The AREVA Midwest Uranium Mining Project, Saskachewan, Canada. Public health and ethical implications

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Section 1: Introduction and outline argument.

I have been asked to examine the question of the consequences of permitting the development of an open case uranium mine at the AREVA site at McLean Lake, Saskatchewan, Canada, termed the 'Midwest Project'. There has been very little time to study the paperwork associated with the project, nevertheless, I will endeavour to cover the main issues of relevance and I will make some points which should be taken extremely seriously by those who are being asked to permit this proposed operation.

I shall begin my making my main point. This project cannot go ahead because new science shows that the basis on which it is environmentally acceptable is false. The whole ethical basis of this project, and indeed uranium mining, refining and dispersion has been overturned by discoveries in science made in the last ten years. This is critical. As a consequence of research into the health effects of Depleted Uranium weapons, first employed in 1991 in Iraq, there has been a new focus on the biological effects of uranium exposure. Scientists have examined the interaction of uranium with biological systems in the laboratory through cell culture experiments and through physico-chemical investigations of uranium oxide particles and uranyl salts. Epidemiologists have conducted surveys of those exposed to Depleted Uranium and of Uranium workers. Gulf war and Balkan war veterans, exposed to uranium particles have been found to exhibit a bewildering range of genotoxic and other effects. Areas where uranium weapons have been used, Iraq, Afghanistan, the Balkans, Kosovo, have shown consequent effects in civilians, cancer, leukemia, lymphoma, hereditary malformations out of all proportion to the intrinsic radioactivity of uranium. Those exposed, whether as miners or as Gulf War veterans, have shown objective evidence of serious genotoxic damage though chromosome aberration analysis. The levels of aberration are out of all proportion to the doses calculated by the conventional risk model, that of the International Commission ion Radiological Protection. And again and again, the many examples of evidence of anomalous response to uranium exposures have been countered by the application of this model by governments and those agencies whose use of this model has continued to support the operation of the nuclear industry, the manufacture and testing of nuclear bombs and, most recently, the employment of uranium weapons. These agencies and governments that employ their erroneous risk models ignore, indeed do not even cite or discuss the massive evidence that their model is worthless when applied to internal exposures to elements that bind to DNA. This is an open scandal. Indeed, the senior advisor to the World Health Organisation (WHO) on radiation and health, Dr Keith Baverstock, recently resigned on the issue of the health effects of uranium and how they were being ignored (Baverstock 2005, APPENDIX E). WHO, which has been constrained by an agreement it signed with the International Atomic Energy Agency (IAEA) in 1959, has made little effort to properly examine the issues of radiation and health, and their documents, written at desks, continue to support the ICRP average dose approach, and to entirely ignore the massive evidence of harm from exposure to internal radionuclides that has increasingly emerged since the post-war atmospheric testing.

It may be asked, what does the general matter of internal fission-product exposure this have to do with uranium mining? It does not take any thought to see that uranium mining is the start of the process that leads to uranium fission, in reactors, in bombs and the dispersion throughout the planetary biosphere of the products: Plutonium-239,

Strontium-90, Caesium-137 and so forth. Those who permit the removal of this dangerous substance from its safe place locked up in the earth are morally culpable. They are arguably at the origin of the cause of tens of millions of deaths (ECRR2003). But it is not only the fission products that cause the genetic and genomic damage that leads to the degredation of human and animal and plant health all over the planet. It is Uranium itself, the element with the highest atomic number of any naturally occurring element, that has an ability to focus external radiation directly into the DNA through photoelectron amplification. And it is this effect, a consequence of basic physical processes which has been discovered recently (or rather rediscovered) that should force any responsible authority to refuse to permit the development of a project which will increase the amount of refined uranium in the world, and expose members of the public in Canada, and the rest of the world, to increased levels of this very dangerous substance.

The Environmental Impact Report of AREVA is a monstrous confidence trick. AREVA, a French company, are the largest builders of nuclear power stations in the world. The Environmental Impact Report fails to mention the fact that the proposed operation is one in which began with this company finding an enormous quantity of what is arguably the most long lived genotoxic substance on earth, safely buried under a lake. They propose digging it out, purifying it and then spreading it all over the planet together with its dangerous radioactive offspring: making it available for millions of humans and other creatures to ingest, inhale and then die of cancer and a whole spectrum of other illnesses. None of this is mentioned: instead we hear about the wolf and the snowshoe hare. And let us not be gulled by the arguments about global warming and sustainability. The amounts of carbon dioxide produced by the mining and purifying activities are not calculated or modeled. They will be very great. All this activity will occur in Canada, which will be putting this carbon dioxide into the world atmosphere to make uranium which will be exported. For money.

I will lay out my argument in the following order:

Section 3. Uranium exhibits serious radiogical genotoxic effects through its affinity for DNA, for nervous tissue and because of its high atomic number (Z = 92) which makes it preferentially absorb natural background gamma radiation and release that energy into the DNA as photoelectrons. The scientific basis for this will be presented. This aspect of its radiological behaviour is entirely absent from the conventional risk model which underpins the Environmental Impact conclusions of the AREVA Midwest Project. The consequence of the theoretical and experimental evidence of the uranium photoelectron amplification is seen in the considerable peer-reviewed published and grey literature evidence that uranium exposure causes genotoxic and neurological effect in populations which have been exposed. Evidence of this will also be reviewed and discussed
Other aspects of internal exposure to uranium will be reviewed as part of a general argument relating to the conventional risk models and their applicability to internal exposure both to uranium itself and to fission products. The risk model of the European Committee on Radiation Risk (ECRR) will be compared with the current models of the International Commission on Radiological Protection (ICRP) and it will be shown that the current risk models are seriously in error for internal radiation. The case in childhood leukemia near nuclear sites will be presented as an example.

Section 4. The AREVA Environmental Impact Statement will be reviewed, taking into consideration the forgoing arguments. The open cast nature of the proposed mine will be addressed. I have conducted research, which I will present, that shows that uranium dust can move over very large distances. It will be shown that air modeling will predict the movement of uranium dust from an open cast uranium mine to parts of Canada with large population densities (e.g. Winnipeg, Manitoba) and this will therefore represent an unacceptable hazard.

Section 5. Ethical issues will be briefly considered from the viewpoint of the ethical framework presented by the scientific committee of the ECRR in their 2003 report. These should have been, but were not included in the EIS case report.

In order to make the arguments readable and concise, I will rely to a large extent on appendices to contain the main bulk of the information.

Section 2
My expertise

2.1. I will begin by briefly outlining my expertise. I have a First Class Honours degree in Physical Chemistry from the University of London and also hold a Doctorate in Chemical Physics. I was elected to the Royal Society for Chemistry in 1974 and I am presently a Member of the International Society for Environmental Epidemiology. I am a scientific reviewer for *The Lancet* and *The European Journal of Cancer, The Journal of Paediatric Radiology, The European Journal of Biology and Bioelectromagnetics* and *Science and Public Policy.*

I have studied the health effects of low dose radiation for more than 15 years both at the fundamental cell biology level and as a radiation epidemiologist. I have been a member of two UK government committees on this issue (Committee Examining Radiation Risks for Internal Emitters, CERRIE, and the Ministry of Defence Depleted Uranium Oversight Board, DUOB). I also have officially advised UK government committees e.g. (The Committee on Radioactive Waste Management CORWM), a US Congressional Committee on Security and Veterans Affairs, The Royal Society, and the European Parliament. I am an official expert witness for the Canadian Parliament on the health effects of uranium. I am a fellow of the University of Liverpool in the Faculty of Medicine and I am supervising PhD students at that University, at the University of Ulster in Ireland and at the German Federal Agricultural Laboratories in Brunswick near Hanover. I am Scientific Secretary of the European Committee on Radiation Risk (ECRR) based in Brussels (*Comite Europeen Sur Le Risque de l'Irradiation (CERI)*) and senior editor of its report *ECRR2003 Recommendations of the European Committee on Radiation Risk: The Health Effects of Ionising Radiation Exposure at Low Doses for Radiation Protection Purposes.* This report has now been translated into French, Russian, Japanese and Spanish and has been used for radiation protection purpose scoping by many organisations including most recently (2006) the UK Committee on Radioactive Waste Management (CORWM). I was invited recently (2007) by the nuclear industry in the UK (CIRIA) to provide advice for best practice in the remediation of contaminated

land, based on the ECRR risk model. I act as a consultant on radiation and health to the Green/EFA group in the European Parliament. I am a member of the Ukraine Association *Physicians of Chernobyl.*

My current cv is attached as Appendix C.

2.2. My particular area of expertise is the health effects of internally deposited radionuclides. I have made fundamental contributions to the science of radiation and health in this area and have published many articles and reports on this issue. My researches have led me to the conclusion that the health consequences of exposure to internally deposited radionuclides cannot be either scientifically or empirically assessed using the averaging methods currently employed by risk agencies (ICRP, NCRP) based on the Japanese A-Bomb studies and other external high dose exposures. The radioactive element dose coefficients published by the International Commission of Radiological Protection and employed in calculations made by these organisations are unsound since they depend on inappropriate averaging of energy in tissue, as I shall elaborate in Section 3. This is actually common sense; and it is increasingly seen to be so by many official radiation risk agencies and committees (e.g. IRSN 2005, CERRIE 2004a, CERRIE 2004b), yet the historic weight of the conventional approach to radiation risk (with whole organisational and bureaucratic structures committed to the simplistic historic approaches) has prevented any change in policy in this area. Such an official acceptance of the scientific illegitimacy of the current radiation risk model for internal radiation exposures would have far reaching and financially costly policy implications.

The large errors involved in employing averaging dose to tissues are most relevant when considering effects from decays of radionuclides in small tissue volumes. For such situations, the concept of absorbed dose, and the various calculations and models employed by conventional health physics, need to seen as, and have been widely accepted as being, strictly invalid.

2.3. In order to understand the elements of the arguments which I will deploy in this matter, it is necessary to know something of the science of radiobiology and how radiation risk has been developed historically. The science of radiation risk is currently in a state of flux, mainly as a result of new discoveries in radiation biology made in the last ten years. In order to not disturb the flow of the argument, I have given the historical background to the current radiation risk model and the arguments relating to it in Appendix A, in the hope that it may prove useful to those without knowledge of this field. The health effects of exposure to uranium pivots on its genotoxic effects and I will examine this aspect in sections 1 and 2. The mismatch between external and internal radiation exposure models is most serious when dealing with radioactive substances which have affinity for and bind chemically to DNA (Busby 1995, Busby 2002, ECRR2003, CERRIE 2004). Interestingly, the most common radioactive element which has affinity for DNA is the naturally occurring element uranium. The health effects of uranium exposure have become the subject of considerable research in the last ten years, largely as a consequence of its use on the battlefield in Iraq and elsewhere. There is now a significant collection of peer-reviewed work showing uranium exposure to be extremely hazardous and to cause anomalous genotoxic effects i.e. out of proportion to

the element's innate radioactivity. I have myself conducted research on uranium genotoxicity (Busby 2005, Busby and Schnug 2007, Elsaesser et al 2007) and have been a member of two UK government committees deliberating the issue. Of course, the uranium contaminating the battlefields of Iraq is only *natural* in the sense that it was not recently produced by fission on earth. It is strictly what is known as TENORM (Technologically Enhanced Naturally Occurring Radioactive Material). Nor is uranium that is mined and removed from the rocks in which it was (relatively safely) locked, strictly natural, since it is now *out there* and available for inhalation and ingestion. Uranium is mined, purified and then enriched in the proportion of the fissile isotope U-235. This enriched uranium goes to the nuclear industry and military as a feedstock for nuclear energy and bombs. The depleted uranium, which is strictly waste material, is then also used for various purposes including by the military as penetrators for armour.

Section 3
Radiobiology: uranium exposure and genetic damage

3.1 Chemistry Physics and Biology

Uranium is a fairly common element on earth, but until the discovery of its radioactivity and its usefulness and interest in the 20th century, it remained locked in the earth's crust in mineral deposits. In Section 4 I will deal with the matter of its dispersion through the proposed uranium mining, but in this short section I will examine its biological effects. These have been the subject of considerable research and scientific argument in the last ten years as a result of the use of uranium in weapons. Uranium weapons were first used extensively in Gulf War 1 in 1991 and later in the Balkans, Kosovo, Afghanistan and then in 2003, Gulf War 2. These conflicts were followed in all the areas where uranium had been used, by increases in a spectrum of illnesses that could be described as radiological, that is, genotoxic. Thus, in Iraq, there were large increases in miscarriages, birth defects, childhood cancers and leukemias, and adult cancer. Gulf War veterans from UK and US armies complained of 'Gulf War Syndrome' a spectrum of conditions that included cancer, leukaemia and lymphoma. There is no question that the personnel were exposed to uranium which remained in the areas and became widely locally dispersed. I myself visited Iraq and Kosovo and measured it in the field and in samples which I brought back. So have others. In Kosovo, UN peacekeepers suffered from excess leukaemia and lymphoma; the only serious attempt to examine these illnesses was carried out by the Italian government and the results of their epidemiological studies confirmed a significant cancer excess in the returning UN peacekeepers. Studies of the Gulf War 1 and Gulf War 2 veterans showed uranium in their urine (Busby, 2006, DUOB). Studies of Iraqi, Kosovan and Afghanistan civilians showed elevated uranium levels in urine (see Busby 2004, see Priest, Durakovic). Cytological studies of UK Gulf War veterans showed high levels of chromosome damage, consistent with exposures of more than 200mSv, roughly 100 times natural background (Schroeder et al 2003, Busby 2003, Schmitz Feuerhake et al 1997). Conventional dosimetry of uranium, the physics-based approach, could not explain this. The most that could be calculated from the levels of uranium exposure were a few microSieverts. Similar levels of chromosome aberrations (a

measure of genetic damage) were also reported in uranium miners from Namibia (Zaire et al 1997) who also were not radiologically exposed to more than a few microsieverts, conventionally modelled. Much of this discussion I have taken from my presentations to the Royal Society, The UK Ministry of Defence Depleted Uranium Oversight Board and the Hamburg World Conference on Depleted Uranium. I attach this as Appendix D.

The military and NATO governments disputed the arguments that uranium was a cause of the illnesses or the chromosome damage. They based their arguments on physics. The conventional position, that taken by the military and UK and US governments was presented well in the 2001 report of the Royal Society (RS 2001). The Royal Society Committee on Depleted Uranium was set up to examine this issue of uranium and health. However, although it was presented with significant evidence that uranium represented a radiobiological anomaly, including evidence that I was invited twice to present, the RS final report remained faithful to the conventional risk model. Since 2001, much more evidence has become available and much has been published in peer-review literature. All the evidence shows uranium to be a serious radiological or chemical hazard or a mixture of the two, one that cannot be modelled using the conventional radiation risk model. The health effects of uranium are at the centre of another wider debate which also has been occurring at the same time: whether it is valid to use the quantity 'absorbed dose' to predict health effects of exposure to internal radionuclides. To examine the problem of uranium we need first to understand how the conventional physics-based dosimetry deals with it. This is the dosimetry employed by AREVA and the Canadian radiological risk agency when calculating and subsequently considering doses to people working at the mine or living nearby or downwind.

3.2 External and internal radiation.

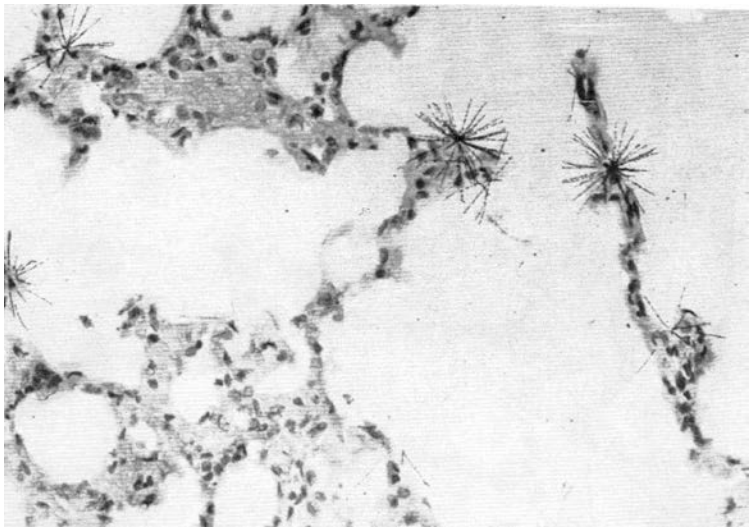
In order to understand the nature of the argument about internal radiation and health it is first necessary to review some basic principles and examine some of the assumptions at the base of radiation risk. These arguments are elaborated in the CERRIE minority report, the CERRIE majority report and in the early chapters of the ECRR2003 report. A more accessible explanation of the basic science is given in my book *Wings of Death 1995* although that book does not consider the new evidence of uranium photoelectrons presented below. Ionising radiation acts through the damage to cellular genetic materials, the genes on the DNA, killing some cells but causing fixed genetic mutation in others, including mutations that signal to descendants a genomic instability message to increase their rate of incorporated error. These genetic and genomic mutations are now known to be the main initiation point in the development of cancer and leukemia and also the origin of heritable damage and increases in many illnesses that were not originally thought to be radiation related. It is the progression of the cellular mutation and the acquisition of further mutations over the lifespan of the cell or its descendants (in the same individual or in the case of germ cells in offspring) that leads eventually to the clinical expression of the cancer or genetics disease. The damage to the DNA is caused either by ionisation of DNA materials themselves directly, or more likely indirectly by the interaction of the radiation track (which is the track of a charged particle, an electron or an alpha particle) with solvent water or other molecules to form 'hot' ionic species which are sufficiently reactive to attack the DNA bases. To a first approximation, it might be argued that over a

certain range of dose, the effect, or likelihood of mutation, is a linear function of the amount of energy absorbed. That is because this energy goes to break bonds and produce ions, and twice the energy produces twice the ions and therefore twice the probability of mutation. But the primary cause of mutation is the *reactive ion* and so it is the *concentration of reactive ions in the cell* which represents the most accurate measure of mutagenic efficiency (although there are other considerations as we shall see). The assumptions that underpin the whole of radiation protection are based on the ideas that the dose and the response are linearly correlated. Thus, if we double the dose, we double the effect. We must note this carefully at this point since it is the basis of the present system of radiation risk assessment, and specifically the basis of the calculation made using the model of the ICRP and all predictions that follow from this approach.

But it is manifestly and philosophically wrong to employ such a model for internal irradiation. This is because the quality used to measure radiation, Absorbed Dose (in rads or rems, Grays or Sieverts) represents the average energy absorbed in unit mass, in the case of Grays, Joules per Kilogram. Such a quantity assumes at the outset that the energy density is the same in all the cells of the tissue irradiated. Whilst this is a valid assumption for external irradiation as in the case of the studies used to determine cancer and leukemia risk (particularly the major study, that of the Japanese A-Bomb survivors) it is manifestly untrue for modeling risk in individuals who have internal irradiation. The reason is that in many internal irradiation regimes, averaging is not appropriate.

Radioactive particles which emit short range radiation like alpha and beta radiation cause high energy density (ionisation) in local tissue (a few millimetres away) but no irradiation elsewhere. Thus cells near to these particles receive large either fatal or mutagenic doses. To illustrate this I show in Fig 3.1 a photomicrograph of decay tracks from a few radioactive particles in rat lung.
This phenomenon is known as an alpha star: the tracks are alpha particle ionization tracks such as those produced from uranium and radium dust particles.

Fig 3.1 Alpha star photomicrograph showing radiation tracks emanating from hot particle in rat lung; track length has the distance of about five cells.



In the case of uranium, it is the anisotropy of ionization that it the cause of anomalous energy deposition into the DNA itself, since the uranium binds to the DNA preferentially (Busby 2005, Busby and Schnug 2007). Averaging the energy into large tissue masses in whole body or in organs, as the conventional model does, dilutes the ionisation density and makes it seem as if the whole body doses are very low, perhaps well below natural background doses. This is the 'air dose' to the public or workers; radioactivity in the air is inhaled and then translocated through the tracheobronchial lymphatics to the lymphatic system from whence it reaches the bloodstream and every organ in the body. But since cancer always starts in a single cell (as we know from mosaic studies of tumours) it is the cell dose that is important, not the overall averaged tissue or whole body dose. The use of external doses to calculate cancer risk (as the ICRP do) is like comparing warming oneself by the fire with eating a hot coal. This argument has now been accepted at the highest level, although little has been done to incorporate it into risk management. It is a major plank of the ECRR deliberations and now in the mainstream of argument in the radiation risk community. Chapters 5 and 6 of ECRR 2003 and pp 48 to 56 of the CERRIE Minority Report discuss the concept of Dose, used by the ICRP model as a measure of radiation exposure, in dealing with health effects. In addition, the matter is reviewed by the CERRIE Majority Report (2004) which agrees that (p13 para 11) *There are important concerns with respect to 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.* (my underline)

This is quoted from an official report of a UK government committee. The point is made regularly elsewhere in the same report, (e.g. para 60 p27) and the Majority Report concludes that there is a conceptual uncertainty associated with the use of absorbed dose of a factor of 10-fold. The Minority CERRIE Report argues that this figure is more like 100-fold to 1000-fold for very low doses and certain types of exposure and

advances proofs of this (see below). In addition, recently, the French official radiation risk agency, *Institut de Radioprotection et de Surete Nucliare (IRSN*), agree that the ICRP dose averaging approach is insecure. In a report published in 2005 they point out that the questions raised by the ECRR2003 report relating to the question of internal doses are valid. The IRSN committee of 15 senior scientists state that these are *fundamental questions with regard to radioprotection* and (p6) that *heterogeneous distribution of radionuclides, the validity of weighting factors for calculating internal doses, the impact of the radionuclide speciation on their behaviour and their chemical toxicity* make it clear that the ICRP approach for certain internal radionuclides is *strictly invalid.* IRSN state that *since the ICRP60 publication, improvements in radiobiology and radiopathology, or even general biology finally might impair [*falsify*] the radiation cell and tissue response model applied to justify radioprotection recommendations.*

[IRSN 2005]

ICRP itself was under pressure on this issue by 2005 and conceded in its draft report on risk**:**

(50) For radiations emitted by radionuclides residing within the organ or tissue, so-called internal emitters, the absorbed dose distribution in the organ depends on the penetration and range of the radiations and the homogeneity of the activity distribution within the organs or tissues. The absorbed dose distribution for radionuclides emitting alpha particles, soft beta particles, low-energy photons, and Auger electrons may be highly heterogeneous. This heterogeneity is especially significant if radionuclides emitting low -range radiation are deposited in particular parts of organs or tissues, e.g. plutonium on bone surface or radon daughters in bronchial mucosa and epithelia. In such situations the organ-averaged absorbed dose may not be a good dose quantity for estimating the stochastic damage. The applicability of the concept of average organ dose and effective dose may, therefore, need to be examined critically in such cases and sometimes empirical and pragmatic procedures must be applied.

But ICRP did nothing to change any of the dose coefficients for isotopes that caused such exposures or to apply such *empirical and pragmatic procedures.* The final 2007 version of the ICRP report had removed this dangerous paragraph. As an illustration of the importance of such a process, I will carry out a pragmatic calculation of dose to the foetus.

3.3 The dose to the foetus

As an illustration of the problem I will briefly examine the absorbed dose to the foetus following exposure to an atom of uranium bound to DNA in the cell. I will merely examine the track energy as the energy involved in generating the dose. The mathematics is simple. Absorbed Dose is represented as energy per unit mass: one Gray equals one Joule per Kilogram. Uranium is an alpha emitter. The alpha decay energy of U-238 is 4.2 million electron volts (MeV). The amount of energy required to create an ion is about 35eV so the nuclear disintegration if U-238 atom produces about 120,000 ions. They are deposited along the track of the alpha particle which extends to about four cell diameters. If we take these cells as the mass into which the energy is deposited, this mass is roughly 5×10^{-13} kg per cell. The decay particle energy of 4.2MeV is 6.7×10^{-13} Joules, and so the energy deposited in each cell is one quarter of this 1.68×10^{-13} Joules. The dose in Grays

is thus 0.322Gy. Now ICRP (and conventional risk Models) have agreed to some extent that it is ionisation density and not absorbed dose that drives mutation. They needed to do this because it was clear from cell killing experiments that highly ionizing alpha particles were 20 times more efficient at cell killing, dose for dose, that gamma rays or X-rays, than external radiation exposure. They have not extended this idea logically to other areas where local ionization is high (as in the present case). Their approach in the case of alpha particles is to define a new unit, one which is not based on physics, but on a mixture of politics and cell culture experiment. This is the unit Sievert, or rem, radiation-equivalent-man and the quantity is termed 'dose equivalent'. It is not a rational objective physics based unit, and it is obtained, for alpha emitters, by multiplying the absorbed dose by 20. Thus the dose to a single cell, from a uranium decay, is now 6.44 Sieverts (644rem). So we can see immediately the error in trying to describe internal exposure using external risk models. And this is most important when it comes to looking at the foetus, which starts as one cell and rapidly grows. In the first few weeks, the foetus grows by continuous cell division and so its mass increases exponentially. If the replication rate is every eight hours, then by the end of a week, the foetal mass is a few milligrams. Development mass with gestation period is known but for the purposes of my argument here I can use a theoretical approach to demonstrate the dose equivalent in the foetus with days gestation from the single uranium atom decay. I assume that the cell replication rate in the first week is every 8 hours. Then the total embryo mass can be written in terms of days growth as 2^{3d+1}m where m is the cell mass. Diluting the decay energy into this exponentially increasing mass results in dose equivalents greater than 1mSv (100mrem) in the first five days from a single uranium decay.

The foetus is highly radiosensitive in any case, since the cell replication rate is almost continuous. Cells are always engaged in cycling and replicating and unlike in adults are rarely in the less mutation-sensitive G(0) or quiescent phase. From the work of Alice Stewart on foetal X-rays, we know for external irradiation, where the doses can be averaged over the whole foetal mass, that this is so. The risk factor for childhood cancer following obstetric X-raying is now accepted to be 50 per Sievert (compared with 0.25 per Sievert for adults) i.e.400 times. And for foetal exposure to Cs-137 from Chernobyl in Europe we can show that there is an additional risk factor error of upwards of 400-fold for infant leukemia, another condition that begins at an early stage in the womb (Busby and Scott Cato 2000, CERRIE 2004, CERRIE 2004b). Since the Chernobyl infants represents unequivocal proof of the failure of the ICRP risk model for internal radiation, I will outline the findings here.

