-made), or natural background radiation (NBR).

These insults are delivered to differing receiving ecosystem stages such as population, community, organism, molecular, and cellular levels. The cumulative outcome of these laboratory negotiations provide differing experimental data concerning both deterministic and somatic, or stochastic effects. All of the forgoing laboratory negotiations are translated into models of environmental management via filtration through, and validation by, differing international and national scientific advisory bodies who produce instrumental institutional knowledge constructs concerning radiation risk, which are embodied in incrementally evolving sets of regulatory regimes enacted by the Nuclear Installations Inspectorate (NII) and the Environment Agency (EA) comprising standard setting for:

- UK nuclear industry nuclear plant operation².
- Environmental emissions from nuclear plant³.
- Secondary calculation of the interaction of those emissions with the human population and the environment⁴.

All the above are intimately interwoven and interrelate. Fundamental science and, hence, regulation attempts to account for these interactions via directed research⁵.

¹ Slow moving heavy (high LET) particles such as alpha particles and neutrons may leave a characteristic pattern of damage. These radiations are densely ionising because they wreak havoc within a short tunnel. Low LET or sparsely ionising X-rays or gammas spread damage along a much longer path. Because the damage from densely ionising radiation is so concentrated, it is far more likely to hit one chromosome several times, triggering deletions or re-ordering of DNA.

² Particular elements of the regulation of nuclear operations have specific legal meanings, including 'as low as reasonably practical' (ALARP), & 'best available technology not entailing excessive cost' (BATNEEC). A humorous acronym often deployed by the alternative network is 'CATNIP' (cheapest available technology not involving prosecution).

³ E.g. general derived limits, consents and authorisations to discharge.

⁴ E.g. critical group, dose, pathway, uptake, and metabolism.

⁵ The LLR risk controversy may be seen as a nested set of debates containing a number of inter-locking elements. Laboratory, and hence, regulatory practice attempts to successfully account for these interactions via an extraordinary weight of directed research. Realms of dispute may include those of knowledge construction about the:

- Relative activity and biological effectiveness of differing species and particle size of radioactive isotopes.
- Levels of acceptable exposure (dependent on epidemiological, cellular & animal laboratory negotiations).
- Applicability of evidence (e.g. differing scopeing assumptions under-pinning differing laboratory and epidemiological research methodology); extrapolation from animal studies; extrapolation from effects at middle/high dose & dose rates.
- Relative carrying, assimilating, diluting or bio-accumulating capacities of receiving environmental systems.

2. UNDERSTANDING RAD RISK

Despite the key nature of the debate, the definition of radiation health risk is by no means agreed - in fact this risk definition remains highly controversial and open to critical analysis. This debate runs parallel to other equally fierce battles between opposing groups proposing differing solutions to questions about military security and deterrence; the disposal of radioactive waste; the half-life of a particle of uranium; the relative costs and benefits of nuclear powered energy in a warming world; and the effect of a micron sized plutonium particle on the tracheal bronchial lymph node of a child.

Direct attention to the question of LLR risk has emerged, almost as an after-thought, to the nuclear project. This has resulted in chronic LLR releases to the environment from civil and military reactors, transports, and waste dumps. Significantly this debate has a history and a trajectory

As discussed, at present the current institutional regulatory view is that, although there is no dose which does not carry a risk, radiation pollution from UK nuclear plants is relatively safe. However, other work on radiation risk provides an alternative view (ECRR, 2003; Lesvos Declaration, 2009) – that chronic man-made LLR pollution does indeed present a significant risk to human health.

3. RADIATION EPIDEMIOLOGY

Radiation epidemiology - the analysis of incidence and distribution of disease, is fundamental to radiation risk determination and standard setting. Epidemiological investigations ranging from A-bomb survivor studies to more numerically and temporally limited studies have provided an enormous weight of evidence about the effects of ionising radiation on humans. Since the link between radiation and the aetiology of cancer and leukaemia is well documented, this aspect of the debate has devolved to an intense, long-lived, and at times vitriolic discussion of the risks of those diseases, in the survivor populations of Hiroshima and Nagasaki, post-Chernobyl, and near to operating nuclear installations.

