

The attached has been written by Dr C Busby, a member of CERRIE, and represents his own views and includes those of Mr Richard Bramhall, also a member of CERRIE. It has not been considered by the committee. For convenience, and at Dr Busby's request, it is circulated to delegates with the committee's permission.

# CERRIE: NATURAL PHILOSOPHY AND DELIBERATIVE SCIENCE

## 1. The Internal Exposure Debate

Internal exposures are from radiation emanating from unstable radioactive isotope sources from within the body (e.g. Strontium-90 from drinking milk) as opposed to external irradiation which originates outside the body (e.g. from X-ray machines, A-bomb flashes). The first real evidence that low dose internal radiation may be dangerous came from the US, where Dr Ernest Sternglass (1963) applied the obstetric X-ray findings of Prof. Alice Stewart (1957) (40% increase in childhood cancer at foetal doses of 10mSv in England) to fallout doses to the public. Sternglass (ibid) found significant increases in infant mortality at the peaks of the global weapons fallout in many countries, including the UK and these findings have been confirmed by more recent research.

Mainstream science successfully marginalised the findings of Sternglass and dismissed the claims of Alice Stewart - but not before Sternglass had persuaded Kennedy to negotiate the Atmospheric Test Ban Treaty. Sternglass's findings were vigorously supported by US Atomic Energy Commission (AEC) scientists: Dr John Goffman and Dr Arthur Tamplin, who then were forced to resign from AEC. Both Goffman and Tamplin have sustained their critique of the institutional low dose radiation model (Tamplin, 1969; Goffman and Tamplin, 1971; Goffman, 1990) - with Tamplin being the first to draw attention to the consequences of internal 'hot particle' exposures.

The discussion about the health consequences of internal exposures to man-made radioactivity became part of mainstream UK scientific debate in the early 1980s following the discovery of the Sellafield child leukaemia cluster. Despite evidence from the National Radiological Protection Board (NRPB) - that doses were too low to explain the excess risk; the Black Commission (tasked with interrogating these findings) recommended setting up a new independent committee to examine the possibility that significant increases in childhood leukaemia/cancer proximal to nuclear plant may be caused by radiation pollution. This committee would remain in existence to monitor scientific developments in this area.

COMARE have published five influential reports on a number of UK radioactive environmental risk controversies. These include investigations into childhood leukaemia excess in areas near Sellafield nuclear plant (COMARE, 1986 and 1996); Dounreay nuclear establishment (COMARE, 1988); AWRE Aldermaston and ROF Burghfield (COMARE, 1989); and former USAF Greenham Common (1998). Without exception, all five reports have concluded that (within the context of the current institutional radiation protection models), the acknowledged clear and significant excess childhood leukaemia and/or cancer in the local populations could not be explained by exposure to ionising radioactive emissions resulting from normal operations of the nuclear facilities in question. In other words, *"the levels of radiation in the*

*local area are so low that they could not be responsible for the local incidence of childhood leukaemia” (COMARE, 1998, p. 40).*

This essentially deductive conclusion is based on the application of the risk models which are based primarily on the A-Bomb survivor studies (ICRP, 1990), and a few other external irradiation studies to the doses that the children near Sellafield could have received. On the basis of these models, there is a mismatch between the expected numbers of leukaemia's and those observed of about 300 times. That is to say that the Seascale children with leukaemia would have needed to receive 300 times more radiation than they could have received on the basis of the known radioisotopic releases and environmental measurements from Sellafield. The 300-fold error in published risk estimates is similar for the child leukaemia cluster found near Cap de la Hague, in France in the mid 1990s (Viel and Richardson, 1990; Viel et al, 1993; Viel and Poubel, 1995). Thus, either:

- the doses are much larger than are admitted, or;
- the radiation risk model based on the A-Bomb external acute exposures is incorrect for internal chronic exposures from man-made radiation, or;
- there is some other effect particular to all these sites which has nothing to do with radiation.