The argument has been published (Busby and Scott Cato 2000). This was a simple and brief analysis of the increase in infant leukaemia in five different countries in Europe in those children who were in the womb at the time of the fallout. The countries were Wales, Scotland, Greece, Germany and Belarus. The increases were statistically significant and could not have occurred randomly since the calculation for all the countries combined makes a probability of 1 in one thousand million that these were collectively a chance observation. Second, since the group being observed was the *in utero* cohort exposed only to Chernobyl fallout it was an effect of Chernobyl fallout. They were reported in separate papers in the peer review literature by four separate groups of researchers so it was not a biased account by one group. The doses (based on ICRP considerations) had been well described and measured. The only known cause of

child leukaemia is ionising radiation. The differences in the levels of leukaemia rates in the exposed cohort and the rate predicted by the ICRP model is greater than 100-fold but varies inversely with the dose. The CERRIE Majority Report conceded this p88 Table 4A6 where it gives the central estimate of error in the ICRP model for Great Britain as 200X, for Greece as 160X and for Germany as 96X. In the paper I published in 2000 with Molly Scott Cato I calculated for Wales and Scotland the effects was greater than 100X and probably about 300X. This is the exact error in ICRP required to explain the childhood leukaemia cluster at Sellafield and in children near many other nuclear sites, and also the present cancer epidemic. These large error factors mean that, for foetal irradiation and infant leukaemia at least, there are 100 to 500 times more leukemias for a given dose than ICRP calculates.

The matter of childhood cancer and leukaemia near nuclear sites has been consistently dismissed as a causal effect on the basis of the conventional risk models. It has has recently been re-opened following a major German government funded study of all nuclear sites in Germany between 1980 and 2003 which shows a statistically significant excess of cancer and leukaemia in children 0-5 living within 5km of the sites Spix et al 2008). These findings support the contention that the ICRP model for such exposures are in error by very large amounts. It should not be forgotten, that the basis for the operation of such plants is the mining of uranium, and indeed, that these plants release uranium as well as its fission products to the local environment.

3.4 The puzzling genotoxicity of Uranium

As I have pointed out, following renewed interest in uranium toxicity generated by the military use of uranium weapons, it has been found that the element exhibits genomic and other harmful effects not predicted by its radioactivity (e.g. Abu Quare and Abou Donia 2002, Craft *et al* 2004, IRSN 2005, Bertell, 2006). This has resulted in two schools of thought: those based on the conventional radiobiological risk assessments (e.g. Royal Society 2001, Wakeford 2001) and those pointing out that there are real genomic effects which cannot be explained or predicted (Baverstock 2005, Bertell 2006). There certainly seem to be experiments which show anomalous genomic or genetic effects (including Bosque *et al* 1993, Miller *et al* 1998-2005, Coryell and Stearns 2006,) but these are usually interpreted as implying some unelaborated 'heavy metal' effect for uranium. Historically, Uranium has been considered both a radiological and also a 'heavy metal' poison, following Calcium in its distribution within the body, i.e. building up in bone, and with the principle target for toxicity being the lung and the kidney (RS 2001). More recently, it has been shown that Uranium also targets the brain (ENVIRHOM 2006). Perhaps because the element is fairly common (and therefore assuming that natural = safe) the US EPA exposure limits in drinking water are as high as 20mg/l, whilst for inhalation the US NIOSH/OSHA give limits for inhalation of dust of 0.05mg m^{-3} , the US NRC giving 0.2mg m^{-3} (compare the German (BbodSchV) limit of 0.25mg m^{-3}).

Uranium has three common isotopes, U-238, U-235 and U-234. The natural U238/U235 isotopic ratio is about 138: 1. With specific activity of about 14 MBq/kg Uranium has been considered to be a low cancer risk. U-238, the main isotope has specific activity of about 12.4 MBq/kg so a concentration of 20mg/l represents 0.25Bq/l. but even counting the two beta-emitting daughter isotopes which are in equilibrium, the activity is still less than 1Bq/l. Uranium can have fast, medium and slow biokinetic

clearance depending on the form: the slow components can remain in the body for a considerable time. ICRP68 gives dose coefficients for inhalation of Fast Medium and Slow dissolving forms of Uranium at 4.4 E-7; M: 2.6E-6; S: 7.3E-6 Sieverts per Becquerel respectively and for ingestion, the ICRP68 dose coefficients are generally 4.4E-8 and for the insoluble oxide UO2: 7.6E-9. On the basis of these figures, and because of a low transfer coefficient, routine ingestion of water at the high end of the EPA limit should not result in doses greater than some tens of microSieverts. The Royal Society (2001) calculated that that a continuous daily contamination by 1mg will eventually result in a steady-state kidney concentration of 12mg/l. Since this results in microSieverts as conventionally assessed, the Royal Society and World Health Organisation (WHO) dismissed the concerns of the Gulf War veterans who suffered from Gulf War Syndrome.

As I have pointed out, there are, of course, fundamental problems with the IRCP radiological risk methods (see ECRR2003, CERRIE 2004, IRSN 2006): the assumption that absorbed dose (energy per unit mass) is an accurate measure of risk is arguable. The decays from particulate uranium, the type resulting from weapons use, are short range and doses near micron sized particles can be large for local tissue volumes within range of the decay.

Nevertheless it is not only exposures to particles from weapons usage that seem to result in health problems: uranyl ion also exhibits anomalous genotoxic effects at low concentrations causing genomic and genetic damage in cell cultures at concentrations where by Poisson analysis there are no significant alpha emissions (Miller et al 2002). Uranium (and also tungsten) particles cause genetic changes in cell culture elements and cause cancer in laboratory animals (Miller et al 2001). Uranium causes anomalous inflammation in lung, kidney, brain and other living tissue in rats and produces neurological effects in mice (ENVIRHOM 2006). Uranium causes chromosome damage in miners and Gulf War Veterans (Zaire et al 1998, Schroeder et al 2003, Paquet et al, 2005). How is this?

Since the ‘absorbed dose’ due to radioactive decay of uranium is very low in these studies, and in one experiment stochastically absent, these effects are puzzling on basis of conventional radiological risk models. They have been ascribed therefore variously to ‘heavy metal toxicity’ or ‘chemical effects’ or some unelaborated ‘synergy between radiation and chemistry’. The genotoxicity nevertheless exists, whatever its origin, and it can be related not to the absorbed dose, but to the concentration of the uranium in tissue.

3.5 Secondary Photoelectrons

This is the most important element of my argument with regard to uranium mining. I have shown elsewhere (Busby 2003, Busby 2005, Busby and Schnug 2007, Elsaesser et al 2007) that an explanation for the anomalies in uranium effects on living systems involves the idea that contamination by elements of high atomic number Z which have significant affinity for DNA will result in anomalously high absorption of natural background radiation by the DNA and its re-emission as photoelectrons. This represents a kind of focusing of natural background radiation (and any other external gamma or X-rays) into the DNA.

Chromosomal DNA is widely believed to be the target for ionising radiobiological effects (e.g. see BEIR V 1990, ECRR2003, CERRIE 2004a, 2004b). It has been known for some time that Uranium binds strongly to DNA phosphate (DNAP) as uranyl ion UO_2^{++} (Zobel and Beer 1961, Huxley and Zubay 1961, Constaninescu *et al* 1974, Nielsen *et al*, 1992). The affinity constant determined by Nielsen *et al* was of the order of 10^{10} M^{-1} at pH values below 5 with binding of one uranyl ion to every two phosphate groups. This would give half saturation of DNAP at concentrations of uranyl of about 10^{-10} M, which represents a cell concentration of 23ng/litre, at the lower end of urine concentrations that have been reported in those exposed to uranium weapons (see Busby and Hooper in DUOB 2007). Given the increasing concentrations of uranium in the drinking water in Los Angeles (which I will turn to) levels higher than this will certainly exist in those living downwind from SSFL, and will have increased as the uranium contaminated soil at SSFL was disturbed and resuspended into the air. At higher pH's the amount of uranyl significantly increases to two ions per phosphate although the affinity constant decreased due to competition with polynuclear complexation reactions. Nielsen et al showed that the uranyl binding to DNA was greater than that of the powerful bidentate chelating agent citrate. The authors employed their discovery that uranyl ion induces photochemical single strand breakage in the DNA following irradiation with visible light (λ420nm), a photoelectron-produced DNA lesion like those I am drawing attention to here, though at lower photon energy.

It is an interesting and well known fact that the absorption of gamma and X-rays increases rapidly with atomic number. Uranium (Z=92) and Lead (Z = 82) are thus employed for shielding purposes. The relationship is often assumed to approximate a fourth power one, though the exponent varies in the range 4.0 to 4.8 depending on gamma energy and element (Krane 1988). I can therefore compare the absorption of external photon radiation by uranium with that of calcium ions, those displaced by the uranyl from the phosphate DNA backbone. For water (Z_{eff} = 3.33) the fourth power ratio is greater than 500,000; for DNAP (Z_{eff} = 5.5) it is greater than 50,000 but for Ca^{++} and UO_2^{++} the fourth power ratio is about 450. Thus Uranium on DNA absorbs 450 times the background gamma and photon ionizing radiation than Calcium. But of course, Ca (Z=20) already absorbs 1000 times more gamma radiation than water and some 154 times more than the DNAP complex. It would thus seem that the Calcium ion associated with the DNAP is the dominant absorber in the genetic material of the cell. This effect is entirely absent from any microdosimetric assessment of risk. I compare Z^4 enhancements of absorption for some tissue components in Table 3.1 where I have normalised the ratio to water. Calcium, Strontium and Barium (included) all bind to DNA but their toxicity increases sharply in the sequence $Ba^{++}>Sr^{++}>Ca^{++}$ as we would expect from these considerations. Also shown are the reported photoelectric cross sections at 100-150keV (NIST Standard Reference Database 8: XCOM).

The amount of energy deposited in different constituents of the DNA in a cell per Gray of radiation absorption has been calculated (Ward *et al* 1988, BEIR V 1990). The cell was assumed to contain 6pg of DNA of which 1.2pg was phosphate. I reproduce the BEIRV Table1-1 (p14) where these fundamental results are shown, as Table 3.2 The calculation takes no account of the atomic numbers (and hence the gamma absorption) of the DNA constituents: the fourth column, which I added, shows that Ward et al 1988 calculate that the energy deposited per pg is the same whether we are dealing with water

(Z_{eff} =3.3) or phosphate (Z_{eff} = 9.4). If, at a cell concentration of about 10^{-10}M, the phosphate were half saturated with uranyl ions, at a stoichiometry of one UO_2^{++} to two phosphate groups we can easily calculate the mass of uranium on the DNA. It is 0.7pg and this represents about 12% of the DNA in the cell by mass. This soaking up of uranium by DNA was actually reported by Huxley and Zubay in 1961 who observed that purified DNA took up nearly its own dry weight of Uranyl acetate from a 2% fixing solution. They employed uranyl acetate as an electron microscope stain.

Table 3.1 Fourth power of atomic number Z for some materials of interest compared with water; photoelectric cross section barns/atom 100-150keV (NIST: XCOM). For H_2O and DNAP these are weighted means of the atomic composition.

Material	**Z**	**Z^4**	**H_2O = 1**	**Photoelectric cross section***
H_2O	3.33	123	1.0	0.026
DNAP	5.5	915	7.4	0.087
Ca	20	0.15 E+6	1220	5.94
Sr	38	2.1 E+6	17073	95.1
Ba	56	9.8 E+6	79675	450
Au	79	38 E+6	308943	544
U	92	72 E+6	585365	1160

We also know from experiments with Auger emitters bound to DNA (e.g.I-125) that DNA is the target for the effects of ionising radiation (Baverstock and Charlton 1988). BEIR V (1990) state this clearly and tabulate results of calculations showing that the amount of energy deposited by one Gray of radiation in the DNAP of a cell is 36keV, of which 7.3keV were absorbed by the phosphate (see Table 2). This leads to 600 (60eV) ionisation events in the DNAP (BEIRV 1990) per Gray. The total absorption of external gamma radiation by uranium contaminated DNAP will therefore include the enhanced contribution from the uranium on the phosphate which is simply 7.3 x 450 = 3285keV per Gray resulting in an overall enhancement of deposition of energy by a factor of almost 100-fold.

Table3.2 Amount of energy deposited in DNA per cell per Gray according to Table 1-1 of BEIRV 1990 and based on Ward et al 1988 with column showing that BEIRV made no allowance for the photoelectric cross section of the various atoms.

Constituent	**Mass per cell (pg)**	**eV deposited**	***eV per pg***
Deoxyribose	2.3	14000	*6086*
Bases	2.4	14700	*6125*
Phosphate	1.2	7300	*6083*
Bound water	3.1	19000	*6129*
Inner hydration	4.2	25000	*5952*

Thus, for an annual absorbed dose of 1mSv, the DNA of tissue containing quite modest and environmentally common levels of uranium would be 100mSv. For those who are occupationally exposed, the enhancement would probably be greater both through the internal uranium concentration term and also the external gamma radiation term. I believe that it is this overlooked phantom radiotoxicity resulting from photoelectron effects that explains the various anomalous findings referred to earlier. The gamma radiation is absorbed preferentially by the Uranium atoms: the absorption cross section for gamma photons is up to 500,000 times greater than that of water depending on the photon energy. But this does not mean that all the energy from the absorption is deposited in the DNA, since the photoelectrons may have various energies, ranges and track directions.

But it is not only photoelectrons that are the ionizing agent near the DNA. Photo emissions include electrons with a spread of ranges and velocities proportionate to the incident photon energy. But there is also ionization of the uranium atom itself with 'catalytic' local effects. The loss of an electron will ionize the uranium and produce an excited or 'hot' species which may lose energy by abstracting an electron from local hydration water or some other local molecule (see e.g. Gracheva and Korolev 1980). This will lead to at least one production of a different reactive hot radical or ionic species at the Uranium site, and note that the effect is catalytic, that the Uranium is regenerated.

As far as the emitted photoelectrons electrons are concerned, for condensed phase DNAP in dividing cells, they will have a high probability of damaging DNAP along their track only where this track intercepts or lies near the DNAP. To assess the likelihood of DNA interception, that is the production of ion pairs close enough to the DNA for damage to occur, we need to examine the spectrum of ranges and thus energies. The energy dispersion of environmental gamma radiation at any point in tissue is a consequence of many energy splitting processes (Compton effect, pair production, Bremsstrahlung, etc.) with the result that the event number (ionization events, counts) increases rapidly with decreasing photon energy. This means that at the DNA, especially deep within the body of human beings (though not, perhaps to such an extent within small animals like mice and rats) there will be the highest density (counts, events) of photoelectrons of low energy and short path length. It will be these that create the highest number of ionizations close to, or inside the DNA.

This photoelectron effect is real and was first considered for X-rays a long time ago when it was discussed in the peer review literature by Speirs (1947). I 'rediscovered' it in 2003 whilst thinking about the anomalous health effects reported for uranium exposures. That my considerations were accurate was shown when the effect was reported to occur for gold particles by Hainfeld et al 2004 who successfully employed it to enhance cancer radiotherapy and obtained a US patent. I presented its implications to the CERRIE international workshop in Oxford in 2003, to the Depleted Uranium Oversight Board in 2004 and also published it in 2005 (Busby 2005). Later, I presented it in 2007 to an international meeting at FaL, Braunschweig organized by the German Government where it received considerable interest. The peer reviewed version of this presentation, written with Prof Ewald Schnug, Director of the German Federal Agricultural Laboratory, is published in the proceedings and I attach it as Appendix B. I am continuing to research this important issue. Monte Carlo theoretical modeling of the photoelectron enhancement using the CERN (Geneva) FLUKA computer program was carried out by my (joint) PhD student Andreas Elsaesser at the University of Ulster, UK. The program shows the huge enhancement of photoelectron emission by uranium relative to water or elements of lower atomic number. Some results of the models results for water, gold and uranium are given in Fig 2 below (Elsaesser et al 2007).

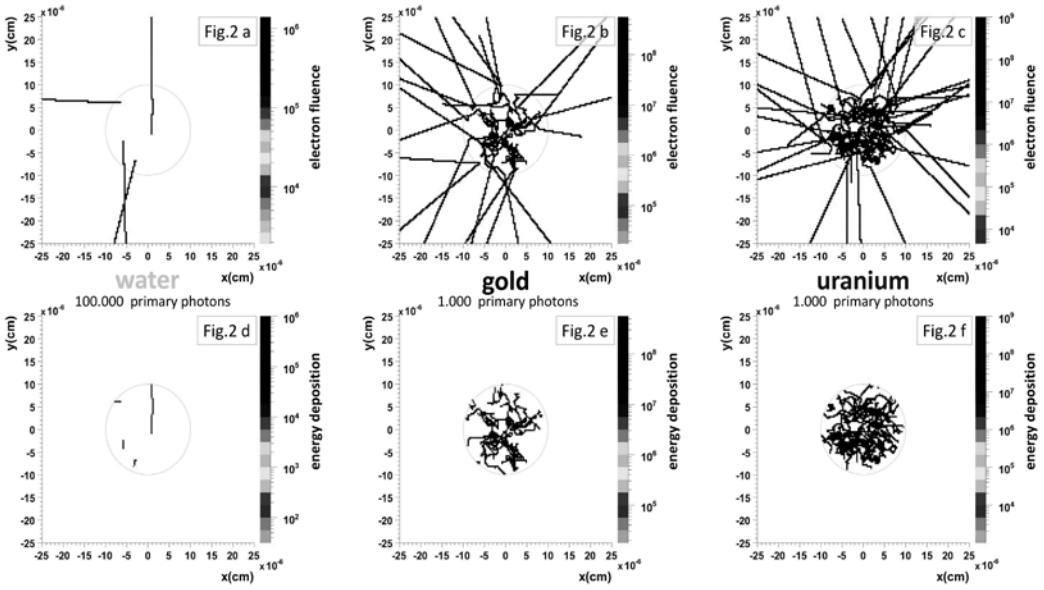


Fig.2: secondary electron production by 100 keV primary photons within the target and escaping electrons overlayed by the target geometry for water (a), gold (b) and uranium (c).Fig.2 (d)-(f) shows the corresponding energy depostion.
Fig.3: ratio of electrons leaving the target material (gold) to incident primary photons (100 keV, 10 keV, 2 keV).
Fig.4: same ratio as Fig.3 but weighted with the perpendicular beam projection area and the target volume.

Summary of Section 3

I have taken space here to explain some fundamental issues in radiobiology and radiation dosimetry, as it is now practiced. The ICRP, whose model is employed by AREVA and others, themselves are aware that their radiation risk model breaks down for anisotropic internal exposures, like those from uranium. As I noted, they stated (2005) for such situations that: *sometimes empirical and pragmatic procedures must be applied.*

The importance of this concession in the present case, as in the case of the Chernobyl infants and perhaps also the other clusters of childhood cancer near nuclear sites (e.g. leukemia near Sellafield and elsewhere), is that it is no longer a defense by those who are contaminating the environment and exposing members of the public that the doses are too low for cancer and other effects to be caused by the radiation. These doses are meaningless. We have to turn to epidemiology for answers to our questions of causality. And classical epidemiological correlations can no longer be countered by arguments from analogies with external radiation doses to Japanese adult A-Bomb survivors linearly extrapolated to near–zero doses. The Japanese A-Bomb survivor doses are only applicable to A-Bomb exposures, and perhaps, if we stretch the point, to other external exposures. They are certainly not generally applicable to internal exposures.

I therefore apply *empirical and pragmatic procedures.* I argue that uranium is to be considered afresh as a substance which is extremely mutagenic. I show that uranium binds to DNA and focuses external natural background into the DNA. I show that this is a real phenomenon which falsifies the entire basis of radiation risk modeling upon which the Environmental Impact Statement for the AREVA project is based.

Section 4: Dispersion of uranium and human exposures. The Environmental Impact of the proposed project

4.1 The AREVA Environmental Impact Report

The health consequences to workers and members of the public from exposure to uranium

I will criticize a number of parts of the AREVA environmental impact statement (EIS) which in my opinion is a normative document; that is to say, it was constructed around the objective rather than attempting to properly address the real issues. It is full of flowery invitations to observe how dedicated the company is to sustainability, but weak on hard facts, and entirely ignores the overall picture, the impact to the biosphere of planet earth. This is to be expected and is normal for reports produced by companies presenting a case to allow them to make money. Unfortunately, there is no proper democratic accountability in these situations. The AREVA report must have involved a huge amount of work and money, but very little investment has been made by society (in Canada) to provide a similar investment in minutely examining the report and asking pertinent question. It is left to activists and members of the public, with few resources, to cobble together what they can at the eleventh hour. In addition, whilst the report must have taken many months and person hours to produce, the response has had to be put together in two weeks.

I will concentrate on three main issues presented in the AREVA EIS report which in my opinion have been either inadequately or erroneously dealt with.
These are:

- The health consequences to workers and members of the public from exposure to uranium.
- The health consequences to biota from exposure to uranium in water air and soil.
- The dispersion of uranium dusts from the proposed workings

4.1.1. Missing human receptors

The relevant parts of the EIS report referring to health risks from workers and members of the public are Sections 8.2, 8.3 and Appendix VII.
In 8.2 we are told that risk to human health was assessed within the framework of an 'integrated ecological approach' as detailed in Appendices VI and VII. The approach is to employ mathematical partition modeling of uranium, radon and daughters (and other toxic elements like arsenic). The end points of these models are contamination of humans through various vectors, drinking water, inhaling air, eating contaminated food, ingesting soil and so forth. The internal contamination of the various human behaviour types, termed *human receptors* are thereby obtained. The chosen human receptors for the AREVA modeling are given in Table 1, (from Table 8.3.1 of the EIS). In Table 1 I have also added a number of obvious missing human receptors. I note also, that the radiological risk model solely has cancer as its endpoint.

Table 1. AREVA model chosen 'human receptors'. Also shown are the obvious missing 'human receptors', the child under 6 years, the fetus, human germ cells and distant population receptors.

Human Receptor	**Description/ location**	**Note**
1a, 1c	*Adult (1a), child over 6 (1c) Wollaston lake resident living in town*	Cancer end point only assessed
2a, 2c	*As above with trapline along Collins Creek*	Cancer end point only assessed
3a, 3c	*Wollaston lake lodge operator located at Hidden Bay*	Cancer end point only assessed
4	*Rabbit lake mine site worker*	Cancer end point only assessed
5	*McLean lake operation JEB mine site worker*	Cancer end point only assessed
6	*Points North worker located on Glove lake*	Cancer end point only assessed
7a, 7c	*Adult (7a or child (7c) Hatchet lake lodge operator located at Hatchet lake*	Cancer end point only assessed
D	As above child under 6	Missing
E	As above fetus	Missing
F	As above germ cell	Missing
H	All exposures to long range uranium laden dust	Missing: See long range dust transfer and NOAA air models

4.1.2 ICRP dose model errors: the ECRR model

The AREVA approach is to employ a *mathematical model* which has not been subjected to any checking of its predictions. It is strictly a deductive approach, and predicts cancer on the basis of linear application of exposure dose health relations obtained from high dose external exposures of Japanese A-Bomb survivors. Since the required end-point in these complex calculations is *human health*, in the application this is modeled as *cancer*. We should note that unless there has been epidemiological examination of these target 'human receptors' the model has not been checked. Where such models have been checked, near nuclear sites, they have found high levels of childhood leukemia, falsifying the model. Internal contamination is converted into an internal effective absorbed dose of radiation using dose conversion factors given in 1996 by the International Commission on Radiological Protection (ICRP72, 1996). For ingestion, these are given in Table 2 below, taken from the EIS. I have also added infant 3 months. Note that there are no dose conversion factors published for the fetus or for germ cells, an alarming oversight given the genetic damage capability of uranium, the affinity it has for DNA and the evidence (including evidence alluded to in the AREVA report for animals experiments) that uranium causes significant heritable damage.

Table 2. ICRP doses from ingested radionuclides employed (*and not considered*) by AREVA.

Radionuclide	f(1) (gut transfer factor assumed)	Coeff; Sv/Bq Adult	Coeff; Sv/Bq Child 6yrs	*Coeff Sv/Bq Infant 3m (Missing receptor)*	**Coeff Sv/Bq Fetus (missing receptor)*
U-238	0.02 *(.04)*	4.5E-8	8.0 E-8	*3.4E-7*	*Not known*
U-234	0.02	4.9E-8	8.8E-8	*3.7E-7*	*Not Known*
Ra-226	0.2	2.8E-7	6.2E-7	*4.7E-6*	*Not Known*
Pb-210	0.2	6.9E-7	2.2E-6	*8.4E-6*	*Not known*
Po-210	0.2	1.2E-6	4.4E-6	*2.6E-5*	*Not Known*

* From the Stewart obstetric X-ray work, which resulted in a cancer risk factor of 50/Sv this factor could be set at about 1000-times the adult coefficient (see text), i.e. for U-238 3.4 E-4 (see text).

The AREVA EIS report gives few details of the calculations involved and does not list the ICRP coefficients for inhalation nor do they discuss the assumptions used in the model or their validity about the removal rates from the respiratory tract. This is a serious oversight since such assumptions strongly influence the cumulative doses for uranium exposures. Again, this is symptomatic of a *paper exercise* aimed at presenting an acceptable case rather than any serious attempt to evaluate the true level of harm to humans. In addition, the health model applied to the doses is that of the ICRP which I have already discussed. This model, besides being unsafe for internal exposures, only has cancer as an endpoint.

The most serious problem is the dose coefficient from U-238, since this, as I have argued, is based upon the simple averaging of all the decay energy from the alpha decays of U-238 into large masses of tissue. It therefore entirely misses:

- the microdosimetric energy distributions of ionizations from an alpha decay (which traverses perhaps four cells)
- the fact that uranium will be bound to DNA as uranyl ion UO2++
- the enhanced fourth power atomic number photoelectron enhancement of external gamma rays

This problem of U-238 has been the subject of studies by the Uranium sub-committee of the European Committee on Radiation Risk, an independent organization of which I am Scientific Secretary (www.euradcom.org). The report ECRR2003 deals at some length with the internal radiation problem, and the Uranium subcommittee have been developing dose coefficients for the element. In 2006, ECRR was asked by the UK Committee on Radioactive Waste Management (CoRWM) to advise it on a scoping exercise it was carrying out for human exposure following deep disposal of nuclear waste. The NIREX model employed by CoRWM was rather similar to the AREVA model; U-238 represented a significant long term risk for future generations owing to its long half life (millions of years). The ECRR approach (see ECRR2003) is to use weighting factors for biophysical and biochemical aspects of individual radionuclide exposures. At the time of

the first report, uranium had not been considered. However, following research into depleted uranium weapons, carried out after 1999, the affinity of uranyl for DNA and also the high absorption and photoelectron enhancement properties of U-238 became apparent. On the basis of theoretical considerations, a preliminary weighting factor of 2500 was applied to U-238 exposures and this was accepted by CoRWM and included in their reports as a maximum risk baseline factor for scoping purposes. Since then, following further research, this weighting factor has been reduced to 1000. Table 3 gives the dose coefficient of the ECRR for exposure to U-238.

Note that the photoelectron effect will not be significant for U-234 since the isotope concentration at which DNA binding occurs will already carry a very high intrinsic radiation dose.

Table 3 ECRR Dose coefficients Sv/Bq for U-238

Radionuclide	Coeff; Sv/Bq Adult	Coeff; Sv/Bq Child 6yrs	*Coeff Sv/Bq Infant 3m (Missing receptor)*	**Coeff Sv/Bq Fetus (missing receptor)*
U-238	4.5E-5	8.0 E-5	*3.4E-4*	*Not known*

* From the Stewart obstetric X-ray work, which resulted in a cancer risk factor of 50/Sv this factor could be set at about 1000-times the adult coefficient (see text), i.e. for U-238 3.4 E-1 (see text).