3.1 Problems with LLR Epidemiology

There are a number of problems that are associated with epidemiological LLR risk studies – including the purpose and limitations of the epidemiological method (see Hill 1965). This is because, as Ron (1998, p S30) notes, *"unfortunately the inherent limitations of epidemiology make it extremely difficult to directly quantify health risk from these (LLR) exposures"*. This

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- Movement of radionuclides within those environmental systems (e.g. deposition, re-suspension, & washout).
 - Pathways to receptor (e.g. inhalation, ingestion, dermal contact).
 - Human receptor susceptibility (e.g. differing age groups, radiation susceptibility, intra and inter-generational exposure history).
 - Human metabolism mechanisms (e.g. concentration in organs such as the thyroid, tracheo-bronchial lymph nodes).

limitation is potentially significant since epidemiology is the primary standard setting tool for LLR risk, e.g. A-bomb survivor data sets, and HPA review of rad-risk associated with Genomic Instability and Bystander Effect.

The epidemiological method contains a number of further limitations and uncertainties. Firstly, since epidemiology is observational, exposure cannot be controlled. Secondly, there exists the potential for the incorporation of uncertainties in radiation dose insult reconstruction for exposed worker and civilian populations. For example, Wing et al (1994) and Richardson et al (2000) question the accuracy of worker LLR exposure data studies and suggest that wide-spread dosimetry practices from the 1950's to the late 1980's (where low dose were not recorded), may have masked problematic LLR effects. In other words, *post-hoc* epidemiology may have been based on uncertain information⁶. Thirdly, epidemiological investigation may neither isolate nor assess the compound negative synergistic effects potentially associated with the problematic interaction of radiation with other carcinogens⁷. Lastly, there is always the potential for confounding factors and bias in epidemiological negotiations introduced via incomplete, inaccurate or misrepresentative historic and current emission and monitoring information⁸.

3.2 A-Bomb Survivor Data

The single most important sources of information upon which institutional radiation risk standards are built are the Hiroshima and Nagasaki A-Bomb survivor studies.

In 1958, 13 years after A-bomb detonation, a cohort of 91,000 people were chosen for long-term follow-up from the 120,000 survivors identified by the US Atomic Bomb Casualty Commission (ABCC) from census data for 1950. These survivors (who had not died of post-blast injuries, disease, increased immune deficiency, malnutrition, and old age between 1945-1950) are known as the Life Span Study (LSS) cohort. In general, the institutional interpretation of this data set tends to suggest that there have been few significant health legacies in the A-bomb survivor population. However, A-Bomb dosimetry has been revised a number of times by radiation protection institutions such as the UNSCEAR, ICRP and IAEA (see UNSCEAR 1988; BIER V, 1990; and ICRP, 1991). Assumptions that were reified in radiation protection regimes (*ibid*) were found to be subject to bias due to a number of factors:

⁶ See Wing (1994) for US worker dose-reconstruction problems; see NRC (1995) for institutional dose reconstruction methodology, & see ECRR (2002) for problems associated with Chernobyl dose-reconstruction.

⁷ See Lord (1998) regards negative synergy between pre-conditioning radiation insult and subsequent carcinogenic chemical insult.

⁸ Some further uncertainties associated with LLR epi risk include:

- Radiation eco-toxicology (Ward-Whicker, 2000⁸; Copplestone et al, 2000⁸)
- Radiation deposition models - the use of simple emission dispersion modelling (Basham and Whitwell, 1999)
- Differential radiation-susceptibility of foetus, pregnant women, and other individuals and populations⁸, e.g. those with Downs Syndrome

All the above have the potential to effect risk distribution and, hence, impact upon LLR risk.