The first of these has been ruled out. The only slightly persuasive example of the alternative explanation for the leukemias is based on a theory of population mixing leading to infection to which childhood leukaemia is a rare response. COMARE concede that this explanation cannot be used for either the Sellafield or the Aldermaston leukaemias since the former is ongoing, and the latter is not geographically isolated.

For those outside NRPB, COMARE, the nuclear industry and its scientists; the application of 'Occams Razor' provides a persuasive explanation. Radiation is implicated in the aetiology of leukaemia - so a discovery of excess leukaemia near the largest source of radioactive pollution in Europe is likely to be caused by radiation from that source. Put simply, there seems to exist a credibility gap between the risk model of the ICRP and NRPB (organisations which share many personnel) and the beliefs of the public, and independent research groups like those in the European Committee on Radiation Risk (ECRR).

CERRIE came about as a result of assertions made by a number of independent environmental scientists and groups that risk models currently accepted by UK government departments and regulators (i.e. the models used by the International Commission on Radiological Protection, [ICRP]) underestimate the health risks from intakes of radionuclides by large factors (hundreds). The basis of these assertions includes:

- Epidemiological evidence and genetic evidence of harm in those exposed to low dose internal man-made radioactive pollution, specifically in children and infants after Chernobyl.

- A priori arguments about mechanistic differences between internal and external radiation and also man made and natural radiation exposures.
- Debates relating to new discoveries from cell biology about radiation effects in cells e.g. genomic instability, Auger location effects, bystander effects and other aspects of exposure at the cellular level.
- The notion that the scientific method is an inductive process that should give weight principally to observations of those exposed to internal radiation in the development of radiation -risk protection models (see ECRR2003).

**Table 1.** Scientific Advisory Developments pre CERRIE

<b>Discovery</b>	<b>Government response</b>	<b>Findings</b>
1. (1983) Sellafield leukaemia cluster	Ask NRPB	Epidemiology mistaken. Doses too low for health effect. If real, not linked to rad-pollution.
2. (1984)Black Committee	Enquiry: Sir Douglas Black Committee	Further epidemiological studies commissioned. Formation of new rad-risk committee on mechanism (COMARE) and on small area risk (SAHSU).
3.(1984-present) Childhood leukaemia near nuclear sites in UK (Dounreay, Aldermaston, Hinkley Point, Bradwell)	Ask COMARE	Effects are real but doses too low to be radiation on the basis of present models: must be something else, perhaps 'population mixing' in Dounreay but not Sellafield or Aldermaston.
4. (1986- present) Chernobyl.	Ask NRPB and COMARE	Chernobyl will have no measurable radiological health impact
5. (1987-present) BSE/CJD	Ask Spongiform Encephalopathy Committee.	Finds no crossing of species barrier.
6. (1987-2000) Infant leukaemia, minisatellite mutations, genomic instability other new science, new epidemiology by Greens. New European legislation on radioactive exposures and discharges.	Ask NRPB ask COMARE	No need to revise present risk models.
7 (1998-2001) representations to government about errors in radiation risk models	Set up CERRIE	

## 2. Deduction and Induction

Those members of CERRIE who criticise the present risk model for internal radiation exposure drew attention to what they believed to be errors in the way in which scientific method had been applied in the historical development of the ICRP model for this type of exposure. At June 2003, these considerations have not yet been discussed; however they will form a part of the general discussion following the July workshop, and of the final report. Therefore a brief outline of the representations made by the critics of the ICRP risk model is given here so that responses at the workshop may deal with this issue.

The critics' argument on the philosophy of scientific method follows the classical exposition of the scientific or inductive method - what is now called Mill's Canons. The two most important of which are:

- The Canon of Agreement, which states that whatever there is in common between the antecedent conditions of a phenomenon can be supposed to be the cause, or related to the cause, of the phenomenon.
- The Canon of Difference, which states that the difference in the conditions under which an effect occurs and those under which it does not must be the cause or related to the cause of that effect.