The result of these increased dose coefficients may be applied to the doses calculated in the models employed by AREVA. Without access to the model and data employed, I can only estimate the effect of increasing the internal U-238 component of the doses. I will conservatively estimate the contribution as 20% of the dust doses. Thus the true exposures to a number of human receptors are given in Table 4. We should note that U-238 is amplifying external gamma into the DNA. If the external gamma is also high (as it is with the workers) then this will impact on the final dose. In general this uranium photoelectron effect will dominate the doses.

Table 4 Some total annual effective doses to different human receptors calculated using ECRR dose coefficients of U-238 exposure and based upon AREVA modeling

Receptors	**Dose ICRP mSv**	***Dose ECRR mSv**
JEB Mill (Table 8.2.1)	2.89 (calc.)	83
Average worker (8.2.3)	3.4 (calc)	183
Grinding/millwright (8.2.3)	5.6 (calc)	305
Child Wollaston Lake 2c incremental (8.3.8)	0.044 (calc)	8.8
JEB Camp worker (8.3.8)	0.116 (calc)	46.4
Adult Woolaston lake resident (8.3.8)	0.006 (calc)	6

* based on 20% of internal radioactive dust ICRP modeled dose

It is clear from Table 4 that the inclusion of ECRR risk model considerations result in doses from the proposed operation above the legal limits to all the human receptors considered. This highlights the critical importance of the U-238 photoelectron enhancements of external background (in this case, often enhanced background). From such doses to workers, we would expect to find objective biomarkers of radiation damage. And indeed, Uranium miners in Namibia and those exposed to Depleted Uranium at moderate levels do show such flags in the form of significant excess of chromosome aberrations (Schroeder et al, 1999, Zaire et al 1997). In addition, we might expect various ill health effects apart from the cancer modeled by the ICRP risk employed by AREVA and there have been many reports of such effects. Craft et al, 2004 reviewed the health effects of exposure to modest environmental and occupational exposures to uranium. Some reported effects are listed in Table 5 below.

It is clear that uranium causes a wide range of serious adverse health effects in humans and in animals apart from cancers. The effects are due to genetic and genomic damage which leads to a wide range of problems including inherited problems. The AREVA EIS report employs the ICRP system of modeling which has cancer as the only end point. This is unacceptable

4.2 Impact upon Biota

I will not dwell on the EIS report on the impact of the proposed project on biota. I will note that the report spends more space examining doses to a bewildering array of what seem to me to be rather unlikely animals, snowshoe hares and wolves than it does on humans. However, I do notice that for many of these animals the exposures are likely to be considerable greater than the exposures to humans, because of the diet of the animals and their proximity to uranium contaminated soil and water. Therefore it should be clear to anyone that the doses to these creatures are likely to be far higher than does to humans. I am not going to engage with the various reference levels cited for exposure limits to animals and fish; they are clearly very high dose rates and arrived at on purely pragmatic bases considering deterministic effects.

Studies made after the Chernobyl accident have shown remarkable effects in animals birds fish and plants at low levels of contamination by radionuclides (see ECRR2006) at both the individual biomarker and population level. In any event, application of the ECRR weighting factors for U-238 would predict that much of the biota in the region around the proposed mine will be at risk from internal exposure to uranium, and on the basis of the genomic damage caused by uranium, there will be species-level effects. Interestingly, the AREVA report does in fact refer briefly to a study which found significant hereditary uranium effects in mice occurring across three generations. In a curious statement about the risk from the levels of uranium involved (3mg/k to dam) it was suggested that the danger limit should be reduced by a factor of 3 (Paternain et al, 1989). I am at a loss to understand the rationale for this decision.

Table 5 Uranium toxicity on various systems according to published studies (from Craft et al 2004 and other sources)

Renal system	Humans : elevated protein excretion, urinary catalase, diuresis Animals: Damage to proximal tubules, glomerular changes In Vitro: no studies
Brain/ CNS	Humans: Decreased performance in neurocognitive tests Animals: Acute cholinergic toxicity. Accumulation in cortex, midbrain and vermis. In vitro: no studies
DNA	Humans: increased cancer, chromosome aberrations, SCE Animals: Increased urine mutagenicity and tumour induction; Heritable genetic damage through three generations of mice In vitro: genotoxic, genomic effects, micronuclei, binuclear cells, cell cycle inhibition, sister chromatid exchanges (SCE), tumorogenic phenotype
Bone/ muscle	Human: No studies Animals: Inhibition of periodontal bone and alveolar wound healing In vitro: no studies
Reproduction	Humans: increase in congenital disease in Gulf veteran children/ Iraq population children. Uranium miners have more first born female children (sex ratio marker for genetic damage). Animals: Moderate to severe focal tubular atrophy; vacuolization of Leydig cells. Heritable genetic damage through three generations of mice
Lungs/ respiratory	Humans: no adverse effects noted Animals: severe nasal congestion and hemorrhage, lung lesions and fibromas, inflammation, odema swelling, lung cancer
Gastrointestinal	Humans: vomiting, diarrhea, albuminurea
Liver	Humans; no effects seen at exposure doses Animals: fatty livers, focal necrosis
Skin	Humans: no assessment available Animals: swollen evacuated epidermal cells, damage to hair follicles and sebaceous glands
Immune system	Humans: chronic fatigue, rashes, ear and eye infections, hair and weight loss, cough, Animals: no studies
Eyes	Humans: No studies Animals: conjunctivitis, irritation, inflammation, edema, ulceration of conjunctival sacs,
Blood	Human: leukemia , lymphoma Animals: decrease in RBC count and haemoglobin concentration
Cardiovascular	Humans: myocarditis resulting from ingestion/ also no effects Animals: no effects reported

4.3 Air models: widespread dispersion on radioactive dust.

The AREVA EIS gives the results of computer air models which it names. The operation of an opencast mine, the trucking of high grade uranium or along a 15km road and all the associated ore operations, milling, grinding, extraction etc will obviously result in a great deal of uranium contaminated dust. The question of the dust in the atmosphere and its inhalation seems to be a paramount one. It is extraordinary that the report does not even have a wind rose enabling independent examination of the dispersion of these dusts, nor are the various concentrations of uranium and other radioisotopes in the atmosphere, as measured, available in the various parts of the existing operation (although I expect these figures will be available).

Stochastic risk modeling using the linear no threshold dose response, the method of the ICRP enables utilization of the concept of collective dose. That is to say that a dose can be defined to a population in terms of the mean individual dose multiplied by the number of individuals exposed. The result is given in person-Sieverts. This enables a rough check on the health outcomes: in the case of the ICRP model, this is always fatal cancer, hereditary defect and nothing else. The ECRR recognized the broad spectrum of harm which occurs following radiation exposure (ECRR2003) and employed factors for birth rate depression, infant mortality, general quality of life and lowering of IQ on the basis of epidemiological observation of large populations exposed to internal fission products. Reproductive risk factors are shown in Table 6 below

Table 6 Risk factors for infant, neonatal, stillbirth and birth rate depression per mSv (ECRR) doses to adults. (ECRR2003 p123)

Effect	Percentage increase in baseline rate per mSv ECRR
Infant mortality	0.05%
Neonatal mortality	0.07%
Stillbirth	0.04%
Birth rate depression	0.05%

The usefulness of the collective dose approach is that it gives the total outcome of any releases and subsequent exposures. Dust inhalation, and the consequent uranium contamination will clearly be higher at the points of generation of the dust, the open cast mine and the various associated plants, but the populations, mainly workers will be relatively small. However, dust can travel for very long distances. Sahara dust travels to the UK routinely, over thousands of miles. In the Gulf War 2, Uranium dust traveled to the UK from Iraq and was collected and measured in air filters around the Atomic Weapons Establishment, AWE Aldermaston (Busby and Morgan 2005). If the uranium laden dust from the AREVA workings traveled to the nearest large centres of population, although the inhalation and cumulative deposition/ resuspension doses might be small, the populations are hundreds of thousands of times larger and so the effects might be significant. In any event this scenario has not been addressed.

From the Sulphur dioxide dry deposition map Fig 2.4.3 of the EIS I note that the predominant wind direction is North West, sending the dust towards the populated parts of Canada and the Great Lakes area of the USA. I employ the NOAA HYSPLIT

computer model (www.noaa.gov) to determine the tracks of dust generated at ground level at the site 58.20N 104.00W, the approximate position of the AREVA operation. Dust was released at 00UTC on 08 Feb 2008 and the computer model result is given in Fig 3 and Fig 4. Fig 5 shows a set of 24 tracks generated on a different day in February 2008 every 6 hours for 6 days. Several of the dust tracks move toward and over Manitoba.

Fig 3. Uranium-laden dust generated at the AREVA site on 00UTC on 08 Feb 2008 moves south east through Winnipeg according to the US NOAA HYSPLIT model.

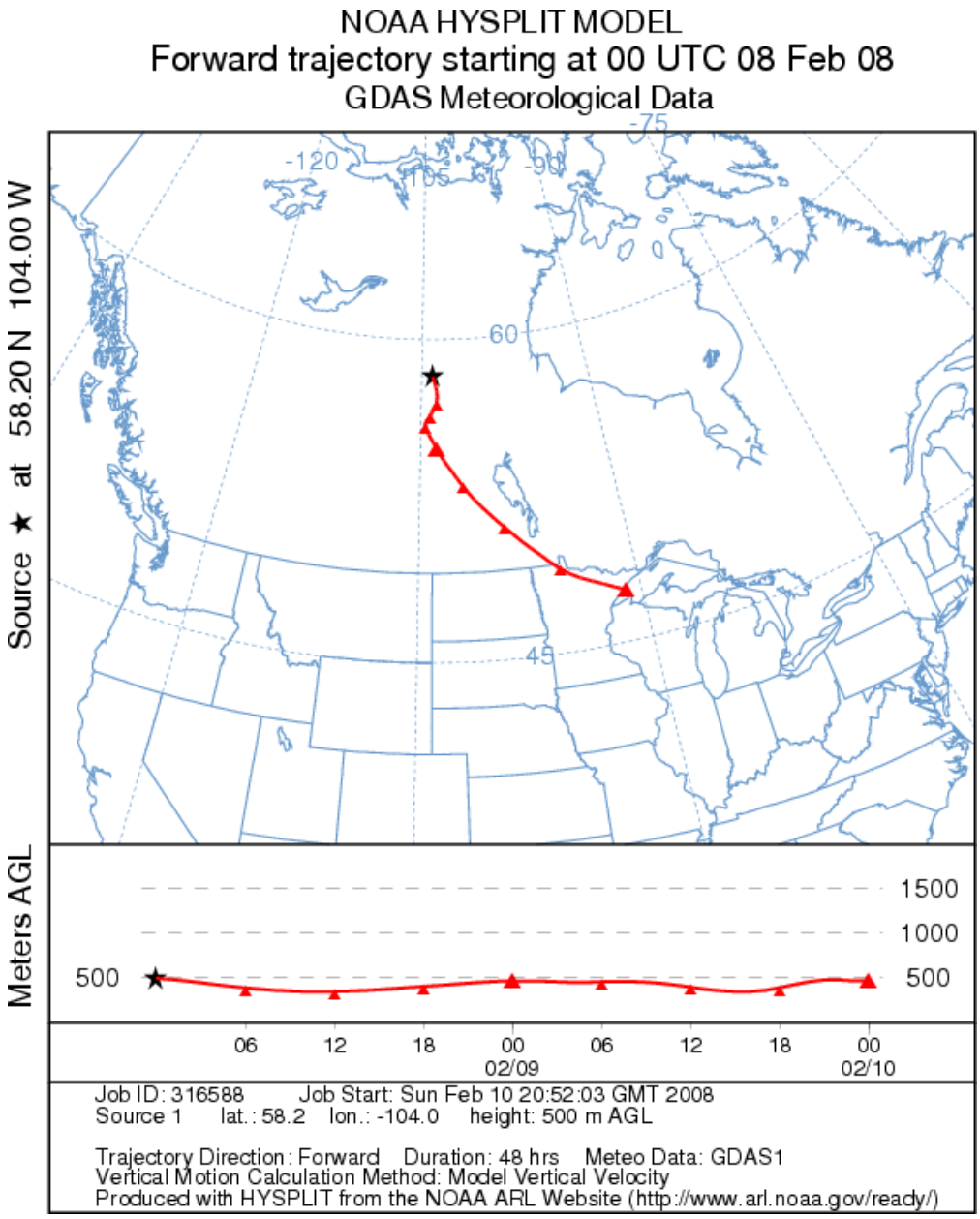


Fig 4 Google Earth satellite version of the plot in Fig 3

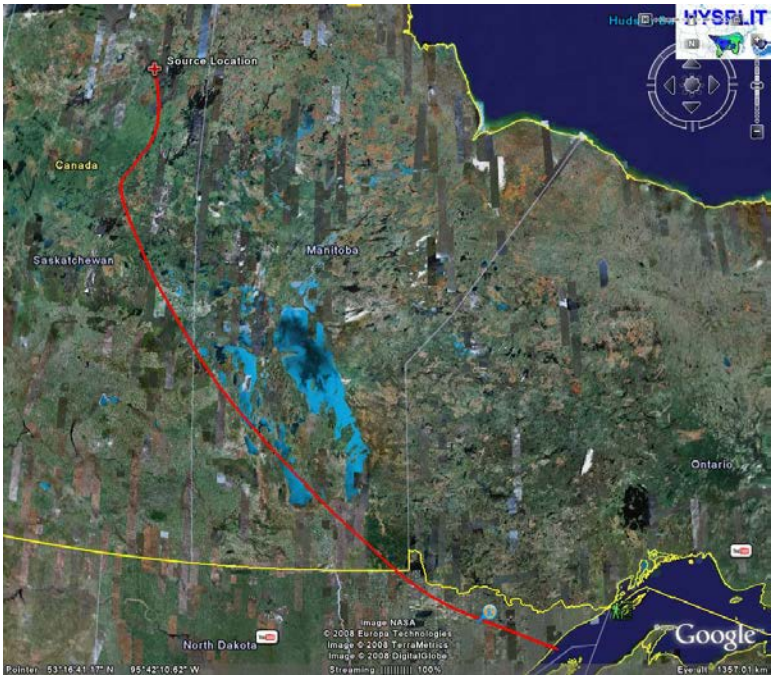
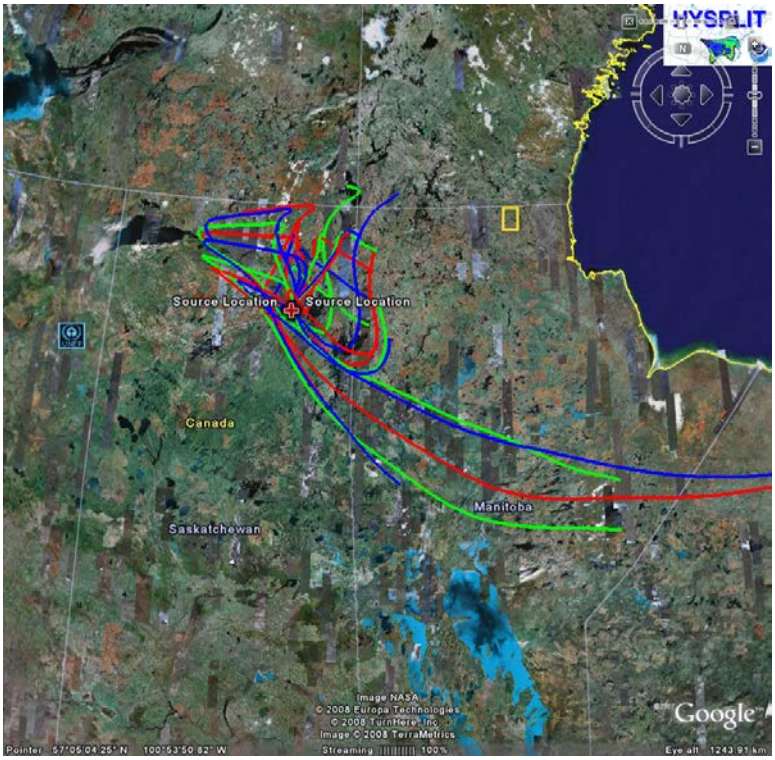


Fig 5 NOAA HYSPLIT models of 6 days of tracks of uranium contaminated air generated at the mine site position in February 2008.



I would therefore be concerned that the EIS report does not address the long range movement of uranium contaminated dust or the exposure through inhalation, nor the cumulative deposition in areas of moderate or high population density at distances from the site greater than a few tens of km, nor the collective dose aspects of the operation.

5. Ethical Issues

The EIS report does not deal with ethical issues relating to the mining of uranium and its subsequent dispersion and use. In my opinion it should, and even if it did not, it should be the business of those politicians assessing the project to consider this matter. Uranium is mined to produce the fuel for nuclear energy and is also the basis of nuclear weapons. Once the fissile U-235 is removed, the waste, depleted uranium is increasingly employed as a weapon, as a penetrator for hard targets, armour and deep penetration of underground bunkers. All these processes result in the contamination of the planet, of humans and of living systems. The contamination is general. The uranium weapons produce particles which travel thousands of miles. Nuclear weapons tests produce uranium and also highly radioactive fission products which travel all over the planet and fall in the rain, contaminating food, and water, and ultimately humans. The nuclear energy fuel cycle produces continuous releases of fission products and the final waste contains huge amounts of radioactive substances often with enormously long half lives, tens of thousands of years.

As the ECRR2003 report points out, the implicit philosophical basis for nuclear energy associated radiation exposures and all the other uranium exposures is cost benefit analysis or utilitarianism. But first, such a philosophical basis of the democratic social contract was abandoned by the end of the 19th century. Increase in overall average good or utility is consistent with a slave society. It was largely replaced by Rights-based philosophy such as that of John Rawles. These are incorporated now into the UN charter. Individuals have rights. These rights are (in principle) paramount: that is they overshadow collective considerations. An individual has absolute right over the integrity of their body, and certainly have the right to say that they do not want it contaminated by uranium dust produced by a mine upwind of where they live. It is not enough to say (as AREVA begin to) that the uranium is good for global warming (through some tortuous thread of reasoning, since the operation itself will be very bad for global warming and produce enormous amounts of CO2, not calculated or assessed in the EIS). Nor is it possible to break these rights by arguing that money can be made through exporting uranium. This separates those who make the money from those who die of cancer or whose children are born with deformities or born dead. In any case, the producers and exporters of an extremely genotoxic and ultimately fearfully apocalyptic substance like pure uranium might reasonably be considered to be in a similar or more extreme category than say exporters of dangerous drugs, opium, heroin, or maybe those companies that made the poison gases that were used by Saddam Hussein on the Kurds. And if this is considered extreme, think of the 350 tons of uranium weapons used in Iraq in Gulf War 1 and the terrible toll of human sickness and death that produced; more recently in Gulf War 2 when more than 1000 tons of uranium weapons were employed by the US forces. If no uranium were produced, there would be no nuclear weapons. If no uranium were

produced by Canada, the Canadian people would be innocent of the hundreds of thousands of deaths which would follow the use of Canadian uranium.

Finally, operations involving radioactivity and exposures of the public and biota are required by the legal framework of the EURATOM treaty as transposed into EU law to be JUSTIFIED. In addition exposures should be kept as low as reasonably achievable, the ALARA condition. There is no discussion of justification in the AREVA report: it is somehow assumed that the operation is a 'good thing'. There is no discussion of ALARA involving alternative scenarios.

6. Summary of deficiencies in the AREVA EIS report, main conclusions and recommendations

I conclude by summarizing the main points.

1. The scientific basis for the risk assessment, the ICRP model, is no longer valid for uranium. It has been overtaken by new scientific research which shows that doses to local human receptors would be illegal under present statutory constraints. Failure to address this point will result in legal challenges which will succeed.
2. The EIS report is inadequate for a number of reasons. First it employs the incorrect risk model. Second it fails to present sufficient data enabling analysis of the mathematical models of dispersion and exposure it does present. Third it fails to present any *justification* for the proposed project, a requirement of the EURATOM treaty. Finally it fails to account for the overall planetary impact of the project both through the distant downstream use and dispersion of uranium and its fission products but also the local production of large amounts of carbon dioxide.

I recommend that before any decision is made, the company be asked to address these matters in a separate report and that the relevant authorities arrange to properly examine the issue of uranium genotoxicity and its origin in DNA affinity and photoelectron enhancement. I should be happy to cooperate in such a process.

C. Busby
Feb 15th 2008

References

References cited in the text are to be found in the reference section of the various Appendices apart from those which are given below.

Paternain JL, Domingo JL, Ortega A and Llobert JM (1989) The effects of uranium on reproduction, gestation and postnatal survival in mice. *Ecotoxicol. Env. Safety.* 17: 291-296 (cited in the AREVA EIS)

Appendix A: Ionising radiation and health

I will condense much of the historical evidence from an earlier review in my book *Wings of Death* 1995 which I refer to.

In 1895 Wilhelm Roentgen discovered X-rays: whilst experimenting with the passage of electricity through an evacuated glass tube, he noticed that a phosphorescent screen elsewhere in the laboratory glowed as some invisible energy was created. He later took X-ray pictures of his wife's hand, which showed the bones and the wedding ring clearly. This was the first use of X-rays to image bones and the medical uses of the discovery expanded from this point to include both investigation and treatment of a huge range of conditions. By 1900 over 20 cases of X-ray injury had been documented in scientific journals, and in 1904, Edison's assistant, who had been seriously irradiated whilst helping to develop a new X-ray lamp, died of cancer. Both hands had become malignant and both arms had been amputated. In 1908, members of the American Roentgen Ray Society listened in silence to a paper describing more than 50 cases of `radiation poisoning'. Many early users of X-rays tried to play down the dangers. A Dr Mihran Kasabian campaigned against the use of the word `burn' to describe the effects of over-exposure on the basis of the emotional connotations: he himself died of cancer in 1910. Shortly after Roentgen's discoveries, Henri Bequerel discovered that uranium ores also gave off similar invisible radiations and the natural elements from which these radiations were originating were researched in the following twenty years. By 1920 deaths from cancers and leukemias amongst the radiation pioneers forced a realization that some protection guidelines were needed. In 1927 the International Congress of Radiology, a consortium of national groups adopted some safety guidelines at its meeting in Stockholm. These were, however, quite arbitrary, and did not relate to the most important question, both then and now: how much radiation is dangerous?

The Biological Measurement of Effect

The earliest methods of measuring biological effects were severe: one yardstick was hair falling out, an after-the-event occurrence that indicated an excessive dose. A more usual marker was the Erythemal (or skin burn) Dose (ED), the amount of radiation which caused the skin to redden. This was a crude measure: the amount of radiation needed to have this effect varied over a range of 1000 for different individuals and different dose regimes. This system of measurement remains in the present assessment of skin cancer risk following exposure to solar radiation. Ionising radiation is vastly more energetic and penetrating.

Such crude immediate biological effects as skin inflammation occurred at radiation levels now known to be enormously greater than those which induce cancer, yet the safety dose limit suggested in 1924 by X-ray manufacturer Arthur Mutscheller in a paper to the American Roentgen Ray Society was 1/100th of the ED per month, or 1/10th per year. The following year, with more logic, Rolf Sievert of Sweden, made the fundamental move that has influenced the perception of radiation hazard ever since. He suggested tying the safe dose to Natural Background Radiation. He had established that people were exposed *externally* to an annual dose of about one thousandth to one ten-thousandth of the ED from naturally occurring radiation. He then decided arbitrarily that humans could tolerate 1/10th of an erythema dose per year without harm, i.e. one

hundred to one thousand times the natural exposure. This figure was close to Mutscheller's. A few years later, two British physicists, Barclay and Cox, published a study of some individuals who had worked with radiation for six years without visible effect: they divided the estimated exposure by a safety factor of 25 to obtain a figure of .08ED per year.

The similarity in these three numbers, though fortuitous, gave a spurious scientific validity to the choice of the first radiation protection standard. Since this was adopted there have been developments in knowledge, but there have been developments in practice also. Any re-assessment of risk has always had to contend with having to force change on people who have been functioning under a standard derived from this first standard. Thus there has never been a total rethink. All that has happened, over the years, has been a minor reduction of each level of safe dose limit against opposing choruses of wails from those in the industry who have been functioning on the previous rule, usually supported by their friends who are, alas, in these instances, employed to defend public safety.

The only logical underpinning of the first dose limit was Sievert's idea to tie exposure to natural radiation. This use of Natural Background Radiation (NBR) as a measure of exposure has continued to the present day. Scientifically, of course, it is only valid if the exposures from natural radiation are the same in type, quality, and magnitude as those under consideration. Owing to the physical methods which were developed to measure radiation and the fact that these were devised by physicists, concentrating on energy and energy transfer, the NBR yardstick approach was not, and is still not, questioned. (For more detail on the development of knowledge on the biological measurement of effect see Caufield, 1989).

During the first twenty years of the radiation age physical science developed many methods for measuring radiation quantity. All of these gave results based on energy transfer. Energy, however, can be transferred in a multitude of ways, and takes many forms; on its own, energy transfer is a totally useless measure of quality of effect. For example, one cup of boiling water at 100 degrees centigrade contains the same energy, the same number of Joules, as a bucket of water at the temperature of twenty degrees. An energy transfer to a person of one waterthrow unit could encompass either a cupful of boiling water in the face or a bucket of water at room temperature: more information is needed before the health consequence can be assessed.

The energy transfer unit developed by the physicists was the Roentgen(R) adopted by the International Congress on Radiology in 1928. The unit was defined as the amount of radiation needed to produce a given number of ions in dry air in an ionization chamber, a device for electrically evaluating such a process.

The necessary step was taken: erythemal dose ED was translated into Roentgens on the basis of common observation in radiation laboratories. Although the range in different individuals was great, an average of 600R was eventually agreed to be the threshold ED. 1/10th of this (the earlier ED defined limit) gave 6R per month as the recommended dose limit. In 1934 the US Committee on X-Ray and Radium Protection arbitrarily divided this by two and rounded upwards to obtain the first tolerance level for radiation exposure. This was 0.1R (or in modern units roughly 1mGy) per day. This is equivalent to an annual dose of 365mGy.

These 1934 standards were presented as being based on a scientifically backed, reasonably precise understanding of the effects of ionizing radiation. They were, in reality, guesses based on inadequate research of overt and gross effects and involved total disregard of the increasing evidence for serious long-term mutation-related problems like cancer. They were based on inadequate sampling, untested assumptions, and on physical models for radiation which were, then as now, far too crude to describe the biological effects of ionizing radiation.

Lauriston Taylor, Chairman of the Committee on X-ray and Radiation Protection in 1933, later said of the work that the standards were based on `This work was seriously flawed, and yet that is still the basis for our protection standard of today. It really is.' (Caufield, 1989: 21)

But if the understanding of radiation effects from external acute delivery using X-ray machines was flawed, then this flaw represented only a minor error, a slight scratch on the surface of the glass, compared with the shattering inadequacy of the acute physical energy-transfer model used to account for biological consequences of substances which delivered their energy from within living tissue. Since I will argue that internal isotope exposure is the overlooked hazard of the nuclear age, it is necessary to back-track and return to the discovery and parallel development during the infant X-ray age of the phenomenon of radioactivity.