- Increased cancer death at longer latency for low dose exposure cohorts.
- The estimated neutron dose received by survivors was underestimated because experimental data was extrapolated from the dry air of the Nevada desert to the humid air of Japan.
- The bomb yield was underestimated by circa 20% because of building shielding and reconfiguration of insult to organs and tissues.
- The exchange of a multiplicative for an additive radiation effect risk model.

Perhaps the most important and cogent critique of the institutional interpretation of A-bomb data is that of Stewart and Kneale (1990, 1992, 1993, 2000) who suggest significant statistical inconsistencies inherent in the LSS A-bomb data⁹. *Inter alia* the institutional networks assumption that the survivors represent an homogenous population. The Stewart and Kneale (ibid) hypothesis (that the survivor populations are not homogenous), is supported by the following evidence:

- The cohorts for the LSS and the Adult Health Study were not established until 1958, 13 years after the bombing. They were, however, defined on the basis of census data for 1950, 5 years after the bombing (Yamashita and Shibata, 1998).
- Thousands of post-detonation deaths occurred before 1950, potentially due to heightened susceptibility to infection due to increased immune deficiency (precluding survival and thereby pre-empting potential development of cancer or leukaemia).
- Those most at risk (the young, the old, the sick) may well have succumbed to bomb related illnesses before 1950. In other words, the cohort may be subject to confounding via age bias and selection for good health.

The main thrust of Stewart's discussion are also rehearsed in a series of detailed studies and reviews concerning LLR risk by Kohnlien and Nussbaum (1991, 1992, 1995, 1998), Nussbaum and Kohnlein (1994, 1995, 1996) and Nussbaum (1998) who revisit the A-bomb data question. They interrogate Stewart and Kneale's (1990, 1992, 2000) non-heterogeneous LSS cohort hypothesis and confirm those findings, concluding that the surviving ABCC collective represents an extraordinarily selected (and therefore unrepresentative) 'healthy population'. Importantly, Kohnlein and Nussbaum's (1991) interrogation of A-bomb victim RERF follow-up data sets (which incorporate the DS86 dosimetry changes [see Shimizu et al, 1988]), demonstrate statistically significantly increased cancer mortality for low-dose sub-cohorts¹⁰. This is important for the LLR debate because they conclude that current LLR insult to worker and civil populations should not be regulated under risk estimates derived from the different A-bomb exposure conditions. Further, they note that excess leukaemia in LSS is associated with doses

⁹ It was Stewart (1956) through her ground-breaking work with *in utero* irradiation effects first challenged the institutional LLR safety claim.

¹⁰ Goffman (1990) also confirms these findings.

down to 1.6 cGy. Indeed Carter (1993 in Kohnlein and Nussbaum, 1994) fits a non-threshold upward convex line to the dose-response curve¹¹. Thus, Kohnlein and Nussbaum (1995) discount the concept of hormesis, not least because of foetal and childhood radio-sensitivity at low doses¹². In the context of A-bomb data, Nussbaum (1998) finds no support for the institutional dose and dose-rate effectiveness factor (DDREF) hypothesis; noting that LSS data demonstrates:

"A statistically significant increase in mortality between 0-10 cSv, and a three-fold change in slope (risk/unit dose) between the lowest and intermediate dose, with a convex... linearity" (ibid, p. 293).

In summary, Kohnlein and Nussbaum suggest that current conventional LLR risk estimates are in error by *circa* 20-fold.

3.3 Post-Chernobyl Epidemiology

On the 26th April 1986 an explosion at the Chernobyl Nuclear power Plant Number 4 in Northern Ukraine resulted in widespread atmospheric pollution by fission-product radioisotopes. Perhaps it should come as no surprise that (as with other aspects of the LLR debate) the epidemiological evidence from post-accident studies is both confusing and conflicting. However the institutional networks evaluation of the health consequences of this catastrophic event is clear and unambiguous. Current institutional epidemiology concludes that there is no evidence of a major public health impact attributable to radiation exposure after the accident.