In addition, the method relies upon the:

- Principle of Accumulation (which states that scientific knowledge grows additively by the discovery of independent laws).
- Principle of Instance Confirmation (that the degree of belief in the truth of a law is proportional to the number of favourable instances of the law).
- Plausibility of Mechanism.

These are the basic methods of science [Mill, 1879; Harre, 1985; Papineau, 1996]. The questions of interest to CERRIE are as follows:

1. As a baseline estimate, what are the health consequences of exposure to external radiation doses at levels below 2mSv? (the approximate annual dose received from natural background).
2. What are the health consequences of exposure to novel internal radioisotopes at whole organ dose levels below 2mSv?
3. Are there differences between 1. and 2.?
4. Is the concept of dose applicable to internal radiation exposures?

Although risks from exposure to high levels of ionising radiation are generally accepted, since they are fairly immediate and graphic, the situation with regard to low-level exposure is curious. There are now two mutually exclusive models describing the health consequences of such exposure. There is the ICRP one, which is that which is presently used to set legislation on exposures and argue that low level radiation is safe, and a radical one, which is espoused by the anti-nuclear movement and its associated scientists.

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

By comparison, the more radical model arises from an inductive process. There have been many observations of anomalously high levels of cancer and leukaemia in populations living near nuclear sites in the UK and in many parts of the world, especially those where the measurements show that there is contamination from man-made radioisotopes, e.g. reprocessing plants. In addition there are populations who have been exposed to man-made radioisotopes from global weapons tests, 'down-winders' living near nuclear weapon test sites, and those exposed to these materials because of accidents (like the Chernobyl infant leukaemia cohort), or because of work in the nuclear industry or military. Many of these findings form part of the CERRIE deliberations. In contrast to the averaging approach of the conventional model, a rational biological model would consider each type of exposure according to its cellular radiation track structure in space and in time. It is not easily possible to employ such a model to predict risks from 'radiation dose' to 'populations' but only from microscopically described doses from specific isotopes or particles whose decay fractionations are considered to interact with cells which themselves respond biologically and bio-chemically to the insults and may (in addition) be in various stages of their biological development with hugely different levels of sensitivity to radiation. The dose-response relationship following from this kind of analysis might be expected to be quite complex. These philosophical models tend to be mutually exclusive.

Critics of the ICRP model for internal exposures problematise the extrapolation of data associated with one set of conditions (high-level, acute, external exposure) in order to model low-level, chronic, internal exposure. Further, critics argue that risks from internal exposure should be informed by studies of those exposed to internal radiation of the type that any model seeks to explain. They suggest that there exists a high risk associated with exposure to man-made fission products or some component or components of such a mixture.

Man-made radioisotopes, often in the form of 'hot' or 'warm' particles, are common contaminants of the areas near nuclear sites where there are cancer and leukaemia clusters; and of nuclear site and test site down-winders; and of fallout-exposed populations. This empirical finding satisfies the Canon of Agreement. The contingency analysis tables with control populations for such studies show that the Canon of Difference is also satisfied: people living in more remote regions than the downwinders show lower levels of illness. The Principle of Instance Confirmation is fulfilled since so many studies have shown that increases in cancer and leukaemia follow exposure regimes at low dose. This only leaves Plausibility of Mechanism, which comprises elements of past and planned CERRIE discussions.

The critics' position on the scientific applicability of the ICRP model to the yield of fatal cancer in a range of exposure types is outlined in Table 2 which is taken from the recent report of the European Committee on Radiation Risk (ECRR2003). The critics feel that it may be illuminating to ask how such a state of affairs (once black-boxed) becomes institutionally crystallised and difficult to challenge.