Radioactivity and its Biological Effects

One year after Roentgen's discovery of X-rays, in 1895, Henri Bequerel, in Paris, found that certain naturally occurring minerals gave off weak, but similar radiation. The rays that emanated from the Uranium-containing ore, pitchblende, were capable of fogging sealed photographic plates, in the same way as X-rays. Bequerel showed that this radiation was capable of passing through thin metal plates. In 1898 Marie Curie coined the word `radioactivity' to describe the effect. She began to look closely at the materials which exhibited the effect and identified, in pitchblende, a novel and highly radioactive element besides Uranium: she called it `Radium'.

Her epic work to chemically isolate Radium, processing thousands of kilograms of radioactive ore, resulted in the isolation of one gram of Radium. She and her husband Pierre shared the Nobel Prize for this discovery. She died of a form of blood cancer (aplastic anaemia or leukemia) in 1934.

In the ten years that followed her discovery, Ernest Rutherford, who was laying the experimental foundations for the understanding of modern atomic theory, was able to describe accurately the quality of the radiation emitted by radioactive substances and identify their source in the nuclei of the heavy atoms involved in the phenomenon. These radiations are the alpha and beta particles and gamma rays.

If their characteristics had reminded Bequerel of X-rays, their biological effects were equally alarming. In 1901 he borrowed from the Curies a phial containing a minute quantity of a Radium salt. After carrying the tube in his waistcoat pocket for six hours, he noticed that he had burned his skin through several layers of clothing. The doctor that he consulted said that the lesion was identical with an X-ray burn.

In the years that followed this discovery, radioactive materials became used extensively as a convenient source of radiation in medicine. One of the developing uses for X-rays was the treatment of cancer: they are still used for this purpose. It had been

discovered that the irradiation of tumours by X-rays or by the radiation from radioactive substances often caused their regression, although the reason for the effect remained obscure. We now know that radiation is selective for cancer cells because radiation kills cells which are dividing more efficiently than cells which are in a stationary phase of their life cycle. (As a treatment, this is a last ditch strategy, since all radiation exposure carries risk of mutation and cancer in healthy cells: thus new cancers can, and do, appear later).

But most of the radiation effects described and understood in this atmosphere of scientific advance and general euphoria, related to exposure from external sources. Thus X-rays emitted from a vacuum tube were directed onto the surface of an individual, who perceived burns. Bequerel's skin-burn was of this type, despite the source difference. Measurements made by scientists using the detectors developed for the purpose were measurements of radiation falling on the detector from an external source. The relation between exposure and background radiation also assumed that energy was transferred to an individual from an external source.

The discovery of Radium and the existence, in Canada, of Radium-bearing uranium mineral ore rapidly resulted in the substance becoming commercially available. Preparations containing Radium, sold as part of the magical new age, as the elixir of life, became incorporated into a wide range of nostrums. There were Radium-containing general tonics, hair restorers, toothpastes and cures for all ills from arthritis to infertility. A hearing-aid was marketed with the magic ingredient, `hearium'. One most popular and widely used preparation was `Radium water', often referred to as `liquid sunshine'. One company in New York claimed to supply 150,000 customers with radium water. Another brand, `radithor' was so radioactive that several users died from Radium poisoning. One of these, a Pittsburgh industrialist and amateur golf champion, Eben Byers, drank a two-ounce bottle daily for several years; he believed it made him fit, and pressed it on his friends. He died of multiple decay of the jawbone, anaemia and a brain abscess in 1932.

The first clear evidence that internal irradiation from radioactive substances like Radium caused serious health problems was the death, between 1920 and 1924 of nine young girls employed by the US Radium Corporation to paint the dials of watches and clocks with a luminous, Radium-containing, paint.

The Tragedy of the Dial-Painters

The story of the dial-painters and their fight to obtain recognition for the cause of their cancers and other grave illnesses is similar in every respect to the many attempts that have been made up to the present day by groups who have tried to argue that their injuries were caused by radiation, from the Atomic Test veterans to the Sellafield leukemia victims. For this reason, and as the first example of the assault on the external versus internal irradiation dose comparison, their history deserves closer attention. (My account is based on that in Caufield, 1989: 29-43.)

The dial-painters used to keep their paint-brushes pointed by licking the tips. Although Radium was known to be highly radioactive, the amounts used in the paint were truly tiny, and it was assumed that the procedure was safe. The underlying assumption, of course, was that the energy transfer was very small. It was also believed, on no evidence, that any Radium ingested would pass straight through the body in a short time.

Nevertheless, the dial-painters began to suffer serious problems. Death certificates cited many different causes of death: stomach ulcer, syphilis, trench mouth, phosphorus poisoning, anaemia, necrosis of the jaw. Many who were still living were seeing dentists, with severe tooth and jaw problems. In early 1924, concerned by the emerging illnesses of the dial painters, the local Board of Health asked the Consumers League of New Jersey, a voluntary group concerned about the employment of women and children, to investigate working conditions in the US Radium factory.

Katherine Wiley, the group's secretary, wrote that four of the dead women had undergone surgery of the jaws, and that many still living former dial-painters were similarly afflicted. But she found no problems with working conditions at the factory, nor did the New Jersey State Department of Labor, which also examined the plant. The US Radium Corporation assured both groups that Radium was not harmful at the minute levels involved, which were vanishingly small compared to the erythemal dose from an X-Ray machine. They ascribed the dial-painters troubles to poor dental hygiene. More recently, in an echo of this, the massive increases in cancer, leukemia and birth defects in the former Soviet Union following Chernobyl have been blamed by the risk agencies on hysteria or on malnutrition (see Busby and Yablokov 2006).

In 1924 a consultant dentist, Dr Theo Blum, who had treated one of the dial-painters, published a paper in the *Journal of the American Dental Association*. In it he mentioned that in 1923 he had treated a case of `infection of the jawbone caused by some radioactive substance used in the manufacture of luminous dials for watches.' This was the first suggestion that radioactivity from Radium may have been the cause. The article was noted by Dr Harrison Martland, Medical Examiner of Health for Essex County, home of the Radium factory. Martland began studying the problem and decided to perform autopsies on the next US Radium Corporation employees to die.

Meanwhile, Katherine Wiley consulted Florence Kelley, the head of the National Consumers' League, who, in turn, passed the problem on to Dr Frederick Hoffman, the Prudential Life Assurance Co.'s chief statistician, to investigate. Hoffman reported to the American Medical Association in May 1925. The epidemiological evidence he presented confirmed that some factor related to work at the Radium plant was causing death amongst workers from illnesses of the mouth and jaw. He stated that he believed that Radium poisoning was the cause. The company continued to argue that this was impossible, that the exposure was too low.

What no one knew, outside the US Radium Corporation, was that the company itself was well aware of the cause of the illnesses, having commissioned its own study one year before Martland's report. Cecil Drinker and colleagues from the Harvard School of Public Health had been asked by US Radium to investigate and had already reported their findings. They had stated that radiation was the cause of the employees' ill health. Examining the girls who worked there, in a darkened room, they wrote: `their hair, faces, hands, arms, necks, dresses, the underclothes, even the corsets were luminous.' Tests on twenty-two employees failed to find a single one whose blood-count was acceptable. That all the workers were exposed to excessive radiation, both external and internal, was in writing and on the desk of the director of the US Radium Corporation one year prior to Hoffman's paper. `It seems necessary therefore, to consider that the cases described, have been due to Radium' the Report stated. The company blocked external publication with threat of a lawsuit. When Drinker learned of Hoffman's scheduled address to the AMA on

`Radium Necrosis' he begged US Radium to allow him to publish. They refused, although they sent an edited version, absolving them of responsibility, to the New Jersey Department of Labour.

At about the time of the Hoffman Report, Harrison Martland was able to do biopsies on the jaws of two dial-painters who were suffering from `jaw necrosis and severe anaemia'. Both died shortly after and Martland confirmed high levels of radioactivity in the women's bones and organs. He tested a number of living dial-painters and found that their bodies contained so much radioactive material that when they exhaled on to a fluorescent screen, it glowed (Martland, 1929).

Martland and co-workers became the first to understand that internally ingested radioisotopes behave in the body quite specifically and in a manner related to their biochemical nature. Instead of passing through the bodies of the dial-painters, Radium, an element of the Calcium family, became stored in bone and teeth instead of Calcium. In addition, as a member of the Calcium family, Radium should bind to DNA. A build-up of radiation caused damage to the tissue adjacent to the storage site which had become a radioactive source. Furthermore, and the main reason why external irradiation studies cannot safely inform internal radiation risk, there was an enormous dose to adjacent tissues from the intensely ionizing alpha-particle radiation characteristic of Radium. *External* dose considerations were wholly inappropriate. The dose from a single decay was lethally effective against the cells close to the atom. Such a dose, delivered externally, would have had no effect whatever, since the alpha-particle would not even penetrate the skin.

Martland continued to investigate Radium: he found that early stages of internal radiation made victims feel well, as the radiation stimulated excessive red-blood-cell production. He found that there was a time-lag between radiation ingestion and the onset of disease, often a considerable time-lag. This time-lag was a death sentence for many who were part of the Radium Company's operation at the time of Martland's report. In 1925 Edward Lehman, their chief chemist, was in good health: he died shortly after of acute anaemia and the autopsy showed radioactivity in his bones and lungs. Since he had not painted dials it was clear that he had acquired his dose by inhalation.

The Radium Company refused to accept the radiation poisoning hypothesis. They commissioned new studies which exonerated them. They blocked reports using legal pressure. Several families sued them for damages, as did Dr Lehman's widow. The newspapers took up the case of `The Five Women Doomed to Die' who had filed for damages. They were so wasted and ill that they had to be carried to the witness-stand: one was unable to raise her hand to take the oath. The Company maintained that there was no scientific proof that the dial-painters' injuries were caused by Radium. Its lawyers, however, chose to fight on a different front, arguing that New Jersey's statute of limitations required industrial injury pleas to be filed within two years of the occurrence. The Court accepted this, the women petitioned, and the case rumbled on. Following huge pressure, the women were granted permission to go to the Supreme Court. US Radium still denied responsibility for their injuries. The case seemed set to drag on for years; the women were dying. Eventually the Company `prompted solely by humanitarian considerations', settled out of court for half the amount that the women claimed. They still had not conceded that internal irradiation from Radium was the cause of the diseases which were killing their employees.

Development of Dose-Response Relations to the Present Day

With the dial-painters' tragedy came the first recognition that ionizing radiation acted in ways that were not predictable from simple physical considerations. Internal irradiation by a specific radioactive element was seen to produce appalling effects, often long delayed, at levels of energy transfer that seemed vanishingly small. Since many preparations freely available on the market contained Radium, guidelines were clearly needed to safeguard the public, and between 1936 and 1938 experiments were begun on animals to try to establish safe limits. But it was only when the need for luminous dials increased with the Second World War that, in 1941, the US Bureau of Standards met to present draft rules for Radium contamination. As in the case of the early external irradiation limits, the results were hurriedly patched together by guesswork: a limit of 0.1 Curies in the whole body was given as a reason for changing personnel to new employment; a limit of 10 picoCuries (pCi) of Radon gas per litre of air was also set, and the 0.12R per day X-ray limit was extended to γ-ray exposure. The establishment of even these high levels of statutory exposure limits probably saved many lives during the ten years that followed; years that saw, with the US Manhattan Project, the development of the atomic bomb.

In 1946, to control the development of all things atomic which, following the Hiroshima bomb were seen to be associated with national security, in the United States the Atomic Energy Commission (AEC) was formed. There soon followed the revival of the US Advisory Committee on X-Ray and Radium Protection, which needed to consider safety levels in view of the new practices and new isotopic contaminants which followed the development, testing, and use of atomic weapons. The Committee changed its name to the National Council on Radiological Protection (NCRP) and expanded.

The NCRP consisted of eight representatives of medical societies, two of X-ray manufacturers, and nine of government agencies including the armed forces, the Bureau of Standards, and the AEC. From the very start, the AEC put pressure on the NCRP to devise a permissible dose level. Of the eight sub-committees set up to consider radiation-related practices, those which were attempting to set dose limits were Sub-Committee One on external dose limits, headed by Giaocchimo Failla, and Sub-Committee Two on internal radiation limits, headed by Karl Z. Morgan. The external dose limits were soon set: by 1947 Failla had reduced the existing X-ray/γ-ray limit from 0.1 rem to 0.05 rem per day (180mGy a year). The reduction was partly based on the new discovery that radiation caused genetic damage. Experiments with fruit flies showed that even tiny doses of radiation resulted in the production of mutated offspring. This raised the obvious question about similar damage to humans. The problem was that practices involving doses to workers and members of the public much higher than those involved in the fruit-fly experiments had already been sanctioned by the earlier guesstimate dose limits then in use. Since, also, national security demanded continued research, development, and testing of atom bombs, there was no way in which NCRP would have been able to set dose limits at zero dose or no exposure. On the basis that such a move would be unrealistic, the NCRP canvassed the nuclear industry on what was the lowest value for the dose limit that they could function with. This figure was the one that was adopted. Owing to arguments between Failla and Morgan, who felt that more control of exposure was needed, the dose limits were not published until 1954.

Sub-Committee Two, under Morgan, had the job of assessing the risks from internal exposure due to ingested radioisotopes. What was required was the development of an understanding of the effects of ionizing radiation delivered by an atom incorporated within living material and decaying to deliver its energy into adjacent tissue. What they proceeded to do instead was to apply the physical model for external irradiation to internal organs which were assumed to be `target organs' on the basis of radio-chemical affinity, and to see these organs as neutral volumes of irradiated water in which a certain amount of energy was dissipated. This is a typical physics-based reductionist trick. It has great computational utility, but as far as biological responses are concerned it is entirely inadequate, and as I shall show, gives the wrong answer.

The primitive erythemal dose threshold arguments together with the development of the physical-energy-based units-- Roentgen, rad, Gray etc.--gave limits for external dose based on a model which involved so much energy transfer with a 70 kg. sack of water called a `reference man'. The modification needed for understanding internal irradiation was obvious. The organ most likely to concentrate the particular radioisotope being considered was defined as a `target organ' for that substance. The dose limit was then set assuming that the organ of mass *m* was a smaller sack of water into which so much energy was transferred. The same erythema-based, *ad hoc*, and arbitrarily developed dose limit could then be applied.

These dose limits were translated into maximum permissible concentrations or MPC of the particular radioisotope. Morgan clearly recognized the dubious nature of these arguments and the shakiness of the whole analysis: his Committee Two proposed that the MPC they calculated be divided by a `safety factor of ten' for people who might be exposed for thirty years or more. This represented official unease about the differences between acute external and chronic internal exposure: the conflict between the understanding of physics and that of biology.

There was much argument about the adoption of recommendations from Morgan's group, and the final report did not include the proposals for people likely to receive prolonged exposure. In 1953 the final form was adopted by the NCRP, and to make the standards seem more weighty, the young US nuclear establishment encouraged, and funded, the formation of the International Commission on Radiological Protection (ICRP). This organization has been described as being little more than the overseas branch of NCRP (Caufield, 1989: 175). It is based in the UK.

These radiation protection advisory commissions, and their offspring, the radiological advisory bodies in most countries like Britain's National Radiological Protection Board (NRPB, which shares many personnel with ICRP, yet cites the latter as an 'independent source' of advice), now publish advice on dose limits and protection which becomes incorporated into law. They control the perception of hazard from all things nuclear. They are all, however, lineal descendants of the first NCRP committee, staffed by people who all had interests in the development of the use of radiation. They remain, to this day, a revolving door through which members of the nuclear establishment or those with research ties to it, pass in and out.

The first recommendations of the original 1953 committee became US law in 1957, yet those recommendations arose in an atmosphere of haste, error, necessity, secrecy, and lack of knowledge. In 1962 an AEC scientist, Harold Knapp, studied the exposure of young children to radioactive iodine in milk. He concluded that standards

were too lax by a factor of ten, and recommended that they be tightened. The response from the AEC director of the Commission of Operational Safety was that
'the present guidelines have, in general, been adequate to permit the continuance of weapons testing and at the same time been accepted by the public principally because of an extensive public information programme. To change the guides would raise questions in the public mind as to the validity of the past guides'. (Caufield, 1989: 132)

This continued to be the case with radiological safety, and it continues still. Present radiation protection laws, based on the cancer yield of acute radiation exposure events like the Hiroshima bomb, leave much of the actual practice to the users and producers of radioactivity by asking them to keep doses `as low as reasonably achievable' (ALARA). Sir Kelvin Spencer, formerly Chief Scientist for the UK Ministry of Power said:
'We must remember that government scientists are in chains. Speaking as a one-time government scientist I well know that `reasonably achievable' has to be interpreted, so long as one is in government service, as whatever level of contamination is compatible with the economic well-being of the industry responsible for the pollution under scrutiny'. (Caufield, 1989: 190)

The 1957 statutory crystallization of the 1954 NCRP recommendations occurred during the period of intense scientific research which followed the Second World War. By 1957 enough was known about cell genetics and DNA damage to understand the cellular origins of radiation effects. It had always been clear that ionizing radiation did not kill by gross energy transfer: the effects were delayed, the amounts needed to kill an individual would not heat the body up by more than a fraction of a degree. With this new knowledge--that it was primarily cellular genetic changes which were occurring--it must have been apparent that there could be no safe dose of radiation. Even then it was known that ionizing radiation caused damage to genetic material in cells under all conditions of irradiation, even for the smallest doses which can occur. It could be shown that there was no safe dose, or no threshold below which radiation is safe, and indeed this is now the affirmed position of both the ICRP, the NCRP, and the Biological Effects Committees of the US National Academy of Sciences (see BEIRV, BEIRVII).

External and internal radiation.

7. In order to understand the nature of the argument about internal radiation and health it is first necessary to review some basic principles and examine some of the assumptions at the base of radiation risk. These arguments are elaborated in the CERRIE minority report, the CERRIE majority report and in the early chapters of the ECRR2003 report. A more accessible explanation of the basic science is given in my book *Wings of Death 1995.* Ionising radiation acts though the damage to cellular genetic materials, the genes on the DNA, killing some cells but causing fixed genetic mutation in others, including mutations that signal to descendants a genomic instability message to increase their rate of incorporated error. These genetic and genomic mutations are now known to be the main initiation point in the development of cancer and leukemia and also the origin of heritable damage and increases in many illnesses that were not originally thought to be radiation related. It is the progression of the cellular mutation and the acquisition of further mutations over the lifespan of the cell or its descendants (in the same individual or in the

case of germ cells in offspring) that leads eventually to the clinical expression of the cancer. The damage to the DNA is caused either by ionisation of DNA materials themselves directly, or more likely indirectly by the interaction of the radiation track (which is the track of a charged particle, an electron or a alpha particle) with solvent water or other molecules to form 'hot' ionic species which are sufficiently reactive to attack the DNA bases. To a first approximation, it might be argued that over a certain range of dose, the effect, or likelihood of mutation, is a linear function of the amount of energy absorbed. That is because this energy goes to break bonds and produce ions, and twice the energy produces twice the ions and therefore twice the probability of mutation. But note here that the primary cause of mutation is the reactive ion and so it is the concentration of reactive ions in the cell which represents the most accurate measure of mutagenic efficiency (although there are other considerations as we shall see). The assumptions that underpin the whole of radiation protection are based on the ideas that the dose and the response are linearly correlated. Thus, if we double the dose, we double the effect. We must note this carefully at this point since it is the basis of the present system of radiation risk assessment, and specifically the basis of the calculation made using the model of the ICRP and all predictions that follow from this approach.

But it is manifestly and philosophically wrong to employ such a model for internal irradiation. This is because the quality used to measure radiation, Absorbed Dose (in rads or Grays) represents the average energy absorbed in unit mass, in the case of Grays, Joules per Kilogram. Such a quantity assumes at the outset that the energy density is the same in all the cells of the tissue irradiated. Whilst this is a valid assumption for external irradiation as in the case of the studies used to determine cancer and leukemia risk (particularly the major study, that of the Japanese A-Bomb survivors) it is manifestly untrue for modeling risk in individuals who have internal irradiation. The reason is that in many internal irradiation regimes, averaging is not appropriate. Radioactive particles which emit short range radiation like alpha and beta radiation cause high levels of energy density (ionisation) in local tissue (a few millimetres away) but no irradiation elsewhere. Thus cells near to these particles receive large either fatal or mutagenic doses. To illustrate this I show in Fig 1 of the main report a photomicrograph of decay tracks from a few radioactive particles in rat lung.
This phenomenon is known as an alpha star: the tracks are alpha particle ionization tracks such as those produced from uranium and radium dust particles.

Averaging the energy into large tissue masses in whole body or in organs, dilutes the ionisation density and makes it seem as if the whole body doses are very low, perhaps well below natural background doses. But since cancer always starts in a single cell (as we know from mosaic studies of tumours) it is the cell dose that is important, not the tissue dose. The use of external doses to calculate cancer risk (as the ICRP do) is like comparing warming oneself by the fire with eating a hot coal. This argument has now been accepted at the highest level, although little has been done to incorporate it into risk management. It is a major plank of the ECRR deliberations and now in the mainstream of argument in the radiation risk community. Chapters 5 and 6 of ECRR 2003 and pp 48 to 56 of the CERRIE Minority Report discuss the concept of Dose, used by the ICRP model as a measure of radiation exposure, in dealing with health effects. In addition, the matter is reviewed by the CERRIE Majority Report (2004) which agrees that (p13 para 11)
There are important concerns with respect to 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.

This is quoted from an official report of a UK government committee. The point is made regularly elsewhere in the same report, (e.g. para 60 p27) and the Majority Report concludes that there is a conceptual uncertainty associated with the use of absorbed dose of a factor of 10-fold. The Minority CERRIE Report argues that this figure is more like 100-fold to 1000-fold for very low doses and certain types of exposure and advances proofs of this (see below). In addition, recently, the French official radiation risk agency, Institut de Radioprotection et de Surete Nucliare (IRSN), agree that the ICRP dose averaging approach is insecure. In a report published in 2005 they point out that the questions raised by the ECRR2003 report relating to the question of internal doses are valid. The IRSN committee of 15 senior scientists state that these are *fundamental questions with regard to radioprotection* and (p6) that *heterogeneous distribution of radionuclides, the validity of weighting factors for calculating internal doses, the impact of the radionuclide speciation on their behaviour and their chemical toxicity* make it clear that the ICRP approach for certain internal radionuclides is *strictly invalid.* IRSN state that *since the ICRP60 publication, improvements in radiobiology and radiopathology, or even general biology finally might impair [*falsify*] the radiation cell and tissue response model applied to justify radioprotection recommendations.*

[IRSN 2005]

ICRP itself was under pressure on this issue by 2005 and conceded in its draft report on risk**:**

(50) For radiations emitted by radionuclides residing within the organ or tissue, so-called internal emitters, the absorbed dose distribution in the organ depends on the penetration and range of the radiations and the homogeneity of the activity distribution within the organs or tissues. The absorbed dose distribution for radionuclides emitting alpha particles, soft beta particles, low-energy photons, and Auger electrons may be highly heterogeneous. This heterogeneity is especially significant if radionuclides emitting low -range radiation are deposited in particular parts of organs or tissues, e.g. plutonium on bone surface or radon daughters in bronchial mucosa and epithelia. In such situations the organ-averaged absorbed dose may not be a good dose quantity for estimating the stochastic damage. The applicability of the concept of average organ dose and effective dose may, therefore, need to be examined critically in such cases and sometimes empirical and pragmatic procedures must be applied.

But ICRP did nothing to change any of the dose coefficients for isotopes that caused such exposures or to apply such *empirical and pragmatic procedures.*

Dose constraints and risk models after 1980

8. As I have explained, the history of radiation and health is one in which the cancer and leukemia risks following exposure have been reassessed continuously upwards over the whole of the radiation age. The annual dose limits have fallen from around 400mGy in

1934 to 200 mGy (or 200mSv) in the early 1950s and by 1974, ICRP 26 recommended an annual limit of 5mSv to members of the public and 50mSv to workers. This was modified by ICRP in 1985 to 1mSv. NRPB in the UK reduced this further in 1987 to 0.5mSv from a single site exposure. In the US the single source exposure level is now 15mRem or 0.15mSv. Levels are now, in the UK and Europe fixed at 1mSv (100mRem) for members of the public and 20mSv for workers. I should explain that the mSv is a unit which derives from the mGy in the same way as the rem is derived from the rad, by the use of a multiplier of effect based on the type of radiation. Alpha radiation is known to give very dense ionization over a short track length of about 40 micrometers (three to five cells). It is assumed to therefore have 20 times more biological effectiveness owing to its 20-fold greater ionization density and thus, for internal exposure carries a weighting factor under ICRP of 20. Thus a dose of 1mGy becomes a 'dose equivalent' of 20mSv. This concession to ionization density effects is not extended by ICRP to other types of internal irradiation (e.g. particles, DNA bound isotopes) where much higher density of irradiation occurs, because to do so would concede the high risk effects of such exposures and point to cancer causality in groups who were contaminated internally. On the other hand, the ECRR model has taken this step and introduced weighting factors for such regimes (see ECRR2003 Chapter 6), and this results in significantly higher effective doses from certain types of internal exposure using the ECRR model than the ICRP model.

It has been conceded since the early 1990s (see e.g NRPB 1995) that there is no safe dose of radiation. It is necessary to realize that these dose limits have stopped being reduced because of pragmatic considerations relating to the operation of nuclear facilities only and not because of a sudden realisation that the health effects are now known and allow us to make accurate limits which we know will prevent the illness of exposed people. For example, the dose limit constraints should have been lowered when the most recent results of the Japanese A-Bomb study data became available in the 1990s and showed that the cancer risk continued to rise in the survivors study group. But this could not be allowed to be incorporated into law generally as it would make nuclear power and certain other industrial activities impossible, and so the concept of ALARA was substituted into most legal frameworks which follow ICRP e.g. the Basic Safety Standards Directive of the EU which became member state law in 2000. ALARA stand for 'As Low As Reasonably Achievable'. In practice, 'Economic and Social Considerations Being Taken into Account.' were added.

By this continuing increase in perceived cancer risk with dose, I mean in relation to the safety of exposures as measured officially using external radiation studies, in particular the Hiroshima survivors study. The matter of internal exposure cannot be informed by these external studies. Indeed, when we look at internal risk through the lens of epidemiology, we see that the risks are hundreds of times higher than predicted by the external risk models based on Hiroshima, and enable us to both predict and explain the clusters of childhood cancer and leukemia near nuclear polluting sites which were discovered in the 1980s.