However, there exists a significant body of evidence that tends to contradict the institutional response to the catastrophe at Chernobyl. This includes epidemiological studies regarding adverse post-Chernobyl health effects - increases in congenital malformations in the new-born (Lazjuk et al, 1993), increase in infant leukaemia (Petridou et al, 1996), a sharp increase in infant leukaemia in Scotland and Wales (Busby and Cato, 2000), and vastly increased incidence of childhood thyroid cancer (Burlakova et al, 1996¹³; Kasakov et al, 1992). These rates of increased incidence of ill health in effected geographical areas were not predicted by extrapolation of current radiobiological knowledge. The Director of the Russian Environmental Policy Centre concludes that:

"Either the Chernobyl ejection was several times bigger than declared... or else our knowledge about the effects of radiation on the human organism is inadequate and must be reviewed. In my opinion, both suggestions are valid" (Yablokov, 1996, p. 5).

Scheer (1992) demonstrates a slowing in the decrease in German neonatal mortality post Chernobyl, and Petridou et al (1997) confirm a statistically significant excess of 2.6 RR for infants exposed to Chernobyl fall-out *in utero*.

¹¹ Excess leukaemia's arise in LLS cohort in the mid 1950's (see Kerr, 1987; Goffman 1990).

¹² RERF also conclude that that some evidence points to increased risk for those exposed at a very young age (Shigematsu, 2000).

¹³ Further, Burlakova (1994; 1995; and Burlakova et al, 1996) posit a bi-modal (bi-phasic or non-monotonic) dose response to radiation insult.

Ivanov et al (1996), in contrast to earlier findings (see Ivanov et al, 1993), find a doubling of leukaemia yield in total population of 'liquidators'. However, the authors conclude that (in the absence of a dose-response relationship) the explanation for this increase may be due to better medical surveillance, and hence registration. In the US Mangano (1997) shows a doubling of risk for childhood leukaemia within *in utero* cohort, and notes a potential for statistical bias and confounding in large European studies (e.g. Auvinen et al 1994; Hjalms 1994) which could account for aspects of their negative findings. The German Childhood Cancer Registry (Michaelis et al, 1997) reported an increase in infant leukaemia in post-Chernobyl, but since results demonstrated a non-linear dose-response relationship (where the highest risk relates to the lowest exposures), the authors concluded that the excess illness was not caused by radiation exposure. Dalko (1988), notes a clear correlation between Chernobyl and A-Bomb non-cancer and leukaemia effects, and suggests a link between increased incidence of disease other than cancer and leukaemia in both of the cohorts¹⁴.

In the UK, Busby and Cato (2000) demonstrate a sharp increase in infant leukaemia in Scotland (RR 4.4) and Wales (RR 3.7), and low birth weight in Wales, for those exposed *in utero* at the time of Chernobyl fallout. Note, infants from cohorts born later show no such effect, thus demonstrating problematic effects of *in utero* radiation insult. Further (with reference to NRPB Chernobyl exposure and leukaemia yield estimates) the authors calculate that the current radiation risk factors are in error by *circa* 100-fold. Savchenko (1995) describes excess leukaemia yield in children, as do the International Commission on Chernobyl (1996). Sperling et al (1994) notes significant increases in trisomy 21 in children in Berlin. Kazarov et al (1992), Baverstock et al (1992), Williams et al (1993; 1994), Williams et al (1994), Williams (1997), Abelin and Eger (1994), and Stsjazkho et al (1995) all report dramatically increased thyroid cancer yield in children of Belarus and relate this effect to releases from the Chernobyl accident. It is of interest to note that commentary on childhood cancer post-Chernobyl by the institutional network (e.g. Boice and Linet, 1994) fail to emphasise the magnitude of this increase. Yamashita and Shibata (1998) find a significant childhood thyroid cancer increase of 36% overall (54% in Kiev), and suggest an association with Chernobyl fall-out not predicted by current radiation risk standards based upon A-bomb survivor data (e.g. see Akiba et al, 1990). The authors also expressed regret at the delay in establishing an appropriate fixed cohort for assessing risks from the accident. A large study by Astakhova et al (1998) concluded that case-control comparisons indicated a strong relationship between significant excess thyroid cancer incidence and estimated radiation dose from the Chernobyl accident.