**Table 2.** Errors associated with ICRP extension of acute high dose external studies to other types of exposure

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

### 3. Cultural Influence in Risk Determination

The relatively intransigent nature of scientific knowledge construction in the late 1950s was addressed by a member of the British Royal Society, the Nobel-Prize winner, chemist and economist Michael Polanyi. Polanyi was interested in the scientific method, and in scientists: his writings pre-dated the Science War philosophers like Kuhn and Latour. He was aware that at any time, the current scientific paradigm was open to reconstruction. In asking how we know anything at all and how we build up a picture of the 'real world' Polanyi saw many similarities between scientists and primitive witch-doctors like the Azande who had been studied in the 1930s by the anthropologist Evans Pritchard (1937), who wrote:

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

Addressing the then scientific world view, Polanyi (1958) concluded:

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

The critics of the ICRP risk model conclude that the ICRP scientists and risk models are good examples of such systems of closed scientific communities and epicyclical logic. Polanyi's comparisons with Azande witch-doctors are familiar territory, they argue, to those who have registered the sequences of denials and (as they argue) implausible explanations which have followed discovery of the Sellafield (Seascale) child leukaemia cluster and other examples of the failure of the ICRP risk models that seem to have rejected automatically and epicyclically any experience which to ordinary people seem both massive and vital.

## **5. The ICRP model**

In this model absorbed dose or doses are defined as energy per unit mass or  $E/M$ . The quantity of mass employed is that of an organ or larger. One Gray is the absorption of 1 Joule by 1 kilogram of tissue. Very early on, ICRP had to recognise that this model was inadequate since experiments showed that it was the ionisation density that was the important factor in cell killing, and so they added a fudge factor or 'weighting' for this to the Gray to give the Sievert. For alpha decays, 1 Gray becomes 20 Sieverts.

According to its critics, the main failure of the system used to calculate dose is that the result is an average. The external dose calculation has just been applied to internal dose by averaging all energy of the decays which occur in a 'bag of water' the same size and shape of the organ over its mass. Why is this problematic? Because, they argue, it is individual cell doses which decide the magnitude of the biological effect, and for internal emitters which are point sources, some cells will receive very high doses whilst other cells receive none. Some clusters of cells will receive high doses whilst others will receive none. This is not the case with external irradiation where all cells will receive the same dose. And because the theoretical dosimetric model is problematic, it may not be appropriate to employ external irradiation epidemiology to inform us of risks from internal exposure. Table 3 addresses the way in which doses have been correlated (by ICRP) with effects like cancer and genetic damage. Critics argue that this correlation represents a problematic application of the scientific method.

**Table 3.** Problems with ICRP System of Relating Dose to Health Detriment

Stage	Problem
Assume linear dose effect response for low dose	<ol style="list-style-type: none"> <li>1. The basis is the calculation of tracks per cell per year from external averaged radiation. In the low dose range, the number of different cells hit is proportional to dose. This is not true for internal exposure.</li> <li>2. A body of empirical evidence now suggests non linear or even biphasic dose response (see Burlakova, 1994, 1995, 1996).</li> </ol>
Use external acute high dose exposure cancer yield (Hiroshima study) to model internal chronic exposure cancer yield	<ol style="list-style-type: none"> <li>1. Theoretically problematic since the internal exposures are qualitatively different</li> <li>2. A large body of empirical evidence points to much higher cancer or leukaemia yields at low internal doses.</li> <li>3. Problems of averaging over populations with different sensitivity to radiation</li> <li>4. Problems extrapolating from wartime Japanese survivors to peacetime European populations.</li> </ol>
Check against internal studies	<ol style="list-style-type: none"> <li>1. The few human internal studies considered by ICRP are of natural isotopes and high dose exposures</li> <li>2. Animal studies are of short-lived species. Those which show significant effects are never cited or followed up (e.g. Luning and Strontium).</li> </ol>
Ignore non-cancer effects	A whole range of non cancer outcomes of exposure has been ignored by ICRP e.g. infant mortality

The linear extrapolation of acute external doses verging on lethal doses have been linearly extrapolated to internal chronic doses at levels close to background radiation levels. Significant evidence of harm at low dose exposure from internal isotopes has been routinely dismissed by ICRP on the basis of the deductive application of the external Hiroshima model. Thus the ICRP model for internal exposure risk has a potentially deeply problematic theoretical basis.