The recent revolution in radiation risk perception

Sellafield and the nuclear sites

The first evidence that radiation risk from exposure to internal radionuclides was significantly greater than that predicted by ICRP was the discovery in 1983 of a cluster of childhood leukemia cases in children living near the Sellafield nuclear reprocessing site in the UK. This discovery, made initially by a TV company, was the subject of a government inquiry which found that the cluster was real but that the ICRP risk model could not predict the levels of leukemia. The difference between the prediction of the ICRP model and the excess leukemias was 300-fold. Remember that number. The matter is discussed in the CERRIE minority and majority reports and in ECRR 2003. The discovery was followed quickly by others so that by the mid 1990s childhood leukemia clusters had been discovered near all three nuclear reprocessing sites in northern Europe and a good many other nuclear facilities. These sites had in common that they released fission product radioisotopes and technologically enhanced natural isotopes TENORM to the environment. In all cases, the relevant authorities discounted causality on the basis of application of the ICRP external model, even though it was a case of internal exposure. In every case, the discrepancy between the doses and the measured and predicted effects were between 300-fold and a few thousand -fold. In the case of Sellafield measurements had been made on autopsy specimens which showed that particulate material released by the plant (Plutonium, Uranium) was most concentrated in the lymph nodes draining the lungs. Thus there was evidence in the mid 1980s that radioactive material from the nuclear site concentrated in small lymphatic masses weighing about 11gms each. The Committee on Medical Aspects of Radiation in the Environment COMARE, the main public body set up after the 1983 inquiry to examine the possibility that the radiation was the case of the leukemia conceded in its Fourth Report (COMARE 1996) into the Sellafield leukemia cluster <u>that the lymph nodes were known to be the site of leukemias in animal studies</u> and yet accepted calculations of the doses to the lymphatic system from enhanced levels of Uranium from Plutonium that used the ICRP dilution model, in this case diluting the energy into an assumed body organ mass of 11kg. Since dose is Energy divided by Mass this dilution reduced the dose by
1000-fold.

New Science

9. The last fifteen years have seen a revolution in the scientific understanding radiation action at the cellular level and of cancer causation by radiation. Much of what I will briefly say here is elaborated in the CERRIE Majority and Minority reports. I will try to just make the most important points .

Genomic Instability and the Bystander effect

It was discovered in the early 1990s that a single track from an alpha particle across a cell caused an effect called Genomic Instability. What happened was that the cell survived but the descendants of the cell seemed prone to spontaneous and random genetic mutations. Prior to this discovery, it was assumed that cancer and leukemia were caused by a specific genetic mutation which was then passed on to daughter cells. However, this latter theory (which is the physical basis for the present ICRP model) was unable to explain the

normal cancer rate in human populations given the experimentally derived normal mutation rate of 10^{-5}.

Further experiments into the phenomenon showed that it was a property of all tissues and was induced by the lowest doses of all kinds of ionizing radiation. It rapidly came to be seen that this was the basis in genetic mutation of most cancer.

But this discovery was followed by a very strange discovery. It was found by several groups that if a cell was hit i.e intercepted by a track of ions, then not only the cell affected suffered genomic instability, but also cells which were not hit and which were up to 400 or more cell diameters distant from the target cell. This phenomenon was termed the bystander effect.

There are three basic implications for radiation protection, and by implication, the present assessment of uranium exposures. The first is that the basis for assuming that the relationship between cause and effect, dose and cancer yield is a linear one (i.e double the dose and you double the cancer risk) is shown to be invalid. The dose response relation of Genomic Instability and Bystander effects is sharply supralinear. It increases rapidly with the first two tracks, then flattens off. This means that you cannot, as ICRP have, extrapolate from high dose (Hiroshima survivors) to low dose. There is a much higher proportionate effect at low dose. The application of Dose Rate Reduction Factors to low dose radiation by risk agencies is strictly invalid and arises out of a mistaken interpretation of low dose points in the experimental results. The same error in interpretation has allowed some to believe that low doses of radiation are protective. The second implication is that two tracks across a cell or into tissue (since the bystander effect connects all the cells in a small tissue volume) has a proportionately greater effect than one track and that after three of four tracks the effect saturates. The outcome is that there is a range of ionization density that has a much enhanced ability to cause cancer. This range is unlikely to be reached in external irradiation until the levels of dose to the whole body are high, but can be reached in the case of tissue exposed to local decays from internal radioactive particles. The activity of such particles needs to not be too high for if the local ionization density involves more than three alpha tracks to a cell, the cell is killed. This leads to the theoretical prediction that in the system as whole, and looking at cancer or leukemia as an end point, the dose response relationship is likely to be BIPHASIC. That is to say there will be a large effect at low doses (the doses being conventionally calculated using the ICRP model), then the effects will fall off as the dose is increased, only to rise again at even higher doses as tissues of less sensitivity are attacked.

The third consequence of the discovery of genomic instability is that it predicts that there will be a *range of harmful effects* from exposure to radiation. There will not just be cancer and heritable damage, but because of the damage to whole systems in the body, there would be expected to be effects in a range of diseases,. Such effects have been reported in those exposed to radiation both after the Japanese A-Bombs and also after Chernobyl (ECRR2003, ECRR2006).

This brings me to another theoretical argument which was developed by me in the late 1980s and is also discussed in the two CERRIE reports. This argument relates to the Second Event Theory (see Wings of Death 1995, CERRIE 2004 and CERRIE Minority 2004)

Doses to local tissue over time.
For external radiation at low dose (1mSv annually), where the track density is low, cells receive on average 1 hit per year. This damage they have evolved mechanisms for dealing with. If the damage is great and surveillance enzymes detect a mismatch between the two halves of the DNA duplex, then the cell may move from quiescent phase into a repair replication cycle and repair the damage and replicate. The period of this cycle (which cannot be halted once started) is about twelve hours. The result is two daughter cells which a have copies of the repaired DNA. However, if a second track damages the DNA towards the end of this period, there is no possibility of a repair and the mutation is copied to one of the daughter cells. This is a very efficient way of introducing a fixed mutation. It is very unlikely to occur with external radiation tracks (since at low dose, to hit the same cell twice is like discharging a rifle in the general direction of Texas and expecting to hit the same person twice). But for internal isotopes bound to DNA or internal particles, this sequence is billions of times more likely. This represents another reason why internal radiation is not modeled by the ICRP model (which assumes at low dose that each cell is hit only once in a year and that all cells in an exposure carry the same probability of a hit).
Finally, it is valuable to note that the most recent research into genomic instability finds a very wide range of genetic based radiosensitivity. The range is often quoted at up to 1000-fold.

Chernobyl Proofs

10 There are two pieces of information that show unequivocally that the ICRP risk model is in error by a large amount when applied to internal irradiation. Both result from examination of populations exposed to the fallout from the Chernobyl accident. They are both discussed in the two CERRIE reports and also in ECRR2003.

In general, the health effects of the Chernobyl accident have not been adequately examined by the 'official' radiation risk community, and the very large body of evidence that the exposed individuals in the ex Soviet territories have suffered and continue to suffer serious ill health outcomes has been largely ignored in the various official reports in the west, though not in Russian language journals. A compendium of these Russian reports is given as an appendix in the CERRIE minority report, and the situation was flagged up by the eminent Russian Academicians Yablokov and Burlakova at the Oxford CERRIE workshop but nothing was done by the CERRIE secretariat. A comprehensive review of the Russian language literature on the effects of the Chernobyl accident, showing the extremely serious effect of the radiation exposures form the internal radionuclides, was published in 2005 (Busby and Yablokov, 2005) and the cover up of the health effects has been reviewed in my book *Wolves of Water* (2006) and W. Tchertkoff's book *Le Crime de Tchernobyl (2006)*.

The problem in the court of scientific opinion (and indeed in a court of law) with cancer causation is that there is generally a time lag between cause and effect, and since there are many mutagenic causes, it is very difficult to make a connection which is unassailable in logic. In the case of the Sellafield childrens' leukemia (and other similar clusters) despite the fact that they lived near the most radioactively polluted site in

Europe, and that radiation is the only known cause of childhood leukemia, it was argued that the ICRP Hiroshima model did not predict the risk and so it must have been something else. Attacking this logic is easy, but does not result in anything approaching proof. It is not like a murder where a knife is thrust into the victim and the body is found with a knife in its back and the culprit's fingerprints.
However, after Chernobyl there were two discoveries which show unequivocally that the ICRP model is, at least in these specific cases, manifestly incorrect by the same orders of magnitude necessary to explain the Sellafield child leukemias and also many other observations that had been dismissed on the basis of the ICRP Hiroshima external risk models.

I will here advance this proof that the ICRP risk model in wrong by at least a factor of 100 times. The argument has been published (Busby and Scott Cato 2000). This is a simple and brief analysis of the increase in infant leukaemia in five different countries in Europe in those children who were in the womb at the time of the fallout. The countries were Wales, Scotland, Greece, Germany and Belarus. These increases were measured in each country. They were statistically significant and could not have occurred by chance since the calculation for all the countries combined makes a probability of 1 in one thousand million that these were collectively a chance observation. Second, since the group being observed was the *in utero* cohort exposed only to Chernobyl fallout it was an effect of Chernobyl fallout. They were reported in separate papers in the peer review literature by four separate groups of researchers so it was not a biased account by one group. The doses (based on ICRP considerations) had been well described and measured. The only known cause of child leukaemia is ionising radiation. The differences in the levels of leukaemia rates in the exposed cohort and the rate predicted by the ICRP model is greater than 100-fold but varies inversely with the dose. The CERRIE Majority Report conceded this p88 Table 4A6 where it gives the central estimate of error in the ICRP model for Great Britain as 200X, for Greece as 160x AND Germany as 96X. In a paper I published in 2000 with Molly Scott Cato (Energy and Environment, 2000), I calculated for Wales and Scotland the effects was greater than 100X and probably about 300X. This is the exact error in ICRP required to explain the childhood leukaemia cluster at Sellafield, and also the present cancer epidemic. These error factors mean that there are 100 to 500 times more leukemias for a given dose than ICRP calculates.

Minisatellite mutations

The second piece of evidence is the objective scientific measurement by several groups of significant mutation rates in the minisatellite DNA of children and adults living in the Chernobyl affected territories but exposed, on average, to ICRP calculated doses of less that 2mSv a year. Various arguments can be employed to show that this represents an error in the ICRP assessment of genetic damage risk of the order of 500-2000-fold. In one particularly elegant epidemiological experiment, children of Chernobyl liquidators who were born after the accident were compared with siblings born before, to exclude explanations other than the Chernobyl accident. A seven fold increase in minisatellite mutations was found. That these effects are significant for health is seen by another study which showed that plumage changes in swallows that migrate to the Chernobyl region are also associated with minisatellite DNA mutations (for references see CERRIE 2004, ECRR 2003).

ECRR

11. As I have explained, the last ten years has seen a revolution in the perception of risk from ionising radiation and from radioactive substances existing inside the body following inhalation or ingestion. This debate was the subject matter of the three year deliberations of the UK CERRIE committee and also of the considerations leading to the risk model of the European Committee on Radiation Risk ECRR.

The European Committee on Radiation Risk arose out of a deep concern among many distinguished scientists and experts that the risk models for radiation exposure currently employed by national governments to set legal limits for exposure were incorrect by a large amount when applied to internal irradiation. Its committee was begun in 1997 and its origins and remit are outlined in the 2003 report and also on the website www.euradcom.org. In the ECRR report, the ICRP models are shown to be scientifically incorrect for internal irradiation since their basis is external irradiation (from outside the body). Such a model is philosophically irrelevant when applied to internal irradiation from a point source (such as a particle or an atom bound chemically to DNA) as I have explained. I refer to chapters 1, 2, 3 and 6 of ECRR2003.

Summary of Appendix A

The history of radiation risk models shows that the exposure levels permitted by policymakers have continuously been readjusted throughout the last 80 years as every new discovery both in science and in epidemiology has shown that radiation exposure is more dangerous than previously thought. This process continues today. Nevertheless the current official radiation risk models have not incorporated the most recent discoveries since to do so would force a complete reappraisal of the current use of nuclear power and the historic harm done by releases of radioactivity in the past. Contemporary radiation risk models are so inaccurate for internal exposures that even some official risk agencies have attacked them: yet they continue to be employed by governments and used by polluters to justify their past and present behaviour. There is now sufficient scientific proof of this in peer reviewed published literature. These discussions are of relevance to those who are exposed to uranium from the proposed AREVA operation since any assessment of risk to them based upon conventional models will be incorrect on the side of predicting too little harm accruing from the exposures.

Appendix B

The conference proceedings of the German Federal Agricultural Laboratory international conference on uranium are now published as a book chapter:
I attach this chapter separately as an ACROBAT file. The reference is:
Busby C and Schnug E (2007) in *Loads and Fate of Fertiliser Derived Uranium*. Proceedings of a conference held at FaL Braushweig, 2007. Eds. de Kok L and Schnug E Leyden:. Backhuys Publishers. See also Busby (2005) for two earlier papers on the photoelectron enhancements of uranium in the *European Journal of Biology and Bioelectromagnetism* copies of which as ACROBAT files I can also supply.

APPENDIX C: CURRICULUM VITAE

PERSONAL DETAILS

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Nationality: British

FURTHER/HIGHER EDUCATION

Education: 1966-69 Chemistry, University of London

TRAINING AND QUALIFICATIONS

BSc, PhD, C.Chem, MRSC

Qualifications: 1969 University of London First Class Honours Special Degree in Chemistry
1970-71 SRC research studentship for PhD Physical Chemistry (nmr spectroscopy), Queen Mary College, London
1974 Elected Member of Royal Society of Chemistry
1974 Chartered Chemist
1981 PhD Chemical Physics (Raman spectroscopy/electrochemistry) University of Kent, Canterbury

Learned Societies:

Elected: Royal Society of Chemistry-
Member: International Society for Environmental Epidemiology

UK Government Committees

Member: (Department of Health and DEFRA) CERRIE
Committee Examining Radiation Risk from Internal Emitters
www.cerrie.org

Member: Ministry of Defence DUOB
Depleted Uranium Oversight Board
www.duob.org

Other Committees

Scientific Secretary: European Committee on Radiation Risk
www.euradcom.org

Policy Information Network on Child Health and Environment PINCHE

1.4 EMPLOYMENT

1969 – 1975 Research physical chemist, Wellcome Foundation, Beckenham

1975 - 1978 Self employed scientific consultant and science writer

	1979 - 1981 PhD student University of Kent 1981- 1982 SERC Research Fellow University of Kent 1983- 1992 Self employed scientific consultant and science writer 1992- present Science Director, Green Audit, commissioned to research the health effects of ionizing radiation and funded by a number of charities and independent bodies. 1995 Funded by the Joseph Rowntree Charitable Trust to write and produce 'Wings of Death- The health effects of low level radiation.'
1997-2000	Directed research at Green Audit Funded by Irish State to research health effects of Sellafield
1997	Appointed UK Representative of European Committee on Radiation Risk (ECRR)
2001 to doses	Appointed Scientific Secretary of ECRR and commissioned prepare the report ECRR 2003- The Health effects of low of Ionizing Radiation (Published 2003) **2001** Appointed to UK Government Committee Evaluating Radiation Risk from Internal Emitters (CERRIE) **2001** Appointed to the UK Ministry of Defence Oversight Committee on Depleted Uranium (DUOB) **2002** Funded by the Joseph Rowntree Charitable Trust to write a new book on the epidemiological evidence of health consequences of exposure to ionizing radiation: 'Wolves of Water' **2003** Appointed Honorary Fellow, University of Liverpool, Faculty of Medicine, Department of Human Anatomy and Cell Biology **1992**- present: Science Director, Green Audit **2003** Funded by Joseph Rowntree Charitable Trust to write Book *Wolves of Water Cancer and the Environment* **2004**- Leader of Science Policy for(EU) Policy Information Network for Child Health and Environment ***PINCHE*** based in Arnhem, The Netherlands **2005- 3** year research funding by Joseph Rowntree Charitable Trust

1.5 TEACHING EXPERIENCE

1970	Taught O level Chemistry part time, Inner London Education Authority
1980-1981	Gave tutorials in quantum mechanics at the Dept. of Chemistry. University of Kent

1995-1997 Invited lecturer at the University of Sussex Dept of Physics.

1995-1997 Invited lecturer in the University of Wales, `Aberystwyth, Physics Department and Geography Department

2000 – 2005 Invited lecturer in the University of Liverpool Faculty of Medicine SSM5 'Environment and Health' addressing internal radiation risk and cancer epidemiology of small areas.

2005 Invited lecturer University of West of England; Radiation Risk and epidemiology

2006 Invited lecturer: Dept of Law, University of Wales, Aberystwyth

2006 Invited lecturer: Dept of Environment, University of West of England

2007 Invited lecturer: Centre for Molecular Bioscience, University of Ulster

1.6 ADMINISTRATIVE EXPERIENCE

Professional Administration:

Senior Scientist
Dept of Physical Chemistry, Wellcome Research Laboratory, Langley Park, Beckenham
Science Director, Green Audit
2004-2006 Leader: Wokpackage 6 Science and Policy; PINCHE (EU)

Editorial boards (Current):

European Journal of Biology and Bioelectromagnetics

Reviewer

European Journal of Biology and Bioelectromagnetics
European Journal of Cancer
Journal of Public Health (Royal College of Physicians, School of Public Health)
Science and Public Policy

1.7 RESEARCH INTERESTS.

Overview of major lines of investigation

Chris Busby spent seven years at the Wellcome Foundation, where he conducted research into the physical chemistry and pharmacology of molecular drug receptor interactions. He subsequently moved to the University of Kent at Canterbury where he studied Laser Raman Spectro-electrochemistry in collaboration with Shell Research and later as SRC Research Fellow, a project which resulted in a PhD in Chemical Physics.

He developed and published theoretical and experimental details of silver and gold electrodes with surface array properties which enable acquisition of laser Raman spectra of adsorbed molecules in dilute solution.

In the late 1980s he became interested in the mechanisms of low dose internal irradiation and developed the Second Event Theory, which distinguishes between the hazards of external and internal radiation exposure. In 1995 he was funded by the Joseph Rowntree Charitable Trust to develop his arguments and write 'Wings of Death: Nuclear Pollution and Human Health', an account of the results of his research into radiation and cancer and also into cancer increases in Wales, which he argued were a result of global weapons fallout exposure. In 1997 he became the UK representative of the European Committee on Radiation Risk. His analysis of the increases in childhood leukaemia in Wales and Scotland following Chernobyl was recently published in the journals Energy and Environment and the International Journal of Radiation Medicine.

From 1997-2000 he was funded by the Irish Government to carry out research into cancer incidence and proximity to the coast. In June 2000 he was invited to present evidence to the Royal Society committee on Depleted Uranium and health, and shortly after this was invited to Iraq to measure DU in the country and relate exposure to health effects which followed the Gulf War. In 2001 he was asked to visit Kosovo to investigate the dispersion of DU using field monitoring equipment. He discovered DU in many areas from analytical measurements made on samples he collected (paid for by the BBC) he showed that there was atmospheric resuspension of DU particles. His work and expertise in the field of environmental health and radioactivity has been recognised recently by his appointment to CERRIE a Government committee reporting on the effects of low level radiation on health. Following his evidence to the Royal Society on the effects of Depleted Uranium, he was appointed to the UK Ministry of Defence committee on Depleted Uranium in 2001. He was invited to address the US Congressional Committee on Veterans Affairs of the Health effects of Depleted Uranium in 2002. He is presently also the Scientific Secretary of the European Committee on Radiation Risk and was commissioned to organise the preparation of the new risk model on radiation exposure and to organise the publication of ECRR 2003: The Health Effects of Exposure to low Doses of Ionizing Radiation, published in January 2003 and now translated into and published in French, Russian, Japanese and Spanish. In 2004, he (jointly with two other colleagues) published the *Minority Report of the CERRIE committee* (Sosuimi Press). In 2006 he produced and jointly edited with Prof.Alexey Yablokov of the Russian Academy of Sciences *ECRR2006 Chernobyl 20 Years On*.
Between 2004 and 2006 he was leader of the Science and Policy Interface Group of the EU funded Policy Information Network for Child Health and Environment and organised the discussions and collation of information leading to their final report on the issue which he wrote large parts of. The culmination of this project which involved over 40 scientists and physicians from all major EU countries was the recommendation that as a result of bias in scientific advice to policymnakers, all advice committees involving areas of dispute and possible harm to the public should be oppositional committees with reports including all sides of any argument.

From 2006 Dr Busby has been conducting laboratory experiments researching photoelectron emission from Uranium and elements of high atomic number. He is currently supervising research at the Centre of Molecular Biosciences in the University of Ulster on this.

1.8 RESEARCH EXPERIENCE

Dr Busby's early research was in the Physical Chemistry aspects of molecular pharmacology at the Wellcome Research Labs. This involved the use of spectroscopic and thermodynamic methods for examining cell drug interactions at the molecular level.For a while he began a research degree in NMR on molecular conformational changes on protonation but left to return to Wellcome and resume his drug interaction research. From there he moved to developing descriptions of intercellular and intracellular communication mechanisms, a subject which he is still engaged in researching in the laboratory. Later he moved to examining molecular behaviuour at charged interfaces and developed Surface Raman spectroelectrochemical method as a Science Research Council Fellow at the University of Kent.

Between 1992 and 2004 Dr Busby was engaged in reseach in three areas associated with ionising radiation and health and also was funded for a year (1997) by the *Foundation for Children with Leukemia* to research the interaction between non ionising radiation and ionising radiation. His research in the area of ionising radiation has been split between the development of theoretical descriptions of radiation action on living cells and the epidemiology of cancer and leukaemia in small areas. After 1994 he conducted survey epidemiology of Wales and England and was the first to point out (in a letter to the British Medical Journal) that increases in cancer in Wales might be related to weapons fallout. Later he examined childhood leukaemia mortality near the Harwell and Aldermaston nuclear sites and suggested that the excess risk might be related inhalation of readioactive particles. These results were also carried in a research letter in the BMJ which attracted considerable criticism. His description of the mode of radiation action from sequential emitters (his Second Event Theory was developed originally in 1987 and has attracted a great deal of interest and also criticism. Between 1997 and 2000 he was funded by the Irish State to carry out epidemiological studies of cancer rates and distance from the Irish Sea using data from Wales Cancer Registry and through a collaboration with the Irish National Cancer Registry. Following this he and his team in Green Audit developed novel small area questionnaire epidemiological methods and applied them to a number of areas in different studies which included Carlingford Ireland, Burnham on Sea in Somerset and Plymouth Devon. In addition he carried out cancer mortality small area studies in Somerset and later in Essex. He extended these to wards in Scotland in 2002. At present he is supervising a PhD student at the University of Liverpool in the Faculty of Medicine in an epidemiological study of cancer mortality in Scotland with regard to proximity to putative sources of cancer risk. In all the small area studies he carried out it was possible to show a significant effect of living near radioactively contaminated intertidal sediment. The papers and reports were all published by Green Audit and most have been presented by invitation at learned conferences in Europe including through invitations by the Nuclear Industry itself.

In addition to this, in 1998 Busby set up a radiation measurement laboratory and equipped it with portable alpha beta and gamma measuring systems including a portable gamma spectrometer made in Dresden which uses a 2" NaI detector. He used these to show the presence of Depleted Uranium in Southern Iraq in 2000 when he was invited by the Al Jazeera TV channel to visit the country as a consultant and examine the link between leukaemia in children and levels of Depleted Uranium. In 2001 he visited Kosovo with Nippon TV and was the first to show that DU was present in dust in towns in Western Kosovo and through isotope measurements funded by the BBC was able to report to the Royal Society in 2001 and theEU Parliament in Strasbourg that DU became resuspended in dry weather and was rained out, and that it remained in the environment for a considerable time. This subsequently led to UNEP deploying atmospheric particle measuring equipment in areas where DU had been used. More recently, from 2006, Dr Busby has been developing laboratory methods for measuring radiation coversion and amplification by high atomic number micron diameter metal and metal oxide particles (Uranium, Gold). It is his recent contention that such particles amplify background radiation effectiveness by photoelectron conversion and he is the author of a patent application for the use of photoelectrons in cancer therapy to destroy tumours.

In 2005 he was invited by various organisations in New Zealand to give evidence on the health effects of Depleted Uranium. In 2005 and 2006 he worked with Prof Alexey Yablokov on the ECRR2006 report on Chernobyl which was published on the 20th anniversary of the accident. Most recently he has conducted a study of the health of people living in the vicinity of the Trawsfynydd Nuclear plant in Wales for HTV and also a study of the veterans of the Porton Down human experiments in the 50s.

In 2007 he began epidemiological studies of the children of A-Bomb Test veterans and also of people living near mobile phone base stations.

1.9 INVITATIONS TO SPEAK.

Year	Place, Subject etc.
1995	House of Commons. Symposium on Low Dose Radiation
1995	Jersey, Channel Islands: International conference on nuclear shipments; Health effects of low dose radiation
1995	Oxford Town Hall: Low dose radiation effects
1995	Drogheda, Ireland: Sellafield effects
1997	Strasbourg EU Parliament: Euratom Directive
1997	Brussels, EU Parliament STOA workshop on criticisms of ICRP risk models
1997	Kingston Ontario: World Conference on Breast Cancer: paper on cohort effects and weapons fallout
1998	Muenster, Germany, International Conference on Radiation: Second Event effects
1998	Manchester Town Hall, Ethics and Euratom

1999	Copenhagen: Danish parliament: Euratom Directive and low dose effects
1999	Carlingford, Ireland: Sellafield effects
2000	Kos Island: ASPIS (EC) meeting on 'Is cancer an environmental effect'; low dose radiation and cancer
2000	London: Royal Society: low dose effects and Depleted Uranium
2001	Strasbourg: Green Group; Health effects of Depleted Uranium
2001	Bergen: International Sellafield conference, Sellafield effects on health
2001	Oslo: Nobel Institute: Health effects of low dose radiation and DU
2001	London: Royal Society: Health effects of Depleted Uranium (again)
2001	Kiev: WHO conference on Chernobyl: paper on infant leukaemia
2001	Prague: *Res Publica* International Conference on Depleted Uranium
2001	Strasbourg: EU Parliament, with UNEP; Health effects of Depleted Uranium
2002	Bergen: Conference on Sellafield
2002	Helsinki: Health effects of low dose radiation
2002	London : US Congressional Committee on National Security: Gulf war syndrome and Depleted Uranium
2002	London Greenpeace: Small area statistics and radiation effects
2002	Chilton: Health effects of radioactive waste
2002	Oxford, British Nuclear Energy Society: Effects of low doses of radiation
2002	Royal Society of Physicians: Small area health statistics and radiation
2003	Birmingham: Non ionising radiation. Chaired
2003	Liverpool University: Depleted Uranium and Health
2003	Oxford University: Helath Effects of Radiation from Internal Emitters
2003	Munich: Whistleblowers
2003	Copenhagen: Radiation and the foetus
2003	Hamburg: Depleted Uranium
2004	Berlin: Low level radiation
2004	London: PINCHE, child health and environment
2004	London, Westminster: Children with leukaemia
2004	Chicago: Radiation studies
2005	New Zealand Royal Society, Wellington
2005	New Zealand, Auckland University
2005	Chicago: Small area epidemiology by citizen groups
2005	Salzburg, Austria. PLAGE; International Nuclear Law and Human Rights
2005	Stockholm, Swedish Parliament; Low Dose Radiation and Depleted Uranium
2006	ECRR, Berlin, Health effects of the Chernobyl Accident
2006	Hiroshima Japan, Depleted Uranium
2007	Kuala Lumpur, Depleted Uranium: War Crimes Tribunal
2007	London, House of Commons: Chernobyl and health; anniversary lecture.
2007	London : Safegrounds Nuclear Industry CIRIA conference; low dose effects
2007	Blackpool: A-Bomb Veterans and low dose radiation effects
2007	University of Ulster: Childhood leukaemia in Ireland and Sellafield
2007	London, House of Commons Select Committee: Nuclear Test Veterans Children Epidemiology study

2007	London, Royal Society: Science Policy Advice and Scientific Dishonesty

Including the above, Chris Busby has given invited presentations at meetings in Strasbourg (5), Brussels (2), Jersey, Alderney, Copenhagen (2), Bergen (2), Oslo (2), Vienna, Helsinki, Muenster, Kiev, Hartford Ct, Kingston, Ontario, Baghdad, Pristina (Kosovo), Manchester (4) Oxford, Newbury (2), Cardiff (3), London (6), Prague, Dublin (2), Carlingford, Drogheda, Harlech, Bangor, Llandrindod Wells, Hastings, Weston Super Mare, Burnham on Sea (2), Bridgwater, Reading, Ulverston, Liverpool, Plymouth, Brighton, Kingston and Aberystwyth.