Further evidence of post Chernobyl problems are reported in: ECRR *Chernobyl 20 Years On: Health Effects of the Chernobyl Accident* European Committee on Radiation Risk, Documents of the ECRR 2006. With each updated report the Chernobyl harvest grows, and there exists a

¹⁴ Non-cancer radiation effects are not emphasised by the institutional network, and have not been subject to similar levels of investigation. Note, it is precisely these effects that are predicted by genomic instability.

significant body of evidence from Russia, Ukraine and Belarus, that clearly contradicts the institutional response to the human and environmental disaster (ECRR, 2006). As Prof Alexey Yablokov, Director of the Russian Academy of Sciences, Moscow concludes:

'Each year it has become clearer and clearer that the real consequences of this catastrophe are much more widespread and severe than has been predicted' (Yablokov, ECRR, 2006, p. 34).

3.4 Childhood Cancer and Leukaemia Clusters near UK Nuclear Installations

There is a proven, highly significant, universally acknowledged, and on-going 10-fold childhood leukaemia excess near the reprocessing plant of Sellafield. There has also been a significant 8-fold increased incidence of childhood leukaemia in Caithness near the Dounreay reprocessing plant in Scotland, and a statistically significant childhood leukaemia excesses were found in the West Berkshire region near the Atomic Weapons Establishment (AWE) at Aldermaston, and former USAF Greenham Common.

COMARE have published influential reports on these radioactive environmental risk controversies (COMARE, 1986; 1988; 1989; 1994; 1998; 1999; 2005). Without exception, all of the COMARE reports have concluded that none of the excess childhood leukaemia's or cancers in the local population could possibly be explained by exposure to radioactive emissions resulting from normal operations of those nuclear facilities. Interestingly, although about half of the members of the Committee Examining Radiation Risk from Internal Emitters (CERRIE, 2004) were concerned that raised rates of cancer and leukaemia near nuclear plants in Sellafield and Dounreay 'may well be linked to radio-nuclides from nuclear facilities'; COMARE's (2004) document on the work of CERRIE did not respond to this concern.

However, the radiation risk and health debate is ongoing. A very recent case control investigation of the German Childhood Cancer Registry (GCCR), carried out on behalf of the Federal Office for Radiation Protection in 41 districts in the vicinity of the 16 nuclear power plant sites in Germany between 1980 and 2003, found that risk of tumour or leukaemia in children under 5 years of age significantly increases the closer they live to a nuclear power plant (GCCR, December 2007).

4. RADIATION BIOLOGY

4.1 Committee Examining Radiation Risk from Internal Emitters (CERRIE)

The internal radionuclide argument is hot because it's here that fundamental scientific uncertainties are greatest – and this has real implications for safety standards. Although conclusions from CERRIE were mixed, the Committee stated that 'uncertainties in dose co-efficients for some radionuclide were large', and that 'a particular concern was the adequacy of current models for

the estimation of risks for short range alpha, beta and auger emitters' (CERRIE, 2004). What this means is that our regulatory protection standards for some important internal radionuclide emitters is subject to uncertainty of an order of magnitude - a factor of 10. In other words - could be out by 10 times. It should also be noted that the CERRIE Minority Report (2004) suggested that current regulatory radiation protection standards are in error by at least 2 orders of magnitude – out by at least 100 times.

4.2 Non-Targeted Effects

4.2.1 Genomic Instability

One of the most significant radiation-biology findings concerns the acknowledged phenomena of 'genomic instability'¹⁵. Genomic instability research has demonstrated a novel alpha particle irradiation effect at low levels (Kadhim et al, 2001). Although the underlying mechanisms (molecular, genetic and cellular) for this phenomenon are not fully understood, the single most important implication of genomic instability is the potential for enhanced germ-line mutation of the human gene pool.