## 5. CERRIE: Deliberative Science

After half a century of science-based policy, the UK and other western democracies are struggling to come to terms with the simultaneous collapse of public confidence in science-based policies and the limitations of scientific advice concerning low-probability high impact risks in the context of scientific uncertainty (Welsh, 2002).

Scientific advisory committees occupy a curiously sheltered position in the landscape of UK regulation. Traditionally their role in policy making has gone largely unobserved. Given the centrality of their role, the activities of scientific advisors are poorly documented and their impact on policy decisions is difficult to understand or evaluate (Jasanoff, 1994). Relatively recent growth in scientific advice has taken place against a backdrop of growing public concern about technological hazards, accompanied by a diminished trust of and ambivalence about the role of scientific experts in policy decision-making. Indeed, a number of commentators have argued that the questions regulators need to ask of fundamental science cannot in many instances be adequately answered (Wynne, 1992, 1994, Irwin, 1995). This deepening scepticism over

expert advice coincides with some of the lowest electoral turnouts ever recorded and a pervasive shift in public trust, acceptance, and willingness to engage with environmental scientific, regulatory and consultative processes (Welsh, 2000). In this context, core questions for UK governmental scientific expert bodies such as CERRIE may run as follows: How can CERRIE:

- Inform policy in a scientifically credible and publicly acceptable way?
- Retain its scientific authority in the context of differing views about scientific phenomena within the committee?

A ready response to such questions is interred within the initial rationale for the purpose of the committee ([www.cerrie.org](http://www.cerrie.org)):

*The committees review takes into account the views of all parties in the debate on risks of radiation. It aims to reach consensus where possible. On topics where differences of view remain after its deliberations, it will explain the reasons for these and recommend research to try to resolve them. CERRIE will agree a report that is agreed by all its members.*

In other words, central to the purpose of the committee is the acknowledgement that the contingency and complexity implicit in internal radiation risk determination must go hand in hand with a respect for plurality. There are two reasons for this. First, since no single way of understanding complex phenomena is ever adequate: the acceptance of inputs from diverse sources of knowledge is a prerequisite for better understanding, and hence improved risk decision-making. Second, because of the historic association between radiation protection practice and issues of national security, and the financial lobbies of the nuclear power industry; there is a clear trust deficit between the public, who (believe they) see clear evidence of harm and the current institutional description of radiation risk.

CERRIE's deliberative method of analysis of the internal radiation risk determination process hopes to overcome, as far as possible, the limitations of the advisory process in the context of scientific uncertainties associated with the:

- complexity of the processes involved;
- sheer range of potential effects;
- spatial and temporal diffusion of potential effects;
- diagnostic variability of cause-effect relationships;
- opportunities and pressures for bias in research and inaccuracy in results and interpretations.

Within normal scientific debate the ambiguity associated with uncertainty can be entirely beneficial (as a spur to further enquiry). However, within the scientific policy context, where highly consequential decisions may need to be taken, grave difficulties may occur if scientific discord about risk uncertainty is occluded for what could be perceived as purposes affecting institutional legitimacy. The corollary to this argument is the acceptance of honest and respectful disagreement, where dispute and uncertainty can be openly

acknowledged within the final deliberations of the Committee. The intention is not to take (or reify) sides – rather to balance and equalise differing knowledges. Its intention is not to decide between the two systems of knowledge and belief but rather to present these in a balanced and fair way. This approach should result in an enhancement of the degree of both democratic legitimacy and consequential efficiency of the UK radiation risk decision-making process. Implicit in this approach is the displacement of certainty within the risk knowledge construction, communication and management process

## **6. 'Oppositional Science'**

The problems of the accuracy of scientific advice to were addressed by Scott Cato *et al.* (2000) who argue for a new government scientific policy model. This model involves what they call 'oppositional science' and is based on the intentional polarisation of viewpoints similar to those employed in legal debate and the British system of parliamentary democracy. The methodological precursors of this model include elements of the sociology of scientific knowledge (SSK), and science and technology studies (STS). The process runs as follows: following a deliberative oppositional dialogue, final conclusions are reported symmetrically and agnostically; and in such a way that policy-makers can identify the main divisions in knowledge, belief or interpretation. Rather than being based on a spurious consensus, the concluding report presents the opposition itself, giving equal space to each side's view.