2. PUBLICATIONS AND SUBMITTED PAPERS

Main Green Audit published papers

Busby C and Scott Cato M (2001) *Increases in leukemia in infants in Wales and Scotland following Chernobyl: Evidence for errors in statutory risk estimates and dose response assumptions. Kiev WHO conference paper.* Occasional Paper 2001/7. Aberystwyth: Green Audit

Busby C C, Bramhall R and Dorfman P (2001) *Environmental risk methodology and Breast cancer mortality near Bradwell nuclear power station in Essex 1995-1999. Occasional Paper 2001/8* Aberystwyth: Green Audit

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Busby C, Dorfman P, Rowe H (2000) *Cancer Mortality and Proximity to Hinkley Point Nuclear Power Station in Somerset: Part I Breast Cancer*. Occasional Paper 2000/2 Aberystwyth: Green Audit

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Busby C, Dorfman P, Rowe H (2000) *Cancer Mortality and Proximity to Hinkley Point Nuclear Power Station in Somerset: Part III All malignancies, lung and stomach cancer. Summary* Occasional Paper 2000/4 Aberystwyth: Green Audit

Busby C, Rowe H (2000) *Cancer Incidence in Carlingford and Greenore, County Louth: Results of the STAD/ Green Audit Questionnaire* Report 2000/06 Aberystwyth: Green Audit

Busby C.C (2000), Science on Trial: On the Biological Effects and Health Risks following exposure to aerosols produced by the use of Depleted Uranium weapons. Invited presentation to the Royal Society, London July 19th 2000 and also given at the International Conference against Depleted Uranium, Manchester 4th November 2000.Occasional Paper 2000/10

Busby C.C (2001) ' Depleted Uranium in Kosovo: Review of UNEP Report of 13th March 2001' Occasional Paper 2001/3 *Aberystwyth: Green Audit*

Busby C.C (2001) *Health Risks following exposure to aerosols produced by the use of Depleted Uranium Weapons. Presentation to Res Publica International Conference Prague 24th Nov 2001. Occasional Paper 2001/12* (Aberystwyth Green Audit)

Busby C.C (2002) 'Review of the Home Office statement on the health Consequences of exposure to Depleted Uranium in Kosovo' Report 2002/2 *Aberystwyth: Green Audit*

Busby C.C, (2000) *Radiation from Sellafield and Cancer near the Irish Sea. The Second Annual progress report from the Irish Sea Group in support of the litigation* Short and Others vs BNFL and Others Aberystwyth:Green Audit

Busby C.C, Dorfman P, Rowe H and Kocjan B (2001), *Cancer mortality and proximity to Oldbury Nuclear Power Station in Gloucestershire 1995-1999. Including all malignancies, female breast, prostate and lung cancer mortality. With an analysis of childhood leukemia incidence in ages 0-4 between 1974 to 1990 in Welsh Areas of Residence.* Occasional paper 2001/6 (Aberystwyth: Green Audit)

Busby C.C. (2002) 'Lymphoma Incidence in Italian Military personnel involved in Operations in Bosnia and Kosovo' Occasional Paper 2002/3 *Aberystwyth: Green Audit*

Busby CC (2000) *From Sellafield to Chernobyl and Beyond: Exposure to man-made ionizing radiation as the primary environmental cause of recent cancer increases*. ASPIS (European Commission DG XVI) Conference: Is cancer predominantly an environmental disease? Kos Island September 2000. Occasional Paper 07/00 Aberystwyth: Green Audit Busby, C (1996) `Childhood Leukemia and Radiation new Newbury', Occasional Paper 96/5 (Aberystwyth: Green Audit).

Busby, C. C. (1996), 'Nuclear waste reprocessing at Sellafield and cancer near the Irish Sea: arguments for an independent collaborative study' *Occasional Paper* 96/1 (Aberystwyth: Green Audit).

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Busby C, de Messieres M and Morgan S (2006) Did Chemical Exposures of Servicement at Porton Down Result in Subsequent Effects on their Health? The 2005 Porton Down Veterans Support Group Case Control Study. First Report. Paper 2006/2 Aberystwyth, Green Audit.

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Busby Chris and Schnug Ewald (2007) Advanced biochemical and biophysical aspects of uranium contamination. In: (Eds) De Kok, L.J. and Schnug, E. *Loads and Fate of Fertilizer Derived Uranium.* Backhuys Publishers, Leiden, The Netherlands, ISBN/EAN 978-90-5782-193-6.

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2004 Translations of the above into French Japanese Russian and Spanish (see www.euradcom.org for details)

Busby CC, with Bramhall R and Scott Cato MS (2000) *I don't know Much about Science: political decision making in scientific and technical areas*. Aberystwyth: Green Audit (this book influenced the structure and formation of the CERRIE committee and advocates an oppositional structure to science advisory committees in order to allow for cultural bias in science advice. It has now been carried forward by PINCHE in Europe.).

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ARTICLES

Numerous articles for 'The Ecologist' on low dose radiation effects have been translated into most languages and reprinted.
Numerous articles and reports in *Radioactive Times: the Journal of the Low level Radiation Campaign*

BOOK REVIEWS

'Chernobyl: the definitive history', by RF Mould (Bristol: Institute of Physics): reviewed for 'The Ecologist' in 2001

EXPERT WITNESS

Since 1997 Chris Busby has been engaged as an expert witness in several cases that relate to the effects of radioactive pollution on health, in several refugee appeals (Kosovo) based on Depleted Uranium risks, several trials of activists accused of criminal damage at weapons establishment and one at the House of Commons (evidence on Depleted Uranium and other radioactive substances), one MoD pension appeals tribunal for the widow of a A Bomb test veteran and once in the Connecticut State Court for an appeal agaiunst licensisng releases of radioactivity from the Millstone reactor on Long Island Sound. He is currently acting or has recently acted as expert witness on two cases in the UK involving the health effects of internal irradiation from Depleted Uranium. One of these is in the Royal Courts of Justice and also in three cases in the USA. Two of these (against Exxon) have recently been settled. The third, a landmark case involving childhood cancer near a nuclear plant in Florida is currently being appealed in the US Supreme Court. He is also advising on the case of Rocketdyne (Boeing) and the Santa Susana Field Laboratory childhood retinoblastoma cluster in Western Los Angeles which has just been settled and also a TENORM radiation case involving Ashland Oil in Martha Kentucky.

APPENDIX D
DEPLETED SCIENCE: HEALTH CONSEQUENCES AND MECHANISMS OF EXPOSURE TO FALLOUT FROM DEPLETED URANIUM WEAPONS

CONTRIBUTION TO
INTERNATIONAL DU CONFERENCE
HAMBURG OCT 16-19TH 2003

CHRIS BUSBY PhD

Occasional paper 2003/06; July 2003
Aberystwyth: Green Audit

1. The DU story is part of a wider concern

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

Mark 4,22

Why is there concern about the health effects of Depleted Uranium? Would there be equivalent argument about the health effects following the use of Tungsten in tank shells or lead in bullets? The answer is straightforward: everybody knows that Uranium is radioactive and everyone knows that radiation exposure leads to cancer, leukemia and genetic damage. No one wants to be exposed to ionising radiation.
So why is such a weapon being used, if this is the case?

The answer is tied to a much larger and more serious issue. This is the issue of the health consequences of exposure to low doses of radiation from nuclear pollution of the planet, a subject which I have studied for more than fourteen years. The reason that DU is employed is that the weapons are astoundingly successful and have revolutionised warfare, rendering the tank and its armour useless. In addition, its use represents a route for the nuclear industry to rid itself of a waste product which would otherwise be expensive to dispose of. But the downside is that the material clearly represents a radiation hazard which is indiscriminate: battlefields are going to be contaminated and civilian populations are going to be exposed. There is an up-side and a down-side. The war will be won but the method will be illegal within contemporary accepted moral arguments. Human rights will be infringed by a randomly dispersed and thus indiscriminate radioactive weapon of mass destruction.

Since 1945, these arguments have been endlessly rehearsed for man-made nuclear pollution. First there were atmospheric nuclear weapons tests which caused global contamination with fallout, followed by pollution from the civilian/military nuclear power cycle which in the UK means pollution from Sellafield. The European Committee on Radiation Risk have recently calculated that more than 60million people have died from cancer as a result of these exposures (ECRR2003) yet Sellafield continues to operate, and nuclear power stations continue to release radioactivity to the environment. Owing to the application of false scientific models, this behaviour is sanctioned legally, and the situation is getting worse. In May 2000, the European Union adopted the 1996/29 Euratom Basic Safety Standards Directive which explicitly permits the re-cycling of radioactive substances into consumer goods.

Let us try to fit the dispersion of Depleted Uranium into this perspective. In terms of disintegrating atoms, radioactivity is measured in Bequerels. One Bequerel represents one disintegration per second. This is a reasonable way of quantifying amounts of radioactivity. The average Natural Uranium content of soil is about 10-20 Bequerels per kilogram, including all the Uranium isotopes. Most people excrete as much as 0.1mBq (0.0001Bq) per litre of Urine as a result of absorption of natural Uranium in food they eat. Pure Depleted Uranium contains about 12,400,000Bq of U-238 per kilogram and in Kosovo, some soil samples analysed by the United Nations Environment Program (UNEP) contained 250,000Bq/kg (UNEP 2001, Annex). The 350 tonnes of DU used in the first Gulf War represents 4.3 TBq (4.3×10^{12} Bq) of Uranium alpha activity (13.0 x

10^{12} if the radioactive beta emitting daughter isotopes are included-more of these below). If Dai Williams (2003) is correct and about 1700 tonnes were used in the latest war, then that represents 63 TBq of activity dispersed mainly into a populated area of perhaps 100km^2 . This gives a mean density of deposition of radioactivity of 630,000Bq/m^2. These sums are instructive and are collected together in Table 1.

These activity comparisons are given just to get some feel for the amounts of radioactivity involved, and to show that the dispersion of Uranium in various recent battlefields is not trivial, as the military and some politicians regularly imply. But the comparisons are slightly misleading because we are not dealing with the same isotopes as were released by weapons fallout which is composed of alpha beta and gamma emitters. Battlefield DU fallout is in the form of microscopic alpha and beta emitting particles. U-238 is an alpha emitter. The U-238 daughters, Protoactinium-234m and Thorium-234 are beta emitters. Having short half-lives, they are in equilibrium and therefore have the same level of activity in a sample of DU. In an area contaminated by DU it is the beta radiation that is detected because it has a range in air of about 30cm unlike the alpha particles which are very short range.

We can find a better comparison for DU. As an alpha emitter and long lived environmental particle DU is more comparable with Plutonium-239, a substance released by Sellafield and a major contaminant of the Irish Sea. Plutonium in the environment is also in the form of micron sized oxide particles.

Table 1. Mean density of deposition of radioactivity from DU in the two Gulf Wars and Kosovo including decays from U-238 and beta daughters Pa-234m and Th-234 compared with other radioactive contamination.

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

** I measured 4000Bq/kg in Gjakove, Western Kosovo, in Jan 2001 in a car park, but these values are averages based on an assumption about the area into which the material has been dispersed.*

Like DU, these Plutonium Oxide particles are also long lived and mobile. Plutonium from Sellafield has been measured in autopsy specimens across the UK, in sheep droppings on the east coast of England 100 km from Sellafield at the same latitude and even in the teeth of children up to 200 km from the site in south east England. Both Uranium-238 and Plutonium-239 are alpha emitters, although Plutonium has no beta emitting daughter isotopes in SECULAR equilibrium. U-238 has a very long half life, 4500 million years, so owing to its much shorter half life of 24,100 years, the specific activity of Pu-239 is far greater. It is 2.3TBq/kg. But this means that 350 tons of DU (or 4.30TBq of U-238) is equivalent in activity to about 2 kg of Plutonium-239. What would governments of the world say to a war in which one army caused the intentional scattering of 2kg of Plutonium-239 over a populated area? What would the ethicists and moral philosophers say? Or ordinary members of the public? What would happen in New York or in London if 2kg of Plutonium-239 was dispersed among the public? The emergency services are geared up in the UK to evacuate whole cities if such a 'dirty bomb' was exploded by terrorists. Actually, for reasons which I shall enlarge on, in terms of health deficit, what has been done in Iraq and Kosovo, possibly also in Afghanistan is much worse. Yet nothing is said by the regulatory authorities. Worse than this: they develop models and enrol scientists in an attempt to minimise any perception of harm and routinely deny or marginalize evidence that shows that the use of DU has had major and serious effects. I compare U-238 and Pu-239 in Table 2.

Table 2 Comparing Plutonium-239 and Uranium-238 in the environment

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

I have compared Plutonium and weapons fallout with DU to demonstrate that we are dealing with the same problem, the health effects of low level exposure to radioactive substances that irradiate our bodies from the inside. The weapons fallout, and other pollution from nuclear sites like Sellafield has been responsible for the present cancer epidemic, the one that everyone has experienced. It has been a major project of the nuclear military complex, and for governments who have been involved in releases of radioactivity, to cover up the link between these exposures and cancer or other ill health. This is why all these committees are controlled and steered by the same people. Recognition that DU caused cancer, leukaemia or lymphoma at the doses experienced by those who were contaminated after its use would lead to inevitable recognition that the

weapons fallout substances, the Strontiums and Plutoniums and Caesiums also caused cancer, leukaemia and lymphoma. The reverse is also true. Recognition of the cause of the Sellafield lukemia/lymphoma cluster would lead to reassessing the risk models to the point where it would be clear that DU would have serious health effects. This is the origin of a massive cover up which extends to the cancer registries and the cancer research organisations.

2. Green Activists

First they laugh at you, then they attack you, then you win.

Gandhi

The truth about the health effects of low level radiation has been covered up by the nuclear /military lobby in many ways for about 50 years. I wrote about this in *Wings of Death* (Busby 1995) and there I explained how different levels of control and bias had been employed to keep the public from realising that they were being systematically poisoned by radioactivity. Others have made this point. John Gofman, once a very senior figure in the nuclear establishment put it well: *the nuclear industry is conducting a war against humanity*. Part of the reason behind the success of this cover-up has been that the process has been tied in with Military and State security in the countries that have nuclear weapons. The process extends to the highest levels. The World Health Organisation (WHO) is tied to the International Atomic Energy Agency by a 1959 agreement which prohibits them from researching the health effects of radiation. This is why we hear that there have been no increases of cancer due to Chernobyl. This is why the WHO take the view that DU is not a health problem. This why the European Commission adopt the EURATOM safety standards and the radiation safety laws are predicated on the advice of the ICRP, a self selected and unaccountable organisation that is part of a network of revolving doors in which the same people pass in and out saying the same things and agreeing with one another.

From very early on I felt that to change this situation a scientific analysis was not enough. There had to be a political analysis as well, and particularly an analysis of power. The power of the nuclear/ military establishment lies in institutions rather than in money. It is these institutions that lend credibility to their position. Increasingly, though the liberalisation of universities and their research funding, it is the grants that drive the direction of science and formulates its current 'Truths'. It is not the quality of the research that decides whether it is published and eventually influences policy. It is the acceptance of the research results into the required institutional view. If you write a scientific paper and the editors or their referees don’t like it, they reject it. You are not told who the referees are. For the Green Activist, who wishes to change this, the answer then is to ignore these institutions and create new ones. What is the point of sending rigorously argued manuscripts to scientific journals if these journals are controlled by the nuclear industry scientists, those they support with research grants and money?. What is the point of sending out Press Releases to the media if they are put in the waste bin?

As a result of the Green Activist approach, the Low Level Radiation Campaign has persuaded the UK government to set up a new committee to examine these effects. We pointed out, following the 'Mad Cow Disease' committee failings that the only way to

get to the truth in science advice was to fund both sides and have them argue the case out in committee. The first committee of this kind is this new Committee Examining Radiation Risk form Internal Emitters (CERRIE, www.cerrie.org). Here, there are scientists from both sides of the debate on low-level internal radiation arguing out the various pieces of evidence that the ICRP risk model is in error and that internal radiation exposure, like that from fallout, from Plutonium, or DU represents a serious health hazard. CERRIE reports finally in 2004, but its preliminary report was considered at an international workshop in Oxford in July 2003. The report drew attention, for the first time, to the existence of major scientific uncertainties in the area of risk from internal radioisotopes.

There is one other independent institution which I helped to set up. This is the European Committee on Radiation Risk, based in Brussels (www.euradcom.org). This committee was intended as an alternative ICRP. It has over 40 independent experts in radiation risk, mainly from Europe and the ex-Soviet Union but some from the USA also. It includes ethicists, doctors, physicists, geneticists, biologists, politicians and philosophers. Together with Prof. Inge Schmitz-Feuerhake and Prof Alexey Yablokov, I launched the ECRRs new radiation risk model in Brussels on 30th June [ECRR2003]. The model incorporates weightings factors for internal radiation exposure. These are based on arguments and evidence which I shall examine now. For DU the weightings are as high as 1000-fold

Let me now concentrate on reviewing where we are in the investigation of Depleted Uranium.

3. The health effects of internal irradiation by man-made radioisotopes and new forms of natural isotopes.

I will summarise briefly here the theoretical and epidemiological evidence that the ICRP external model is in error by orders of magnitude when used to predict or explain the consequences of internal irradiation. A fuller explanation is given in ECRR2003.

3.1 Theoretical considerations

External radiation produces ionization tracks in tissue that are uniformly distributed. Thus each cell receives on average one track per year and the linear dose response used by the ICRP to predict cancer from the Hiroshima survivors breaks down if there is more than one track intercepting a cell in the time it takes for the cell to repair damage, about ten hours. For internal sequentially decaying isotopes and for internal long lived, hot (or warm) particles the probability of a cell local to the internal decay receiving two or more hits is very much higher than the equivalent probability for the same dose delivered externally. There are two consequences. The first is that the cell response is in the 'dose squared' region of the accepted ICRP model and the dose response in no longer linear. This is because the probability of a DNA double strand break occurring increases sharply for two or more hits to the cell. Such a lesion carries a high degree of certainty that a fixed mutation will follow. The second possible consequence is that the first hit to the cell will either induce a repair replication cycle in the hit cell, or if the cell is killed, in local cells which will begin to replicate to supply a replacement. Whilst replicating and repairing the initial lesion, a second hit at the critical point in the replication process will cause a fixed unrepairable mutation. This is the second Event Theory. There are further problems with internal isotopes which relate to their chemical affinity for DNA. Both

Strontium (e.g. Sr-90, Sr-89) (Sr^{++}), Barium (Ba-140, an Auger emitter) and Uranyl UO_2^{++} ions bind strongly to DNA (Wu et al, 1996) and so their decays will be extremely hazardous since they are localised near the target of interest. Work with the covalently bound Auger emitter Iodine-129, and also manmade Auger emitters like Cr-59 with bind to DNA show that these localisation effects carry very high risks which are not modelled by their apparent average doses. U-238 itself is an Auger emitter (31 % decay 10keV) and the high concentration gradient of of UO_2^{++} ions near the surface of a UO_2 particle would result in a high level of DNA localisation near the particle. Particles are, of course, highly likely to cause second event and multiple hit effects to nearby cells and the local doses from DU particles are considered below.

In the last ten years, evidence has emerged that low doses of radiation cause genomic instability in cells that are hot, but also in cells that are near the cells that are hit, up to about a 300 cells radius. Using computer controlled microbeams, individual cells can be targeted and the effects in nearby cells counted using various endpoints. In all these experiments, the dose response is very clearly non linear and increases sharply up to two or three hits per cell when it saturates. Miller et al (1999) have shown that cancerous transformation is almost exclusively caused by two hits rather than by one hit/ the effect for chromosome aberration as an end point seems to saturate after three hits (Prise et al, 2002). The cell volumes around damaged cells respond to the damage through a communication field, and therefore it is the location of radiation doses and ionisation effects within this field that is important in establishing future effects in the tissue like cancer. It is clear that physics no longer informs us of the effects of radiation at the cellular level. The key problem is that the evidence shows that concentration of ionization in a small volume of cells, or inside a single cell results in very high yields of mutations. It is high local ionisation density that is important, not dose; but this fact has been obscured by experiments with such high densities of ionisation that cell killing is the result. This is why the hot particle experiments show such equivocal results. These new discoveries in biology make a nonsense of the basic science underpinning the ICRP averaging models and therefore we have to look to appropriate epidemiology to see what the health consequences of exposure to these novel isotopes and forms are. To use epidemiology of externally irradiated groups to inform on internally irradiated groups is not using scientific method (Busby 2001 RS, Busby 2002 BNES).

3.2 Epidemiological considerations

If we cannot extrapolate from external radiation and Hiroshima, and we cannot use linear no threshold dose responses to mathematically model health effects where does that leave risk assessment? The scientific answer is that we have to look at the effects themselves and use them to define risk. This is done by epidemiology of populations exposed to the radiation sources we are interested in. It is not good enough to say that the model does not predict the cancers, as the risk agencies said about the Sellafield leukaemia cluster. If the models is theoretically unsound, we must re-examine the issue and consider whether the cancers were caused by the radiation. When we do this for the famous Sellafield child leukaemia cluster, we find that the error in the ICRP risk model needed to account for the cancers is about 300-fold. Looking at the other leukaemia clusters the error needed to explain the cancers is between 300-fold and 2000-fold. This may seem like an enormous error, but if it consistently turns up, we should as scientists begin to look at how it can

occur. Tamplin in 1972 examined hot particles of plutonium and concluded on the basis of theoretical assumptions that they, were more hazardous that the ICRP model suggested by a factor of 115,000, so these large numbers are not as silly as they may seem. They essentially represent the difference between local dose to tissue and averaged dose to body from a hot particle. And since it is the tissue that develops the cancer over a long period by amplification through cell division of various DNA lesions, it is not surprising that it is the tissue dose that is important, and not the whole body or whole organ dose.

Although many studies of nuclear sites, downwinders, and other contaminated individuals have pointed to large errors in the ICRP model (see Busby 1995 and the web site: www.llrc.org) it was only after Chernobyl that we were able to obtain sufficiently unequivocal evidence. Despite the cover-ups in the ex-Soviet territories and the efforts of the cancer agencies (e.g. IARC, IAEA, WHO) to deny any effects two sets of evidence emerged which falsified the conventional position that the only effects of Chernobyl were the deaths of a few liquidators and some thyroid cancers. There were two pieces of evidence that forced the UK government into a reappraisal of the issue of internal radiation. The first was the Chernobyl infants and the second was the minisatelite DNA mutations.

3.3 The Chernobyl infants

Following the Chernobyl accident in 1986, the cohort of children who were exposed in their mother's womb to radioisotopes from the releases suffered an excess risk of developing leukaemia in their first year of life. This 'infant leukaemia' cohort effect was observed in six different countries. It was first reported in Scotland [Gibson *et al.*, 1988], and then in Greece [Petridou *et al.*, 1996], in the United States [Mangano, 1997] and in Germany [Michaelis, *et al.*. 1997].

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

Because the National Radiological Protection Board had measured and assessed the doses to the populations of Wales and Scotland and because they themselves had also published risk factors for radiogenic leukaemia based on ICRP models it was a simple matter to compare their predictions with the observations and test the contemporary risk model. The method simply assumed that infants born in the periods 1980-85 and 1990-92 were unexposed and defined the Poisson expectation of numbers of infant leukaemia cases in the children who were *in utero* over the 18 month period following the Chernobyl fallout. This 18 month period was chosen because it was shown that the *in utero* dose was due to radioactive isotopes which were ingested or inhaled by the mothers. Whole-body monitoring had shown that this material remained in the bodies of the mothers until Spring 1987 because silage cut in the Summer of 1986 had been fed to cattle in the following winter. The result showed a statistically significant 3.8-fold excess of infant leukaemia in the combined Wales and Scotland cohort ($p = 0.0002$). The leukaemia yield in the exposed *in utero* cohort was about 100 times the yield predicted by the ICRP model. Table 3 compares the effect in the three main studies. In this table, the B cohort were those children exposed to the internal exposure from Chernobyl *in utero* in the 18 month period following the event and born between June 1987 and January 1988.

These exposure periods were defined by the whole body monitoring results. The control periods A and C were the ten years before (1975-85) and the four years after 1988 for which data was available.

The possibility of the effect being due to chance may be obtained by multiplying the p-values for the null hypothesis that the effect was due to chance in each of the separate countries to give an overall p-value less than 0.0000000001. Thus it was not a chance occurrence: it was a consequence of the exposure to low-level radiation from Chernobyl.

The infant leukaemia results represent unequivocal evidence that the ICRP risk model is in error by a factor of between 100-fold and 2000-fold for the type of exposure and dose, the latter figure allowing for a continued excess risk in the cohort being studied.

Table 3 Unequivocal evidence of ICRP risk factor errors: comparison between infant leukaemia rates after Chernobyl in Wales and Scotland and similar data from Greece and from the former Federal Republic of Germany

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

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

3.3 Minisatellite mutation rates in Chernobyl children

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

Although subtle genetic effects on sex ratio were apparent in the LSS offspring, the RERF researchers excluded them from the study because they did not accord with their notions of the expected direction of such an effect [Padmanabhan, 1997]. Neels's exclusion of the sex ratio effects resulted in the belief that the genetic effects of 10mSv in the first generation would be unmeasurable. Thus BEIR V gives the incidence of total genetic effects including chromosomal effects (unbalanced translocations and trisomies) at 6 per million offspring compared with the natural rate of 4,200. It predicts a 10mSv

excess risk of 10 cases of congenital malformation in a natural rate of 25,000 per million offspring and similar vanishingly small increases are given for autosomal dominant, X-linked and recessive disorders. Using a combination of mouse studies and the epidemiology of the LSS, the doubling dose for spontaneous genetic burden has been estimated to be 1 Sievert. [e.g.BEIR V, 1990 p 70]

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

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

These results follow the use of a new objective analytical method fro examining individuals who have been exposed. In this sense they cannot be subject to the arguments used against epidemiological studies. The mutations are there and are measurable so there can be little argument. The doses are known and the comparison is safe. It shows a large error in the ICRP model and raises many issues relating to the overall outcome of irradiating human populations.