Not only does genomic instability suggest that radiation health effects are potentially far more widespread, but risks potentially arise after exposure to doses far lower than current safety limits allow. The number and complexity of the biological effects of differing qualities of radiation tends to bring into question the concept of dose.

The implications of genomic instability are many and varied. Since these changes are unpredictable they are potentially implicated in a range of diseases other than cancer, e.g. immune suppression and degenerative diseases - thus traditional epidemiological methods may fail to pick up this link since the level of effect is too uneven, and the numbers of diseases (potentially induced) so wide.

It is significant that genomic instability demonstrates substantial differences between different qualities of radiation. For current radiation protection purposes, alpha radiation is considered to act similarly, albeit more effectively than other radiations. However, genomic instability demonstrates that the difference is not simply a matter of efficiency - rather there is a real qualitative difference in the action of differing radiations. In other words, the concern is that genomic instability provides a mechanism whereby low-level alpha radiation can transmit down to the blood-forming system.

4.2.2 Bystander Effect

The 'Bystander Effect' (BE) represents a totally unforeseen twist in the LLR debate. Complex cellular laboratory negotiations have found an unexpected

¹⁵ Morgan et al (1996, p. 247) define genomic instability as an 'all-embracing term to embody a variety of genomic alterations, including chromosomal de-stabilisation, gene amplification and mutation', thus 'genomic instability is characterised by the increased rate of acquisition of alterations in the mammalian genome'.

sensitivity to mutation induction in cells proximal to other cells insulted with very low-level dose alpha radiation (see Nagasawa and Little, 1992, Lehnert and Goodwin, 1997; Azzam et al, 1998; Blakely et al, 1998; Edouard et al 1998; Edwards, 1998; Nagasawa and Little 1999). BE's are observed in cells in the vicinity of other cells subject to cytoplasmic irradiation. In other words, the effected cells are not themselves irradiated but are in the neighbourhood of irradiated cells. BE's include P53 protein expression, sister chromatid exchanges, cyto-toxicity, gene mutation and chromosomal instability. Thus, problematic responses in cells can result from indirect radiation insult. In effect, this means that a cell that has not been traversed by any radiation track can still potentially incur damage.

4.2.3 Implications of GI and BE

As Wright & Coates note (Mutation Research 597 (2006) 119–132

Untargeted effects of ionizing radiation: Implications for radiation pathology):

The dogma that genetic alterations are restricted to directly irradiated cells has been challenged by observations in which effects of ionizing radiation, characteristically associated with the consequences of energy deposition in the cell nucleus, arise in non-irradiated cells. These, so called, untargeted effects are demonstrated in cells that have received damaging signals produced by irradiated cells (radiation-induced bystander effects) or that are the descendants of irradiated cells (radiation-induced genomic instability). Radiation induced genomic instability is characterized by a number of delayed adverse responses including chromosomal abnormalities, gene mutations and cell death. Similar effects, as well as responses that may be regarded as protective, have been attributed to bystander mechanisms. Whilst the majority of studies to date have used in vitro systems, some adverse non-targeted effects have been demonstrated in vivo. However, at least for haemopoietic tissues, radiation-induced genomic instability in vivo may not necessarily be a reflection of genomically unstable cells. Rather the damage may reflect responses to ongoing production of damaging signals; i.e. bystander responses, but not in the sense used to describe the rapidly induced effects resulting from direct interaction of irradiated and non-irradiated cells. The findings are consistent with a delayed and long-lived tissue reaction to radiation injury characteristic of an inflammatory response with the potential for persisting bystander-mediated damage. An important implication of the findings is that contrary to conventional radiobiological dogma and interpretation of epidemiologically-based risk estimates, ionizing radiation may contribute to malignancy and particularly childhood leukaemia by promoting initiated cells rather than being the initiating agent. Untargeted mechanisms may also contribute to other pathological consequences.

5. SUMMARY

There exists very real concern about, and significant lack of consensus on, the definition of LLR risk, and hence current LLR risk policy. Current radiation risk standards are subject to large levels of fundamental scientific uncertainty, and may underestimate risk to public health.