In the case of CERRIE, a further approach used by the committee was to focus on the answers to 29 questions about radiation risk from internal emitters. These defining questions were later extended in the course of the deliberations (see below). It was hoped that by concentrating on these questions the resulting answers would help to clarify the various positions of committee members, and the reasons for these positions (see below). This, in turn, may result in suggestions for further research that might help resolve any differences of opinion, risk definitions or data interpretation.

## **7. Defining Questions**

1. Has scientific method been used properly in the evaluation of the evidence from the nuclear site child leukemias?
2. Can we agree that the external model implicit in the Hiroshima study and other external radiation studies e.g. ankylosing spondylitis, tinea capitis, fluoroscopy etc is the basis of radiation risk assessment for exposure to internal isotopes?
3. Does the committee agree that there are any studies in humans which provide evidence of risk following exposure to internal isotopes; and if so what are they, & what are their limitations and what do they show?
4. Is it scientifically valid to extrapolate from high dose to low dose using a linear model; and if so, why?
5. Is it scientifically valid to average doses to cells in space for internal irradiation?

6. Is it scientifically valid to average doses to cells in time from internal irradiation?
7. What is the explanation for the quadratic portion of the dose response relationship found in external irradiation studies?
8. Are there circumstances where internal irradiation would involve local doses in a range or type that would be in the quadratic response range? (this discussion should argue through the points about correlated multiple hits to cell nuclei and the ideas that underpin the linear no threshold dose response relation for low dose and quadratic responses for higher dose).
9. Does exposure to internal isotopes result in a higher probability of local cells receiving double or multiple track traversals than exposure to external radiation at the same dose?(this point should be considered and decided on for (a) atomic single decay isotopes (b) atomic sequential decay series and (c) hot particles).
10. What evidence is there that there are not two sensitive sub groups of cells in living tissue and that their combined response to increasing radiation dose would not or does not result in a biphasic dose response relation?
11. What studies are there that show such a biphasic response?
12. Is it a correct application of scientific method to assume that the highest dose group in a comparative study must show the highest degree of effect?
13. What arguments are there that Second Event processes are theoretically irrelevant?
14. What evidence is there that Second Event processes do not carry enhanced hazard?
15. What is the significance of the bystander effect for radiological protection?
16. Does cancer involve cell communication field effects and what are their significance for radiological protection?
17. What evidence is there that transmutation of a bonded internally incorporated radionuclide has no significant biological consequence?
18. What is the radiological significance of transmutation?
19. What is the limiting size of particle that can cross the placenta?
20. Is cancer mainly an environmental disease?
21. Is there sufficient scientific evidence to exclude the exposure to Sr90 in global fallout or from other sources as a significant component in the cancer epidemic?
22. What is the likely cause of the present cancer epidemic
23. Is breast cancer caused partly by environmental factors?
24. Is there sufficient scientific evidence to exclude the exposure to Sr90 in fallout or from other sources as a significant component in the breast cancer epidemic?
25. Should the doses to the lymphatic system of children living near Sellafield be calculated using a model which averages the energy over a large mass of tissue or should the energy be averaged over the tracheobronchial lymph nodes or smaller tissue volumes?
26. Should the full calculations supplied by NRPB to COMARE be made available?