I will now turn to the effects of DU.

4. The health effects of Depleted Uranium

I want to consider the DU case under four headings. They are:

- The nature and dispersion of DU and its routes for human contamination.
- Theoretical radiation biology effects and science.
- Evidence of harm at the cellular level
- Evidence of harm from epidemiology

3.1 Particle doses and hot coals

To recapitulate, the ICRP model is the presently accepted risk model for radiation and health. It is based on the idea that radiation is external to the body. Examples of external radiation exposures are medical X-rays and gamma rays from atom bombs. The ICRP model bases the amount of ill health produced by doses of radiation of different sizes on a large study of the Hiroshima survivors. These people received a very large dose and some of them were incinerated. But among those that were not, some of them developed cancer much later on. The ICRP model relates the numbers of cancer to the large dose they received and argues that at half this dose there should be half the cancers and so forth. So if the dose is very small, there are very few cancers. The problem is, that this model is not strictly applicable to internal radiation. Absorbed dose, in Grays or Sieverts or rads or rems is measured as energy per unit mass. Therefore it would not distinguish between a man warming himself in front of a fire or the same man eating a hot coal. The average energy per unit mass is the same. This a good analogy for why the DU or plutonium situation is wrongly modelled. In the case of DU particles the decay energy is all absorbed in the local cells. So one single particle will give a big dose to the local cells and no dose to the rest of the body. The ICRP will say that the dose is very small, but because the alpha decay range is small, the dose to the cells nearby, is very large. This is a trick and I show how it is done for a 2 micron diameter particle of DU trapped in the lymphatic system of a person who inhaled it.

The calculation in Table 4 shows the dose to the tissue within range of the particle alpha decays and the dose to (a) the whole body and (b) the lymphatic system that NRPB and ICRP would calculate. [see e.g. NRPB, R-276 p 86 1995) The NRPB reference is to actual calculations made by NRPB on the doses from Plutonium particles to the public near Sellafield. Two things are immediately apparent. The cells close to the particle receive a significant dose and they also suffer an enhanced risk of receiving multiple tracks. The dose calculated by the ICRP model is vanishingly small, so it is easy to see how the Royal Society, the Ministry of Defence, the United Nations, the IAEA/ WHO say that DU cannot cause any cancer.

Table 4. Doses to local tissue within range of a 2 micrometer particle of DU compared with doses calculated using the ICRP model and an NRPB version of it.

	Value	**Comment**
Uranium oxide U_3O_8		
Density	8.6	
Decay energy/Bq	4.45MeV = 7.12 x 10^{-13}J	
Particle diameter	2μ (2 x 10^{-4}cm)	Common size
U-238 mass in particle	3.05 x 10^{-11}g	
Particle activity	3.79 x 10^{-7} Bq	
Mass of 30μ radius sphere of tissue (ρ = 1)	1.13 x 10^{-10}kg	
Dose to this tissue per Bq	6.3mGy	
Equivalent dose	126mGy	
Hits to tissue per day	**0.03 α–and .06 β-tracks per day**	**11 α– tracks per year and 22β– tracks**
Equivalent dose to this tissue per day	**4.12mSv**	Or **1500mSv per year**
NRPB calculated equivalent dose to 'lymphatic system' per day	**5.8 x 10^{-11}mSv (effectively no tracks)**	*Assumes 8kg or **2.1 x 10^{-8}mSv per year**
ICRP calculated equivalent dose to 'lymphatic system' per day.	**5.8 x 10^{-10}mSv (effectively no tracks)**	**Assumes lymphatic system as 800g (ICRP) **2.1 x 10^{-7}mSv per year**
ICRP calculated dose to tracheobronchial lymph nodes per day	**3.1 x 10^{-8}mSv (effectively no tracks)**	**TBN Mass = 15g **1.1 x 10^{-5} mSv per year**

*for lymphatic system modelled as lymph nodes, liver, spleen, kidneys, pancreas, uterus, thymus, thyroid, stomach, both intestines, colon, red bone marrow and cells on bone surfaces [NRPB, 1995]
** values from ICRP standard man [ICRP23, 1975]

3.2 Borrowing radiation energy from background: second order scattering
There may be a second source of error here although it is difficult to quantify. Uranium is very dense and the particles have an enormous combined surface area. It is possible to calculate that for the smaller particles of 0.2μ diameter a 5mg inhalation loading represents some 10^{11} particles with a combined surface area of about 250cm^2. Now small particles smaller than the wavelength of incident scatter incident radiation so that the particles act as secondary scatterers for the gamma rays from natural background radiation or medical X-rays or other internal emitters including other local particles. In addition, the lower energy component of this radiation, below 100keV photon energy will quantitatively be converted into photoelectrons from the particle surfaces. These are short-range highly ionising electrons which will increase the ionisation density in the immediate vicinity of the particles. This effect is increased because Uranium happens to have a very low photoelectron work function and even releases electrons when irradiated

by UV and visible light so that Uranium salts are light sensitive and can be used for photography. In addition the release of photoelectrons from the particle surface will cause it to acquire an electric charge and attract negative ions which will perturb the biochemistry taking place close to the particle with unknown consequences. None of these considerations are included in the ICRP model.

3.3 Particle environmental dispersion

The military and other authorities have dismissed the possibility of widespread dispersion of DU particles. The US Department of Defense papers make this claim but have not been able to justify it. The particles of less than 2μ diameter are easily resuspended by wind or by electrostatic repulsion in the earth's electric field. In addition they become charged by photoelectric effects owing to the low Uranium work function (see above) and these charges would assist their resuspension although no experiments have been done to my knowledge. I discovered DU dust in western Kosovo one year after the war. It was in road dust at several sites under conditions where it was clear that the material had been washed out by snow. In addition the ratio of activity of the beta emitting daughter isotopes to the parent Uranium-238 showed that the U-238 was being preferentially resuspended. I gave this information to the Royal Society but their experts said that mathematical models showed that DU particles could not be resuspended and would remain where the targets were a few metres from the site of impact. I also gave a paper on this at a meeting organised in the European Parliament on DU. At this meeting I asked the head of UNEP, Dr Snihs why UNEP had not examined air filters in their November 2001 survey of Kosovo. He stated that the DU would not widely disperse and would not be found in the air so there was no need. However, I note that UNEP did deploy air measuring equipment later in Bosnia and Montenegro. This equipment detected DU in the air. The UNEP response was that the material had been resuspended by their disturbing of the soil. The UNEP Kosovo report tabulated the presence of DU in 46% of all the samples they measured but the tables were not given to the Press at the launch of the report in Geneva and the executive summary says there is no widespread dispersion of DU. If you read the report closely, their definition of widespread dispersion is of DU which would be a cause for concern in health terms, a qualification that was lost on the journalists. Here again is an example of spinning a report . Since the results tables were not given out (and have since disappeared from the report on the website) no one was able to argue the point. For those who are interested, I have a copy of the UNEP Kosovo tables and have written a critique of the whole way the results were presented. The study also showed the presence of DU particles larger than 0.2μ in a rainwater pond in Vranovac (Busby 2001).

I also found widespread DU in southern Iraq when I visited there in September 2000, or rather, I found areas of high beta counts on the ground in the area of the 'Mother of All Battles' and saw a few A10 penetrators lying on the ground also. In Iraq, I found significantly higher alpha activity in the air in this area. Unfortunately the Iraqi authorities would not let me remove any samples.

3.4 Human contamination and biokinetics

Shortly after my visit to Kosovo in January 2001, Prof Nic Priest visited the same region with BBC Scotland and took urine samples from some 20 people including his BBC

cameraman. Priest has access to sophisticated mass spectrometry equipment and can measure Uranium isotope ratios in urine. He found that all the urine samples were contaminated, including the cameraman Donald Macleod who had only been there for five days. These results have now been published (Schroeder et al, 2003) and they show conclusively that the people in the area are contaminated with DU. We also have the results of measurements on the urine of Gulf War veterans by at least three teams. All show the presence of DU in the urine some ten years after the exposure.

The only way that this could happen is that there remains in these people some depot or store of DU which is slowly leaching out. At the time of the Royal Society first report the biokinetic models of DU were based on the studies of natural Uranium in animals. It was conceded that DU particles were extremely insoluble and had a very long half life in the body after inhalation. Recent studies [Ansoborlo et al, 2001] show half lives for the inhaled ceramic U_3O_8 and UO_2 particles to be of the order of 5000days or 13 years.

If this is so, then the amount excreted per day in the 11th year after the initial loading can be determined from an exponential decay equation such as:

$$M = M_0[\exp(-0.693t_d/T_{1/2})-\exp(-0.693(t_d+1)/T_{1/2})$$

This gives a fraction of 0.03 of the initial loading being lost in the 11th year and a daily excretion of 8×10^{-5} (divide by 365) of the initial loading. So for an initial loading of 5mg, assuming a 10% translocation through lung and a 50% insoluble fraction there should be about 20ng a day of DU excreted in the urine if this half life is correct. However, it is not at all clear that there may not be material that has a very much longer half life, or more likely that with such high levels of insolubility the concept off half life breaks down and there remains DU trapped in certain tissue for the lifespan of the individual which does not relate to the measured concentration in the urine. If, for example 20% of the initial translocated material were trapped in the tracheobronchial lymph nodes and entirely inaccessible to dissolution and transfer to the greater system, this would leave 100μg of DU in an organ with a mass of a few grams irradiating cells over a period of ten or more years. We can calculate that this represents 2×10^9 particles of 0.2μ diameter, about one particle for each cell in the lymph nodes. For even if the DU were trapped, the photoelectrons and beta or alpha particles would still cause damage to DNA in cells which were local to the trapped material. And uncertainties in the rate equations as applied to urine measurements over the periods involved in animal studies (mice live a less than two years) would easily accommodate such a situation, so we should be cautious about using the results of urine tests to work back to initial contamination or its effects.

For 1 μ diameter DU particles biokinetic models employed by the Royal Society based on the ICRP66 human respiratory tract model suggest that 10 years after inhalation there would be a daily excretion of about 10^{-7} of the original loading but I have been unable to replicate their calculations. (Royal Society 2001).

Since levels of 20ng have been reported for UK Gulf veterans some 10 years after their contamination, the value of 5mg may be a reasonable assumption for their initial contamination on the bases of my calculations.

3.5 Chromosome aberrations in Gulf Vets

The question of the levels of exposure and the level of resultant damage has been informed by an important set of measurements of chromosome aberrations in the peripheral lymphocytes of a group of UK Gulf War veterans organised by Albrecht Schott. These results have now been published [Schroeder et al 2003]. It is possible to compare the levels of chromosome damage with the many earlier studies which related chromosome damage to earlier radiation exposure and conclude that the veterans received between 50 and 200 mSv. I have used a recent review of the relationship between chromosome damage and dose to back calculate [Hoffmann and Schmitz Feuerhake 1999] . The best value for the fraction of dicentric chromosomes (DiC) per cell per mGy obtained by regression is 5.21×10^{-5}. The Gulf veterans group showed a mean fraction of 0.0027 DiCs compared with 0.0005 in the controls. This suggests a mean dose for the group of 50mGy in the previous year which I assume must be from the 50% of the DU still in their system. For a relatively high 50mg initial loading in 1991 and 5mg getting through the lung we can calculate the mean ICRP dose to the 800g lymphatic system in the two years prior to the chromosome test.

It is vanishingly small: about 1.4×10^{-3}μGray. This suggests an enhancement of the radiation effect of about 500,000. (100,000 is the value that Tamplin calculated in 1971 for the enhancement of effects from hot particles).

On the other hand, comparisons with chromosome aberration studies of Chernobyl NPP workers who had film badges and therefore had recorded external doses [Shevchenko et al 1996] suggest more like 500mSv.

A value for particle dose effectiveness enhancement of 1000-2000 was adopted by the ECRR for their weighting factor for particulate DU enhancement in the recent 2003 report but this may be a conservative value. Something seems to be going on here that is not adequately captured in present models and it may be that the ideas about scattering and secondary effects from background exposures need to be examined more closely. Such experiments would be easy to perform. However, these results do suggest that there should be increases in somatic genetic and heritable genetic damage and cancer in such individuals. Since the doses are mainly to the lymphatic system, some form of leukaemia or lymphoma would be the first evidence of such an effect.

3.6 Epidemiology

3.6.1 Iraq

The first reports of cancer and leukaemia came from Iraq. I was invited to the country in 2000 and met with senior health officials in Baghdad and Basrah. I examined cancer statistics from the Iraq cancer registry. There were sharp increases in leukaemia and lymphoma indicated, particularly in children born around the time of the 1991 war. The Iraqis have been accused of making up their cancer figures. However, there are pieces of data that they would not thought of making up. The main problem with cancer data epidemiology is the population base. After a war, people are killed and move about the country; there are massive population upheavals. But you can still look at the cancer numbers and assume they are a sample from an unknown population. Then you can make comparisons within the sample. For example, we can look at the numbers of cases of childhood cancer in the period 1995-1999 [Iraqi Cancer Registry, Baghdad 1999]. I show some data in Table 4 for male children (I should really say little boys) where I

compare the numbers of cancer cases with those expected on the basis of the England and Wales rates for the same cancers and in Table 5 show the relative risk in the war birth cohort, those aged 5-9 in 1995-99. This calculation uses the rates in England and Wales to calculate the expected numbers of cases in each age group is the Iraq children had the same rates as the England and Wales children.

Table 4 Male childhood cancer in Iraq, 1995-1999 (Source; Iraqi cancer registry, 1999)

Cancer site	**Male 0-4** Iraq, numbers *England and Wales* **numbers (rates)*	**Male5-9** Iraq, numbers *England and Wales* **numbers (rates)*	**Male10-14** Iraq, numbers *England and Wales* **numbers (rates)*
Lymphatic Leukemia	69 *69 (7.1)*	112 *31 (3.2)*	70 *25 (2.6)*
Non Hodgkins lymphoma	58 *58 (1.0)*	82 *75 (1.3)*	53 *75 (1.3)*
Hodgkin's Disease	7 *7 (0.3)*	52 *12 (0.5)*	42 *11 (1.5)*
All Cancer	279 279 (19.8)	399 171 (12.2)	354 158 (11.2)

Table 5 Relative risk of leukaemia, lymphoma and all cancers in the male children born at or just after the Gulf War in Iraq.

	Observed	**Expected**	**Relative Risk (p)**
Lymphatic leukaemia	112	31	3.6 (<0.0001)
Non Hodgkins lymphoma	82	75	1.09
Hodgkins disease	52	12	4.3 (<0.0001)
All cancer	399	171	2.3 (<0.0001)

We can conclude that childhood cancer increased in the war birth cohort. The effect was driven by lymphatic leukaemia and Hodgkins disease, which is a cancer of the lymphatic system. As to the accusations of inventing the data to make a political point, there would be more mileage in making all the leukaemia numbers large immediately after the war. In fact, this was not done, although figures for different districts show a correlation in increased in adult leukaemia with the areas where DU was mostly used.

3.6.2 The Italian Kosovo Study

The question of whether there has been an increase in leukemia/lymphoma or other cancers in occupants of or peacekeepers deployed in the Balkans has been a source of argument of a similar order and type as the question of increases in leukemia/ lymphoma and birth defects in Iraq. In the case of the Balkans, there is very little hard evidence (e.g cancer registry data) which is available for independent scrutiny, and indeed some of the problems associated with the kinds of population movements that follow a major conflict would make such analyses very difficult. There was been a leak of a table of cancer

incidence in Sarajevo from the cancer registry there which suggests a more than 10-fold increase in leukemia and lymphoma even allowing for a doubling in the base population. This information was given to the Royal Society as evidence last year but was not included in their report or followed up by them [Busby 2002]. In addition , there has been anecdotal evidence of increases in leukemia/lymphoma in the Italian and Portuguese peacekeepers and these have led to misleading statements from the authorities. Recently, in a letter to Caroline Lucas, MEP, a UK government minister, Dr Lewis Moonie suggested that 42 leukemia deaths per 100,000 peacekeepers was a reasonable sum and that therefore the handful of deaths observed should be seen as a normal situation. However, Moonie should certainly know better than to try on this rather silly attempt to blind us with numbers. It was easy to show that the 42 was a ridiculously incorrect number based on people of all ages and that the true figure (based on the actual age group of 20-40) defined a significant excess risk of about 1.5 deaths in every 100,000 persons.

In January 2001, Nippon TV who took me to Kosovo were told of there were 7 leukemia deaths in Italian Kosovan peacekeepers (assume 50,000) and more recently Eddie Goncalves, a journalist in Portugal, reported 5 deaths from leukemia in the Portuguese Kosovan peacekeepers (5 deaths in 10,000 with two in the 20-30 age group). Thus in those groups we observe 12 leukaemia deaths where 0.9 are expected, a relative risk of 13. Even if we use a two-year period since the war the Relative Risk is still 6.5

But in May 2001 the Italians commissioned a proper epidemiological study of their peacekeepers from Kosovo and Sarajevo [Italian report, 2001]. The study of 39,491 persons found a significant excess risk from Lymphoma, particularly Hodgkins. The results are shown in Table 6.

Table 6. Expected and observed numbers of lymphoma cases in Italian DU study group with statistical significance based on cumulative Poisson probability.

Disease	**Expected**	**Observed**	**Risk Ratio**	**Poisson p-value**
Non Hodgkin	4.1	4	0.97	NS
Hodgkin	3.38	10	2.95	0.003
Lymphoma	7.48	14	1.87	0.02

I obtained this study through the Italian Greens and used the data given to calculate the true relative risk after allowing for the' healthy worker effect'. I could use the ratio of lymphoma to all cancers to show that the true excess risk was RR = 7.5. So the Italian veterans had a 7.5-fold excess of lymphoma, mainly Hodgkins disease. The interesting aspect was that the disease had emerged a very short time after the exposure, a year or two. I gave paper on this to the Ministry of Defence DUOB.

3.6.3 Cancer in the UK Gulf Veterans

The UK government have been very poor at examining the health effects of DU. But various questions have been asked in Parliament by individual MPs and the Gulf Vets themselves and non-Governmental Organisations like the Low Level Radiation Campaign have put pressure of the Ministers to investigate risk. The MoD set up a Gulf Veterans Illness Unit and these people produced a report in November 2002 which compared deaths in all Gulf Veterans compared with deaths in a matched control group

who were not deployed in the Gulf. Results show that there were 19 deaths from leukemia and lymphoma combine compared with 11 in the control group.
This is a statistically significant finding (p = 0.018) but nothing was said about the finding, and my attempts to obtain a breakdown by type of cancer have so far failed.

4.The US Department of Defense.
Because this paper is about the ways in which the establishment attempt to dismiss concerns about DU I will now turn to a widely quoted report about DU in the Balkans. This is the US Department of Defense report, *Depleted Uranium Environmental Surveillance in the Balkans.*[US DoD, 2001]. The UK government Home Office use this report to justify their own position on repatriating refugees to areas of Kosovo where DU was used, and as a result of various appeals cases I have had to study the DoD report quite closely. I produced a critique for the Appeals Tribunal in 2002 [Busby, 2002]. The DoD document makes two assertions and bases these on 83 references, apparently to independent scientific work. The assertions are:

- The studies undertaken on DU in Kosovo have not detected any significant levels of DU.
- Studies have not shown any significant risk to health of the population of the province from the presence of DU.

As I demonstrate, here and in other papers, both of these statements are incorrect. But all I wish to observe here is that the references on which the DoD report is based are almost all references to a NATO website or other NATO reports. I show the distribution of the sources of the conclusions of the DoD report in Table 7.

Table 7 Distribution of the sources of the conclusions about DU in the Balkans: Number of citations of specific sources in the 2001 Department of Defense report on DU in the Balkans [USDoD, 2001]

Source	**Number of citations in DoD**
NATO website	18
NATO report AHCDU-N (2001)38, April 3rd 2001	30
NATO letter IMSM-164-01, March 5th 2001	15
Royal Society Report, May 22nd 2001	4
UNEP environmental reports, Oct 1999, May 2001	3
WHO, DU report, April 2001	6
EC Article 31 group, March 6 2001	1
Available independent relevant studies	1
Peer reviewed studies	None

My conclusions are that the position taken by the establishment is not based on science, but on wishful thinking. The NATO website and other NATO documents are reports of NATO meetings where everyone agreed that there was no problem. These positions were informed by a few meetings where military investigations agreed there were no problems. Other reports of the results of environmental surveys found no DU. This was probably because they were deploying Geiger Counters which only detect gamma rays. Later on, when there were some discoveries of DU made by the second UNEP survey,

the statement 'no widespread dispersion of DU' was changed to, 'no widespread dispersion of DU at levels that would constitute a health risk'. And of course, these levels are those predicted by the ICRP risk models.

5. COMARE, NRPB.UNEP, WHO, The Royal Society, European Union Article 31 Group.

These organisations all agree with each other that there is no health consequence of exposure to DU. They have all produced reports stating this. All these reports are 'armchair' reports based on the health model of the ICRP. None of them have used scientific induction to look at the health of people who are exposed and work backwards to the exposures. Instead they look at the cancer yield in the Hiroshima survivors and say that at the doses imparted by the DU there can be no ill health. This is not science, as I argued in my first paper for the Royal Society (Busby 2001). Scientific method is based on induction. The deductive conclusion about DU and health is similar to the deductive conclusion that the Sellafield leukaemia cluster is not caused by radiation from Sellafield. Both arguments are scientifically bankrupt.

5 The DUOB, Department of Health and the British Ministry of Defence

In 2000, Molly Scott Cato, Richard Bramhall and I published a small book, *I Don't know much about Science* (Scott Cato et al 2000). In it we analysed the results of questionnaires sent to UK Members of Parliament to see what qualifications in Science they had. We also addressed the question of scientific advice to government in the immediate post Mad Cow disease period and asked how such a situation could have come about. We concluded that science advice committees were biased in the direction of Industry. Molly, who has studied politics and philosophy at Oxford suggested that the only way to allow for such bias was to have oppositional science committees. In these structures, there are scientists from both sides of the argument, funded by government, who debate the issue within the committee and finally publish a report which draws attention to the consensus but also to the disagreements with suggestions for research that might resolve these. Shortly after this book two committees came into being where this approach was adopted (but without the funding). These were CERRIE, which I have mentioned above and the Depleted Uranium Oversight Board. Because the DUOB has members from all sides of the argument, from the Veterans and the Defence establishment, it is possible to ensure to a large extent that there are honest investigations of DU in the urine of the veterans. We have tried to ensure, by elaborate mechanisms of coding and questionnaires which are photocopied and redistributed to several organisations, that there can be no James Bond exercise in which the MoD dilute the samples or alter the questionnaires. And so at the end of this process, I believe that we can get a real understanding of the levels of DU, some ten years after the Gulf War I. However, the Chair, David Coggon, has been able to force the question of asking the veterans who are being tested whether they have been diagnosed with cancer off the questionnaire. It was clear from the discussions that he was terrified that this question, properly answered, would enable us to analyse the samples to show that there was a significant effect. This process has to be left to 'expert epidemiologists' which many feel

means 'tame scientists' who will find nothing. This serious for us and for the veterans because we do not trust the epidemiological studies that are supposed to be happening, if indeed any happen at all.

But there is an interesting development. It seems that the DUOB may be put in charge of the whole testing and medical exercise in the Gulf War vets and perhaps also in the Gulf II veterans in which case we will able to ensure that the epidemiology is above board. But is this does not happen, and the epidemiology is done outside the DUOB without our close inspection, then we cannot have any confidence in the results.

6. Summary: Depleted Science

So finally my conclusion. The DU story is the tip of a large iceberg, which represents the health effects of low dose radiation from man's activities in the last 100 years. Since the discovery of radioactivity the planet has been slowly filling up with radioactive material. The trend in the increase in child leukaemia since 1900 has closely followed the trend in Uranium mining and Radium production, an observation first made by Bramhall [Busby 2002]. The present cancer epidemic is a consequence of the testing in the atmosphere in the period 1959-63 of bigger and bigger atom bombs. People living in the Chernobyl affected territories, near the test sites in Kazakstan, Nevada, the South Pacific, Australia, near Sellafield and the contaminated Irish Sea, all are suffering. And now we are seeing the health effects of these widely dispersed DU particles. It will not only be the military who are affected, it will be everyone. And the reason this has been permitted is that the health effects of internal low dose particulate radiation has been assessed by looking at high dose acute radiation from a nuclear bomb. This is Depleted Science.

In addition, there is a cover-up of the cause of the present cancer epidemic, and the cause of cancer generally. Cancer is an environmental disease and is increasing because of the runaway contamination of the environment by the products of industrial expansion and radioactivity from industry and the military. If we were able to examine the rates in people who live close to contamination sources this would be apparent. But we cannot. The data exists but is kept secret. The cancer registries are part of a huge and high level cover-up of the cause. The data would not even be released to the UK environment minister Michael Meacher, who was concerned about the effects of nuclear sites and wanted to examine the data himself. After two years of pressing for the release of small-area anonymised cancer figures, he was sacked in June 2003. So we cannot examine this and people will continue to die. Only last week, I was informed, to my astonishment, that the limited small area cancer mortality data that we have been buying from the UK Office of National Statistics, (and which we have used to show cancer excesses near two nuclear power stations, Hinkley Point and Bradwell) was no longer being sold to us as of September2003. This new decision, and the cover up of health data is a most serious matter which requires the attention of the Green and environmental movement and all honest people everywhere on this small green planet if we and our children are to survive.

Appendix: Cancer in Basrah Hospital, Iraq, 2003

From data supplied by Dr Jawad Kadhim Al Ali MRCP Consultant physician and oncologist, Oncology Center, Basrah Hospital, Iraq (email: Jawadalali44@yahoo.com)

I met Dr Al Ali in Hamburg and was given this data by him. He is the cancer specialist at the Basrah hospital and was trained in the UK. His observations included the following extremely rare events:

1. Familial clustering of cancer. More than one case of cancer in a single family in 58 cases.
2. Two different cancers in 9 patients'
3. Triple cancer in one patient.
4. Change in the age of cancer expression with cancer normally seen in the old being diagnosed in younger people.
5. Increased incidence of congenital anomalies which he puts down to radiation exposure of pregnant women by DU

Table 1 Incidence of malignant disease among children (0-14) in Basrah

Site	**1990**	**1993**	**1994**	**1995**	**1996**	**1997**
leukemia	15	15	14	25	24	24
lymphoma	2	4	1	5	8	8
Brain	1	4	3	2	5	6
neuroblastoma	0	0	0	0	0	3
Wilms tumour	1	3	2	4	1	0
others	0	1	1	0	0	2
All	19	27	21	36	38	43

Site	**1998**	**1999**	**2000**	**2001**	**2002**
leukemia	24	30	60	70	85
lymphoma	9	19	13	18	35
Brain	2	2	3	3	7
neuroblastoma	4	6	3	2	12
Wilms tumour	0	3	0	0	6
others	3	5	13	7	15
All	42	65	92	100	160

Table 2 Incidence rate of malignant disease among children in Basrah from 19993-1998 compared with 1990

year	**Population 0-14**	**cases**	**Rate per 100,000**	**Indexed to 1990 (p-value)**
1990	476549	19	3.98	1.0
1993	518929	27	5.2	1.3
1994	533877	21	3.93	0.98
1995	459234	36	7.83	1.97
1996	565055	38	6.72	1.68
1997	581332	42	7.22	1.8
1998	627754	42	6.69	1.7
1999	605045	65	10.7	2.7
2000	604015	92	13.1	3.3
2001	792017	100	12.6	3.2

2002	863909	160	18.5	4.6

Table 3 Geographiacal prevalence of malignant disease among children 0-14 in Basrah area in 2002

	Children 0-14 population	Cases	Incidence rate per 100000
Basrah centre	362731	20	5.51
Al Hartha	68947	25	36.25
Qurim	95202	28	29.41
Al Mudina	70457	30	42.57
Al Zubier	147798	45	30.44
Abu al khassib	82325	2	2.42
Shat al araab	36449	10	24.43

Note: incidence rate per 100,000 in England and Wales was about 12 per 100,000 in 1994
Cancer in children in Iraq was low before 1990.

Table 4 The distribution of child leukaemia patients in Basrah according to age.

	0-4	**5-9**	**10-14**	**0-14**
1990	2	9	4	15
1993	5	6	4	15
1994	5	5	4	14
1995	10	9	6	25
1996	10	10	4	24
1997	10	10	4	24
1998	10	9	5	24
1999	14	11	5	30
2000	34	14	12	60
2001	41	9	20	70
2002	53	17	15	85

Table 5 Mortality rate among children with leukaemia in Basrah

	New cases	leukemias	deaths	Rate%
2000	92	60	35	58
2001	100	70	50	71
2002	160	85	60	71

Table 6 Incidence rates for congenital malformations in Basrah 1990-2001

Year	Number of births	Number of congenital malformations	Rate per 1000 births
1990	12161	37	3.04
1991	9845	28	2.84
1992	11800	23	1.95
1993	12416	28	1.31
1994	12250	36	2.93
1995	10576	46	4.35
1996	10470	48	4.56
1997	13653	32	2.34
1998	10186	79	7.76
1999	13905	136	9.78
2000	12560	221	17.6
2001	11445	254	22.2

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Appendix E:

Keith Baverstock, the senior radiation advisor to the WHO was sacked because he drew attention to the matters I have raised in this report. This is his paper on the issue, published in a peer review journal.

Science, Politics and Ethics in the Low Dose Debate

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The roles of science, ethics and politics are identified in respect of the risks of exposure to low-dose radiation. Two case studies, the epidemiology of the United Kingdom nuclear test veterans and the risks to civilians associated with the military use of depleted uranium, are considered in the context of their ethical framing, scientific evaluation and political resolution. Two important issues for the present and future, the safe management of UK radioactive waste and the future of nuclear power, in which the science of low dose effects will be crucial and where the ethical issues are much more complex, are introduced. Specific consideration is given to the potential hereditary effects of ionising radiation in relation to the current state of radiobiological knowledge. It is concluded that for science to be useful in public health policy making there needs to be some reform from within the profession and the political imperative for freely independent scientific institutions.

KEYWORDS Depleted uranium Ethics Genomic instability Nuclear power Nuclear weapons tests Politics Radiation risks Radioactive waste

Introduction

A life without any risks whatsoever would be boring and some would say totally uncharacteristic of human nature, so we must accept that risk is a part of life. But how much, of what nature, and how caused, are important issues not to be dismissed lightly.

Alice Stewart identified a risk to children from the exposure of pregnant women to diagnostic X-rays in the 1950s, which was to prove to be pivotal in transforming our perception of risk from low-dose ionising radiation.(1) It was by any standards a remarkable piece of dedicated scientific investigation. It caused alarm and concern in the radiological protection and medical communities when the result was first published in 1956, when Alice was a relatively newly qualified doctor. The initial ‘establishment’ diagnosis was that there was a mistake, but as the evidence was consolidated and a similar

result was reported with a larger number of cases and controls, the personal criticism started. It was not to be until the 1970s that Alice's claims were vindicated. The International Commission on Radiation Protection (ICRP) recently published a report (2) that devotes great attention to Alice's contribution (the Oxford Survey of Childhood Cancers is the biggest study on this issue ever mounted), but generally in a highly critical tone, with an eventual rather grudging acceptance that Alice was right.
Alice's professional life illustrated a phenomenon that can be seen when someone, as the great British geneticist CH Waddington noted, not a member of the 'dominant group' in scientific society, claims a discovery. First, attribute the 'discovery' to a simple error born of lack of experience; then, when the claim to the 'discovery' is not withdrawn, attribute mental instability to the discoverer; and finally, to point out that this 'discovery' was not a discovery at all but had in fact always been known. Now the ICRP has added a 'post-final' stage which, in essence, notes that the basis for the discovery was in fact highly suspect and the discoverer the beneficiary of a great deal of luck in that although the correct result was obtained it was not by a scientifically valid method. I will return to the issue of flawed epidemiology later. But let there be no doubt that Alice overturned what was the established view of radiation as being rather a benign agent with great benefits to humankind, and the process she started in the 1950s is still changing our view of this particular agent. So in this sense Alice lives on.

Radiation Risks

We know that exposure to ionising radiation does present serious risks to health. Exposure to, say, one gray (Gy) in periods of a few hours will produce health consequences that are directly observable and attributable to the exposure and we understand what causes what happens as a result of scientific investigation. But when we try to study what effect such a dose might have when spread over several years, measurement of the effect in a population, through epidemiology, becomes much more problematic. Some findings point to a risk, others are inconclusive, and still others seem to indicate a beneficial effect. Chance has started to play a role. In these circumstances science comes into play again, by constructing models of what might be happening and extrapolating those models on assumptions that are judged or believed to be reasonable and realistic. An example is the linear no-threshold (LNT) model, upon which radiological protection is based. I believe that LNT is credible and realistic (as well as an appropriate modus operandi for radiological protection) on fundamental grounds; nevertheless, that is a belief and not knowledge. Others believe otherwise. However, societal decisions concerning risk acceptability are essentially political. In democratic societies such decisions are taken by elected governments, or bodies nominated by them. Most governments make strong claims to be basing their decisions on risk on the best available scientific opinion, but we must be aware that this opinion involves beliefs as well as knowledge.

Beliefs of another kind are also relevant in this context, which can collectively be called ethical considerations. These almost always 'frame' the risk issue, influencing the perceived importance of various aspects of the risk debate; they therefore also impact on the science. Risk is a simple sounding word but it is quite a complex concept. It involves both the degree of impact and the frequency of both detrimental and beneficial effects, which sometimes leads to ambiguity and therefore misunderstanding. Uncertainty is usually entrained in any risk assessment and has to be identified and addressed. In policy making risks have to be traded, mitigating one risk may enhance another, one detriment might be accepted to obtain a benefit elsewhere, economic cost in mitigating one risk might exacerbate another, and so on. Good policy seeks the best compromise in a very complex web of scientific and social issues, but my thesis is that it requires above all sound and honest science and careful and sensitive ethical framing.

The aim of this paper is to explore, initially with the help of two relatively simple examples, how science, ethics and politics have inter-played, one with another, in practice. In each case I will first outline the setting for the risk issue, then discuss the ethical and scientific considerations, positing what might be the correct political outcome, and finally I will describe what has in fact happened. I then introduce two issues for the future where the scientific, ethical and political dimensions are much more complex.

Two Case Studies

The examples I wish to use to illustrate my points are firstly the health of the United Kingdom test veterans: Some 25,000 people, mainly young men, served their country by providing the backup and support for the UK weapons testing programmes in Australia and the South Pacific in the 1950s. For more than the past 25 years many have felt that their health has been adversely affected but many do not receive compensation for their injuries. The second example is the use of depleted uranium (DU) weapons. DU has been used in many battle theatres since the Gulf war of 1991. Although DU is acknowledged to be radioactive, its specific activity is low and it is therefore not thought to present a serious radiation hazard.

I cannot, in the time available, give exhaustive consideration to either one of these, let alone both, so I will have to be selective, but I also hope to be even-handed. I consider each in turn and try and draw some conclusions at the end.

The UK Test Veterans

In 1983, following pressure from veterans associations, it was decided to mount an independent epidemiological study on the UK test veterans, and the National Radiological Protection Board (NRPB) was funded to undertake the work by the Ministry of Defence (MoD). The study was designed to compare the test veterans with a similar cohort of servicemen from the three services whose tours of duty took them to tropical areas but not the nuclear tests in Australia or the Pacific. There have been three

analyses of the survey, the latest published in 20033 together with a fuller report.(4)

There was a strong ethical element in mounting this study, as it was considered at the time that if there was reasonable concern that an adverse health consequence would accrue from any occupational exposure, the possibility of resolving the concerns through epidemiological study should be considered. It is certainly the case that no one thought that the study was intended to add to scientific knowledge about radiation as a cause of cancer; the survey had a purely socio-ethical justification.

The NRPB initially accepted from the MoD, apparently without independent verification, the primary data in the form of names of servicemen attending the tests and the rather sparse dosimetric data. It was recognised from the outset that there was incomplete ascertainment, especially of RAF personnel, and after the first analysis in 1988, further veterans were found by the veterans' organisations and notified to the NRPB. These persons were put in a separate category called 'independent responders' and have not been included in the main analysis but have been analysed separately.(5) It is now established that there might well be a shortfall of 15 per cent on the ascertainment of the test veteran population.

From a scientific point of view and contrary to the claim made by the NRPB, this shortfall raises the prospect of a serious flaw in the methodology.5,6 The exposed and control populations can no longer be guaranteed to be free of bias as there can be no such shortfall in the controls, who were not ascertained from a finite and defined population (they were simply 22,000 service personnel not having served in the test areas). The NRPB claims that in spite of the shortfall the population is representative.

The veterans' associations maintain that the independent responders should be included in the main analysis. Sue Roff maintains that the NRPB analysis has missed 30 per cent of the cases of multiple myeloma and thus an analysis missing 15 per cent of the veterans cannot be representative.(7) There does not have to be any deliberate wrong-doing for there to be a serious bias problem here. Records compiled in the 1950s may well be incomplete some 20 to 30 years later, especially if there were no compelling reasons to keep the records in good order. If the loss of the 15 per cent of records was associated in any way with the health outcomes being studied, then the fact that there is not a comparable loss in the controls (for the same reason) immediately introduces bias. One reason for the loss of records may well be their relocation in connection with a claim for compensation or diagnosis of illness or death. When all in this population have died there will be some 25 per cent of deaths due to cancer. At the last analysis, almost 23 per cent of veterans had died, with seven per cent from cancer, that is, less than the 15 per cent missing from the study population. Thus it is entirely possible for the missing 15 per cent to conceal an excess of cancer deaths.

A second issue concerns the results of the survey, which found an excess of all leukaemia, excluding chronic lymphatic leukaemia (which is not

thought to be associated with radiation) in the veterans population when compared with the controls but not when compared with the general population.(3) This results from a large deficit of leukaemia in the controls for which the NRPB has not found an explanation, so attributes it to 'chance'. Now there is an ethical issue here. Having chosen at the outset to compare the veterans with a control population, it is unacceptable both scientifically and ethically to 'move the goal posts' when the result is known. Even more interesting, but not disclosed by the NRPB (although they claim to have known it), is the fact that in the veterans population, the excess leukaemia risk, compared to the controls, appears to be concentrated in those who served in the Pacific.(6) Those attending tests in Australia seem to have a similar leukaemia incidence to the controls. Of course, as the populations are subdivided so the statistical significance of the result declines, and it becomes more difficult to define risks as attributable.

The lack of dosimetric data also is a factor. Some duties, such as decontaminating planes and vehicles, are likely to incur higher doses than others such as servicing the canteens. Lumping together the exposed and unexposed in the absence of individual dose assessments, however crude, will 'dilute' any exposure-related excess of disease.

The correct political conclusion is that the NRPB survey, for a number of reasons, is deficient and that the data have not been exploited to the full extent that is possible to resolve the impact on health of the test veterans. Further work needs to be done.

The present political position is that, according to the NRPB survey, the veterans have not had their health damaged by their participation in the tests and thus the MoD is able to conclude that there is no case for compensation for injuries that are claimed as due to radiation.

In this case it is clear that the science and the associated ethics (of recognising the need for an independent study) have been perverted for political ends. It is sad that the NRPB, which should be an independent and technically competent body, was complicit in this process.

Depleted Uranium

Depleted uranium (DU) has a lower specific activity than naturally occurring uranium, which contains greater quantities of two other uranium isotopes, U-234 and U-235. Technically it is a waste arising from the enrichment process that produces U-235 for weapons and civil nuclear power plants. In 1991 it was used in the Gulf War as a weapon. It is not its radioactivity that makes it effective as a weapon but its density.When delivered as a bullet to a hardened target, a DU munition will have sufficient momentum to penetrate armour and buildings. After penetration the bullet fragments and burns, causing the release of an oxide smoke consisting of very fine particles. Typically, when a tank is hit, up to four kg of depleted uranium oxide dust is formed.

There is of course an ethical dimension to war of any kind, but putting

that to one side for the time being, the Geneva Convention seeks to minimise the impact of war on civilian populations. Over time battlefields usually return to civilian use and weapons that remain, such as unexploded land mines and cluster bombs, are not supposed to be left to pose a health risk to the civilian population. The question here is whether DU, either as unburned metal or as oxide dusts, poses such a risk. If it does it should be cleaned up or such weapons banned.

Natural uranium (NU) is ubiquitous in the environment and the chemical properties of DU are identical to those of NU. DU metal buried in soil will, over many years, dissolve and enter ground water where it may raise the uranium concentration, perhaps by a few per cent. This is unlikely to pose a public health hazard.

As far as the International Commission on Radiological Protection (ICRP) and the International Atomic Energy Agency (IAEA) are concerned, DU oxide dust can be treated like any other uranium oxide.(8,9) However, although there is extensive exposure of workers in uranium mines to naturally occurring uranium oxide dust, there is no natural analogue for depleted uranium oxide. What we know from occupational exposure to uranium compounds, including relatively soluble oxides, is that uranium is chemically toxic. In fact, the ICRP regards uranium primarily as a chemical hazard and not a radiological one, an exception being insoluble uranium containing particles retained in the lung. However, the product of burning of depleted uranium is a mixture of two oxides, one insoluble and the other sparingly soluble. The more soluble oxide of depleted uranium, when in the body, for example retained in the lung, results in the formation of the uranyl ion. This, while soluble in tissue, binds avidly to DNA and proteins and so is only slowly transferred from the lung tissue to blood, from where it is transferred to other tissues, particularly the bone, before finally being excreted through the kidney. Damage to the kidney from exposure to the uranium is generally regarded as the principal toxic effect. However, recent results have indicated that while it is in transit to the kidney, that is, retained over long periods deep in the lung, it may give rise to genotoxicity, mediated not by radiation alone but by its chemical properties in combination with its radioactivity.(10) There will be a period, ranging perhaps from months to years, where a slowly dissolving particle in the deep lung is surrounded by cells containing uranyl ions. Typical particles may emit an alpha particle once every few weeks, and thus there is the possibility of a synergistic effect between a chemical carcinogen and radiation. There is also the possibility, particularly important for lowspecific-activity alpha emitters, of effects mediated by the bystander effect, where cells not actually irradiated, but located close to ones that are, exhibit radiation effects.

From articles published in 2003 it is clear that neither the ICRP (9) nor the IAEA (8) have taken these three potential effects into consideration when they assess the risk from inhaling depleted uranium dusts. It is also the case that when the World Health Organisation were advised of these three potential

mechanisms they ignored the information in the preparation of a monograph on the health effects of depleted uranium published in 200111 and subsequently suppressed the publication of a paper postulating these three mechanisms.

In 1991 the United States forces discharged 300 tonnes of depleted uranium in the area around Basra. More than 800 tonnes are said to have been deployed by the US in the latest Gulf War. Given the arid and dry climate that affects much of Iraq it seems likely that the resultant oxide dusts will remain potentially dangerous, if re-suspended, for a considerable time. This contamination presents a serious potential hazard to health for both the Iraqi population and the coalition forces.

The science here is clear. The hazard is not certain, that is, the risk is not attributable, but it is not so speculative that it should be ignored. The ICRP routinely uses essentially untested models to determine the risks from internal emitters. I suggest that the science behind the postulated mechanisms I have just described is somewhat harder than that underpinning some of the ICRP models. But there is also an ethical issue in connection with the role of the IAEA in responsibility for the safe use of nuclear technology. Depleted uranium is a by-product or waste of nuclear technology and thus should come under the safety mandate of the IAEA. But its responsibility should not be limited to the radiation effects, but should consider all the hazards associated with the material. Nor should the ICRP consider that its responsibility is confined to radiation effects. Where there is the possibility of an interaction between two carcinogenic processes the non-radiation one should not be ignored. Perhaps the most serious violation is that of the WHO, whose mandate to protect public health has surely been compromised. In an ideal world the WHO would have alerted the IAEA and the ICRP to the potential hazard of DU oxide dusts in Iraq.

The political situation as it should be is that, until there is clear evidence that DU oxide dusts are harmless, either the weapon should be banned or battlefields where it has been used should be cleaned up. Also there are alternatives to depleted uranium for its military uses. Tungsten is almost as good a penetrator but it does not break up and catch fire, so it is not as effective at killing people as depleted uranium. We cannot therefore ignore the possibility that the IAEA, ICRP and WHO are responding to political pressure not to disclose the potential health consequences to either military or civilians in the use of depleted uranium. In fact, Dr Thomas Facy (personal communication) notes the discrepancy in the US between the military attitude to the hazards of uranium and that taken by the civil uranium industry, where effective and elaborate precautions are taken to protect the workforce. Clearly there are double standards operating here.

Implications

These two examples illustrate how science can be distorted to achieve political ends, in the first case to avoid paying compensation and in the second retaining the military capability of DU. I could have given many

other examples but I chose two that are topical, one because of the relatively recent report by the NRPB on the test veterans study, and the other because the existing contamination of Iraq by DU has, within the past year or two, been made even worse, and three international agencies have recently endorsed a less than precautionary approach to the effects of DU on health. All the organisations involved – the NRPB, the WHO, the ICRP and the IAEA – claim 'independence' and technical/scientific excellence. At a Nuclear Energy Agency workshop in Villigen, Switzerland, I even heard Abel Gonzales of the IAEA, who is also a member of the ICRP Main Commission, claim that the ICRP is a 'scientific academy'. While it has to be recognised that several highly qualified people are members of that organisation, it hardly rates as that. But it is the case that the international agencies (WHO and IAEA) and the NRPB do employ people who should be scientifically and technically competent and trustworthy. So if it is not incompetence, what is it that has led to such perverse political outcomes when the science and the ethics are so clear, even if the science is not sufficiently strong to produce irrefutable evidence to allow risks to be objectively assessed – that is, it depends on judgement to a degree?

The fact that situations such as these exist has a profoundly negative effect on policy making, by corroding public trust in science and technology. In the UK this process has been underway since the early 1980s where radiation is concerned. Previously, the Medical Research Council played the role of a 'referee' between the pro- and anti-nuclear lobbies through the application of objective scientific risk assessment. There was considerable public (and 'player') trust in the result, and as a consequence there was not the same social concern about the risks of low doses as there is today. Politics, aided and abetted by some in the scientific community, can be said to have poisoned the well that sustains democratic decision-making.

Two Problems for the Future

At the present time we face at least two very crucial and inter-related issues. The first is especially important to the UK: the future management of the accrued radioactive wastes over the past half-century. The second is a global problem but one for which there has to be national policies, namely the future of energy supply and the role that civil nuclear power will play. I have a particular interest in the first problem as a member of the Committee on Radioactive Waste Management (CoRWM) set up recently to advise government on a long-term management strategy that is both implementable in the foreseeable future and commands public confidence.

Both these problems have very significant ethical dimensions relating to environmental sustainability, equity and fairness to future generations and to those in whose backyard existing and future wastes will end up. Both are also extremely technically challenging, the first requiring a means of protecting the environment from the release of radioactivity for very long periods and the second technical ingenuity to ensure future generations sufficient energy supplies. So far we have barely started to solve these

problems. Clearly, as far as the second problem is concerned, we have to consider the future of nuclear energy, a non-greenhouse gas source, with an abundant fuel supply but a largely unsolved waste problem, in which the low dose issue is writ large.

With respect to the second problem we do have a choice as to whether to solve it or not, but that option does not exist for the first problem, since we already have the waste. This must be managed in some way, even if this involves continuing to store it on the surface, at some considerable cost, which takes resources from other possible uses, and where the waste is vulnerable to accidents and terrorist action.

An issue common to both problems is that the ethical framings through which individuals see these problems can be very diverse, even directly opposed. This exposes the need for some serious risk trade-offs. In the case of the future of nuclear power, the health risks of low dose exposure have to be traded against those of global climate instability, so long as carbon based fuels are the only viable alternative to nuclear. A similar issue arises in respect of the stocks of plutonium in the UK. This material can be seen either as a threat because of its potential to make weapons or as future fuel. Here the risk of misuse now or in the future has to be traded against the benefits that could accrue to future generations if the plutonium is available to be used as a fuel.

Clearly, the solutions to these major challenges are not only a matter for scientists and technologists but require, in a democratic society, the close involvement of the public and those who have a particular interest, whom we call stakeholders. Nevertheless science and technology has a crucial role to play. We should not be forgiven by future generations if we fail to use, to the full, the best scientific and technological knowledge known to man. The failure of good science to prevail in the two examples above and in the many others I could have quoted, has eroded the trust of the public and stakeholder communities in science and technology to the extent that there has grown up a phenomenon called ‘cognitive relativism’. This believes that there are no truths and no best solutions to problems that have a strong scientific and technological element. In the view of a cognitive relativist risks from low dose radiation are a matter of belief, not reality.

Cognitive relativists would advise us to find solutions to the challenges of future energy supply and nuclear waste management primarily on the basis of public opinion. In the UK it is estimated that less than ten per cent of the population claims to know anything about nuclear waste. It would seem to me that relying on that approach would be little better than tossing a coin to choose between options.

Clearly we must do better than that, but it is a real problem to find a way to ensure that any solution to the waste problem is safe and satisfies the legitimate requirements of democracy. This problem has been made significantly worse by the experience of the test veterans and by the way the DU issue has been handled, as I have just presented to you and by many other examples in a similar vein. Sadly it is the case that some of those

scientific and technically based organisations that have taken on the responsibility of being 'independent' technical bodies have misused science in a way that overrode the strong ethical issues in which the problems being tackled were framed by society. Done once this can be seen as an accident, but when it is done repeatedly we know that it is deliberate political interference. It becomes increasingly difficult for society to trust these bodies, and ultimately those who set them up, namely politicians, with the overridingly important task of protecting public health, and the door is open to cognitive relativism.

Back to the Science

Even without this problem we would, as scientists, have an extremely complex task in solving the radioactive waste problem. I want to focus on only one of many uncertainties in predicting future risks from radiation exposure, that is, the future genetic consequences. The phenomenon of radiation induced genomic instability was only discovered just over a decade ago,12 which, because of the large target size for the effect, is important at low doses. Somewhat more recently the transmission of a form of instability, mini-satellite DNA mutation along the germ line13–17 has been revealed. So far mini-satellite mutations have not been linked with a specific health effect and they occur spontaneously at a relatively high frequency in any case. Does this mean we can ignore this effect in projecting over many generations the risks of exposure to low doses of ionising radiation?
One feature of this phenomenon that concerns me is the lack, in the studies with mice,(13) of 'dilution' of the effect in the 'grandchildren' of the irradiated mice, that is, the mutation frequency is the same in the two offspring generations. There are two implications:

. there is likely to be no fading-out of this effect over generations in the future as would be expected in classical genetic effects;

. a mechanism that is presently wholly unknown must be involved.

These implications must decrease considerably the confidence we can have about the health of future generations after exposure to radiation.
Perhaps the first question to answer is; 'are these observations reliable'?
The phenomenon of mini-satellite mutation has been seen in the children of Chernobyl exposed fathers (14,17) and in the two generations of offspring from fathers exposed to weapons testing in Kazakhstan. (16) An extremely closely related phenomenon, tandem repeat mutations, has been seen in mice.(3) However, mini-satellite mutations have not so far been observed in the children of the survivors of the atomic bombings in Japan,(18) in Chernobyl clean-up workers, (19) or in a study of radiotherapy patients.(20) There could be reasons for this lack of uniformity of observation and we should recall that we assume a genetic risk in radiological protection largely on the basis of studies in mice and not direct observation in humans. I think we have to

assume that at least under some conditions the phenomenon of germ-line transmission of mini-satellite mutations exists. We should, therefore, exercise caution even though we do not know how seriously the phenomenon impacts on health, as the effect may be irreversible and potentially with major consequences.

I contend that phenomena such as this, and genomic instability in general, can be understood if we assume that the genome is a dynamically stabilised or self-organising entity.(21) For life to have evolved over more than three billion years requires astonishing robustness. Yet, when we look at living systems we see incredible complexity, requiring a very high degree of organisation both spatially and temporally that surpasses anything that can be man-made even in the simplest of living creatures. Usually qualities of robustness and complexity do not go hand in hand. It is as if a living organism is an object with the simple robustness of a steam traction engine and the complexity of a Formula One racing car. Perhaps we find it difficult to comprehend such an object because we have so far not really studied dynamically stabilized objects very thoroughly. Another such system is the climate, which while capable of producing extreme conditions, is in fact extraordinarily stable given its possibilities. Yet we have the 'butterfly wing effect', in which it is said that a flap of a butterfly's wing in Hong Kong can cause a hurricane in the Caribbean. In other words the system is both robust and sensitive. The climate is a dynamically stabilised system. Very little attention has been given to the possibility that the genome is such a system. The point here is that as scientists we must be open to possibilities not so far conceived. Perhaps it was Alice Stewart's 'inexperience' in her early professional years that allowed her to take seriously results that her peers would probably have rejected as artefacts. This was Waddington's message when he derided the 'conventional wisdom of the dominant group', which he coined as the acronym COWDUNG.(22) There is, however, a genuine paradox here; we need 'stability' in the knowledge base to make scientifically informed policy, but we also need to move forward in our scientific understanding of nature in order to ensure that policy remains realistic. How to achieve the most beneficial compromise is a true challenge for the scientific community.

What should not be such a challenge is how to use our existing scientific institutions to better serve the policy-making process. There are clear aberrations here and they can be corrected with the appropriate determination of the scientific community and the appropriate political will.

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SCIENCE, POLITICS AND ETHICS IN THE LOW DOSE DEBATE 99
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