27. Should COMARE have published a report on the Sellafield leukemia cluster the conclusion of which was used for policy making but which was based on an unpublished, unchecked and confidential calculation by NRPB?
28. Is the population mixing theory an adequate explanation of the nuclear site leukaemia and cancer clusters? Is the most likely cause of these, radiation exposure?
29. Does plutonium and other material discharged to the Irish Sea from Sellafield attach to the fine particles and become deposited in offshore mud banks and estuaries where low tidal energy conditions exist?
30. Does this material end up in the bodies of people living near the shores of the Irish Sea and further inland through sea-to-land transfer? What is the evidence that this occurs?
31. What is the form of the plutonium and other isotopes involved in sea to land transfer and which part of people's bodies does the material end up in?
32. What evidence and arguments are there that would suggest that sea to land transfer of plutonium and other isotopes discharged to the Irish Sea would not represent a risk of cancer in populations living near the shores of the Irish Sea.
33. Does plutonium and other radioactive material discharged to the sea or estuaries from coastal nuclear sites end up in sediment on the shores of the coast or in estuaries?
34. Does the radioactivity find its way into the bodies of people living near the shores of the sea through sea-to-land transfer?
35. Is there any evidence that shows that this exposure route has no effect on cancer rates in those exposed?
36. Is there any evidence that childhood leukaemia risk is not associated with exposure to radioactive material re-suspended from coastal intertidal sediment and inhaled?
37. Is there evidence that plutonium from the Irish Sea is ending up inside people in England and Wales?
38. What is the likely provenance, identity and size of the radioactive particles found in airshades near Aldermaston?
39. What evidence is there that plutonium particles trapped in the lymphatic system do not represent a cancer risk?
40. What research could be recommended to investigate the hypothesis that plutonium and other radioactive micron sized particles in air are a cancer risk?
41. What is the limiting size of particle capable of transfer across the placenta?
42. Does the observation of increased infant leukaemia in five countries following Chernobyl show that the risk models of ICRP for radiogenic leukaemia are in error? Is this error upwards of 100-fold? What is the significance for radiological protection of the minisatellite mutation increases in offspring of people exposed to low level radiation from Chernobyl?
43. What size of error in the ICRP risk model does such a finding indicate?
44. Has the ICRP model adequately considered non-cancer effects of internal irradiation?

45. Has ICRP or other risk agencies like BEIR or UNSCEAR considered the non-cancer health consequences of exposure to global fallout or Chernobyl exposure which include birthrate, birthweight neo-natal and infant mortality, IQ effects and genetic damage echoes?
46. Has ICRP discussed the evidence that low level radiation causes infant mortality?

## **8. Conclusion**

The CERRIE process represents a novel attempt to overcome the problems associated with scientific uncertainties about low-probability high-impact radiation risk, and the impact that may have on the radiation protection policy process. The approach also addresses problems of bias, culture and trust where there are perceived tensions between groups in society with disparate views. CERRIE is concerned with the serious problem of radiation risk from nuclear pollution and is intended, at minimum, to tease out the essential differences in scientific beliefs in this field and recommend scientific and epidemiological research that might help resolve some of the arguments. The process has implications that go beyond radiation risk. In a world where there exist powerful economic arguments for permitting development of novel scientific procedures which involve the release of novel substances (e.g. genetic modification, nanotechnology); regulators and politicians (who represent the public) may be unduly subject to embedded values of expert scientific committees tasked with providing 'best scientific advice'. The oppositional format at least ensures that if there exists some possible problem associated with proposed developments which may carry risk, all the arguments are heard and reported. Then if it seems there may exist a potential for significant risk associated with such a process; politicians may exercise judgement and deploy policies based on the precautionary principle. In a court of law, if there is any doubt about the guilt of a defendant, he or she must be found not guilty. The burden of proof associated with current radiation protection philosophy seems biased in favour of the maintenance of the nuclear industry. In other words, unless critics prove conclusively that there exists significant risk attached to the operation of nuclear facilities, then releases (and the production of waste) from those facilities will continue. Some members of CERRIE believe that in the case of low level internal radiation exposure, significant harm to individuals, communities, and populations may well have already occurred as a result of those releases.

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